Body Cues, Not Facial Expressions, Discriminate Between Intense Positive and Negative Emotions

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The distinction between positive and negative emotions is fundamental in emotion models. Intriguingly, neurobiological work suggests shared mechanisms across positive and negative emotions. We tested whether similar overlap occurs in real-life facial expressions. During peak intensities of emotion, positive and negative situations were successively discriminated from isolated bodies but not faces. Nevertheless, viewers perceived illusory positivity or negativity in the nondiagnostic faces when seen with bodies. To reveal the underlying mechanisms, we created compounds of intense negative faces combined with positive bodies, and vice versa. Perceived affect and mimicry of the faces shifted systematically as a function of their contextual body emotion. These findings challenge standard models of emotion expression and highlight the role of the body in expressing and perceiving emotions.

Jennifer checks the numbers in her lottery ticket, when she realizes she hit the 10-million-dollar jackpot. Michael fumbles for his car keys while his 3-year-old son steps into the street and is hit by a passing car. In a split second, Jennifer and Michael experience the most intense reactions of their lives. Intuitively, their emotional expressions should differ vastly, an assumption shared by leading models of emotion. For example, basic emotion models, which posit distinct categories of emotions such as anger and fear, predict that intense emotions activate maximally distinct facial muscles, which increase discrimination (1, 2). Similarly, dimensional emotion models, which posit that valence is a primary dimension of emotion perception, predict that intense emotions are located on more extreme positions on the pleasure-displeasure axis and thus their positivity or negativity should be easier to decipher (3).

The question of affective valence discrimination is theoretically important for the structure of emotion models and is central for understanding how social communication takes place in highly intense and potentially dangerous situations. Yet, although it is commonly assumed that facial expressions convey positive and negative affective valence in a highly distinct manner, there is still room for question on both methodological and theoretical grounds. From a methodological standpoint, most studies to date have used posed prototypical facial expressions (1) that have been carefully designed to signal clear and distinct emotions (4–7), and indeed the higher their intensity, the more accurately recognized they become (8–10). However, expressive facial behavior may be different in real-life situations. Studies that have shown successful affective valence differentiation using ecological expressions have not focused on the transient peaks of intense emotions. For example, a study on face expressions in the Judo Olympics sampled reactions across a relatively extended duration (~15 s) after the transient emotional peak, potentially diluting the most intense reactions (11). Other work showing good accuracy in differentiating pain and sensual pleasure did not focus on intense emotional peaks altogether (12). Therefore, it remains unclear how distinct peak intensity expressions of opposite affective valence actually are.

Further, a clear-cut distinction between positive and negative expressions may be theoretically unwarranted. Neurobiological work has shown that the opioid and dopamine systems modulate both pain and pleasure (13), and brain imaging studies consistently show regions activated by both positive and negative emotions, including the insula, striatum, orbitofrontal cortex, nucleus accumbens, and amygdala (14–19). Similarly, research in motivation and emotion has shown that a sharp distinction between positive and negative emotion experience is sometimes hard to draw (20–22). These findings all hint at potentially overlapping and shared mechanisms across positive and negative emotions.

We therefore examined whether facial expressions of opposite affective valence might also overlap during highly intense peaks of emotion. We defined a peak emotion as the apex of a highly intense emotional experience and focused on the immediate peak expressions in response to real-life situations such as undergoing a nipple piercing, receiving an extravagant prize, winning a point in a professional sports match, and so forth. Furthermore, unlike most previous work (e.g., (23)), we took a “full person” approach (24) and

References and Notes
24. V. Kasprowicz et al., J. Virol. 82, 3154 (2008).

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examined the face-body dynamics during expression. We predicted that during peak intensity moments, facial reactions would overlap and be nondiagnostic for the affective valence of the situation. Consequently, we expected that body context would “step in” and aid in disambiguating the face.

To test this hypothesis, we first used peak expressive reactions to winning and losing points in professional high-stakes tennis matches that typically evoke strong affective reactions (experiment 1). Three groups of 15 participants rated the affective valence and intensity of either the full image (face + body), the body alone, or the face alone (Fig. 1, A and B) (25). Consistent with our prediction, whereas participants failed to rate the affective valence of winners as more positive than the affective valence of losers when seeing the face alone, they succeeded when seeing the body alone or the body and the face together [mixed analysis of variance (ANOVA): $F(2, 42) = 74.05, P < 0.0001$] (Fig. 1C). Specifically, affective valence was higher for winners than losers when ratings were based on the face + body ($P < 0.0001$), or on the body alone ($P < 0.0001$), but not when ratings were based on the face alone ($P > 0.3$). Notably, although faces were not diagnostic for affective valence, they were diagnostic for intensity [mixed ANOVA: $F(2, 42) = 37.5, P < 0.0001$] (Fig. 1D). Thus, intensity ratings were higher for winners than losers when based on the face + body ($P < 0.0001$), or the face ($P < 0.0001$), but not when based on the body alone ($P > 0.1$).

Interestingly, we found an illusion in the perception of the nondiagnostic faces: 53.3% of the participants who completed the perceptual face + body affective valence rating task reported relying on face cues (which in fact were nondiagnostic), whereas 46.6% reported relying on body cues (no significant difference). Furthermore, among a separate group of participants who were given a description of the type of images in our task (without seeing the stimuli), 80% chose the face as the part that would be most diagnostic for affective valence discrimination, 20% chose the face + body as equally diagnostic, and none chose the body. We refer to this phenomenon as illusory facial affect: the perceptual attribution of clear positive or negative affect to an inherently ambiguous face while disregarding the objective diagnostic source of the affect in the body.

Illusory facial affect hints at a critical role for the body in shaping the perceived affective valence in intense expressions. We tested this proposition directly by creating face-body compounds with photos of losing faces combined with winning bodies, as well as winning faces combined with losing bodies (supplementary text). Participants rated the facial affective valence in these manipulated images, alongside the original images (experiment 2, Fig. 2A). The critical images were diluted amid a large number of filler images, and participants were unaware of the manipulation. As predicted, the perceived affective valence of the same faces shifted categorically depending on the body with which they appeared [repeated ANOVA: body effect $F(1, 14) = 118, P < 0.0001$] (Fig. 2B). Indeed, the effect of the body was slightly stronger for the incongruent face combinations, indicating again that the face itself was nondiagnostic [repeated ANOVA: interaction effect $F(1, 14) = 10.9, P < 0.005$].

Furthermore, the nondiagnosticity of faces generalized to a wider range of intense emotional situations (experiment 3, Fig. 3A). Participants rated faces from three intense positive situations [which included joy (seeing one’s house after a lavish makeover), pleasure (experiencing an orgasm), and victory (winning a tennis point)] and faces from three negative situations [which included grief (reacting at a funeral), pain (undergoing a nipple or naval piercing), and defeat (losing a tennis point)]. Isolated faces were nondiagnostic for the affective valence of the situation. Indeed, faces from positive events ($M = -1.4$, $SE = 0.16$) were rated as more negative than faces from negative events ($M = -0.82$, $SE = 0.19$) [repeated ANOVA: $F(1, 14) = 29.6, P < 0.0001$]. A comparison of the facial reactions within each pair of opposing emotions also failed to demonstrate the correct affective valence direction: joy ($M = -1.40$, $SE = 0.13$) versus grief ($M = -1.42$, $SE = 0.16$), $P > 0.5$; pleasure ($M = -1.42$, $SE = 0.15$) versus pain ($M = -0.07$, $SE = 0.24$), $P < 0.001$; and victory ($M = -1.40$, $SE = 0.24$) versus defeat ($M = -0.9$, $SE = 0.23$), $P < 0.001$.

We further tested the inherent ambiguity of the faces by combining all face exemplars with positive-valence (a victorious body) or negative-valence (a person undergoing piercing) (Fig. 3B) (25). Although the influence of the bodies was stronger for some faces than for others [repeated ANOVA: interaction effect, $F(5, 70) = 4.9, P < 0.001$], the effect of the body was significant [repeated ANOVA: body effect, $F(1, 14) = 96.9$, $P < 0.0001$] and held for every pair of emotions.

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![Fig. 1. Experiment 1. (A) Examples of reactions to (1) winning and (2) losing a point. (B) Examples of isolated faces (1, 4, 6 = losing point; 2, 3, 5 = winning point). [All photos in Fig. 1 credited to a.s.a.p. Creative/Reuters] (C) Mean valence ratings for face + body, body, and face. Results are converted from the original scale, which ranged from 1 (most negative) to 9 (most positive), with 5 serving as a neutral midpoint. (D) Mean intensity ratings for face + body, body, and face. Asterisks indicate significant differences between the ratings of winners and losers. Error bars throughout all figures represent SEM. ns, not significant.](https://www.sciencemag.org/content/sci/338/6104/1226/F1.large.jpg)
As the piercing image may have been unusually potent and unambiguous, we further tested the contextual malleability of the joy, grief, pleasure, and pain images, combined with winning and losing bodies. For each of the four face categories, the results fully replicated the categorical face valence shift as a function of the body [repeated ANOVA: body effect, $F(1, 9) = 63.01, P < 0.0001$; all paired contrasts, $P < 0.001$]. Thus, intense isolated faces across a broad variety of emotional situations were nondiagnostic for affective valence, and their perceived positivity or negativity shifted categorically as a function of the body.

Finally, to test if participants’ perceptions of the faces actually change depending on the body, we again combined winning faces and losing bodies, and vice versa. However, rather than rating the faces, participants were instructed to pose and simulate in their own face the exact facial movements portrayed by the tennis players (25). If perceptions of the contextualized faces actually change, then we should expect the systematic shift to extend to the motor simulation of the faces (26, 27).

This study (experiment 4) included two phases. In the “posing phase,” participants viewed contextualized faces. The stimuli included (i) winning faces on winning bodies (the original images), (ii) winning faces combined with losing bodies, (iii) losing faces on losing bodies (the original images), and (iv) losing faces combined with winning bodies. As in experiment 2, these critical images appeared amid a large number of filler trials (which were not analyzed) and...
debriefed participants were unaware of the manipulation. Posing participants viewed the images one at a time and adjusted their facial expressions to fit those in the face on the screen. Face movements were captured on video, and the resulting poses of the critical facial expression were converted from the video to still images.

In the “perception phase,” a new group of participants viewed the images of the posers’ headshots obtained in the first phase and categorized their affective valence. Specifically, participants simultaneously viewed two faces of an individual poser simulating the exact same face in different contexts. For example, picture 1 would show the face of a participant posing a winning face combined with a winning body, whereas picture 2 would show the face of a participant posing the same winning face combined with a losing body. Participants viewed the two posed faces and decided which expressed more positive valence.

As predicted, the results showed that the affective posing of identical faces shifted systematically as a function of the body’s affective valence [exact binomial test, $P$ (two-tailed) < 0.006] (Fig. 4). Specifically, losing faces were posed as more positive when the poser viewed them on winning bodies than on losing bodies [exact binomial test, $P$ (two-tailed) < 0.01]. Conversely, winning faces were posed as more negative when the poser viewed them on losing bodies than on winning bodies [exact binomial test, $P$ (one-tailed) < 0.03]. Notably, posers had unlimited viewing of the contextualized faces, yet their motor simulation shifted as a function of the body’s affective valence. This suggests that the illusory facial affect previously reported reflects a genuine and automatic perceptual effect.

In sum, contrary to lay intuition and basic models of emotional expression and perception, the studies presented here suggest that transient peak-intensity facial expressions elicited in a wide variety of emotional situations do not convey diagnostic information about affective valence. Paradoxically, although the faces are inherently ambiguous, viewers experience illusory affect and erroneously report perceiving diagnostic affective valence in the face. This process seems to be automatic as participants have little awareness of the actual facial ambiguity and the original diagnostic source of the valence (28, 29). In line with recent work on expression perception (30), the current data highlight the critical role of contextual body and scene information in the perception of facial affect (31–34) and confirm the elusive gap between artistic truth (the expressions people expect) and optical truth (the expressions that actually occur) (35, 36). Although previous work has shown that specific emotions are hard to recognize from weak and vague spontaneous expressions (37), such findings do not challenge standard models of emotions because these models can easily accommodate context effects on ambiguous emotional states. In contrast, the finding that peak face expressions of highly intense situations cannot be discriminated on the most basic dimension of positivity and negativity poses a major challenge to standard models of emotion.

We suggest two putative complementary explanations to account for the nondiagnosticity of intense faces. At the muscular level, the nondiagnosticity may reflect a transient signaling breakdown occurring because the facial musculature is not suited for accurately conveying extremely intense affect. Much like speakers blaring at maximum volume, the quality of the facial signal becomes degraded and noisy. At the affective level, the nondiagnosticity may also reflect an overlap in experience during high-intensity emotions. Specifically, the overwhelming high intensity may move to the front of the stage of conscious experience, irrespective of the affective valence of the emotions (38). This transient degradation in signal quality need not be considered dysfunctional because (i) the ambiguity is rapidly resolved by contextual information and (ii) the face resumes diagnosticity shortly after the peak intensity resides.

Finally, although the current studies focused on the nondiagnosticity of intense facial expressions, future work may show that the underlying principles need not be limited to the visual facial modality. For example, peak emotional expressions in the auditory modality (consider intense vocal expressions of grief versus joy or pleasure versus pain, and so forth) may exhibit essentially the same patterns of nondiagnosticity as seen in faces.

## References and Notes

A Mutation in EGF Repeat-8 of Notch Discriminates Between Serrate/Jagged and Delta Family Ligands

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Notch signaling affects many developmental and cellular processes and has been implicated in congenital disorders, stroke, and numerous cancers. The Notch receptor binds its ligands Delta and Serrate and is able to discriminate between them in different contexts. However, the specific domains in Notch responsible for this selectivity are poorly defined. Through genetic screens in Drosophila, we isolated a mutation, Notch\textsuperscript{jigsaw}, that affects Serrate-but not Delta-dependent signaling. Notch\textsuperscript{jigsaw} carries a missense mutation in epidermal growth factor repeat-8 (EGF\textsubscript{R}-8) and is defective in Serrate binding. A homologous point mutation in mammalian Notch\textsubscript{2} also exhibits defects in signaling of a mammalian Serrate homolog, Jagged1. Hence, an evolutionarily conserved valine in EGF\textsubscript{R}-8 is essential for ligand selectivity and provides a molecular handle to study numerous Notch-dependent signaling events.

The evolutionarily conserved Notch (N) signaling pathway affects numerous cellular fate and differentiation events as well as proliferation and cell death (J). Signal activation is initiated by the binding of N receptor to ligands, Delta (DI) or Serrate (Ser) (2). The majority of the extracellular domain of N receptor is composed of epidermal growth factor repeat (EGFRs) (Fig. 1A). EGF\textsubscript{R}-11 and EGF\textsubscript{R}-12 are necessary for ligand-receptor interactions with both DI and Ser (3), whereas EGF\textsubscript{R}-24 to EGF\textsubscript{R}-29 (Abruptex domain) negatively regulate these interactions (4). Although the in vivo role of most EGF\textsubscript{R}s is unknown, mutations in these repeats are associated with numerous human diseases, including cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoecephalopathy (CADASIL) (5), Alagille syndrome (ALGS) (6), aortic valve diseases (AVDs) (7–9), and squamous cell carcinoma (SCC) (10–12). Mammals have four N paralogs (NOTCH1 to NOTCH4) that share similar structural organizations, but only one N gene exists in Drosophila, simplifying structure-function analysis in vivo (table S1).

To obtain N mutations, we performed an F3 mosaic genetic screen on the X chromosome (fig. S1) and isolated 42 additional alleles of N. Twenty-one alleles carry different missense mutations and were grouped into eight distinct classes on the basis of molecular and phenotypic features (tables S2 and S3). All mutated residues are conserved in most human N paralogs. One mutation, Valine361-to-Methionine (V361M) in EGF\textsubscript{R}-8, \textsuperscript{n}\textsuperscript{jigsaw}, exhibits defects in the wing margin without affecting venation or bristle development in mutant clones (fig. 1, B to F, and table S3). Hemizygous mutants of \textsuperscript{n}\textsuperscript{jigsaw} are pupal lethals, and \textsuperscript{n}\textsuperscript{jigsaw} flies do not display wing notching. \textsuperscript{n}\textsuperscript{jigsaw} fails to complement the lethality of null alleles of N and is rescued by a genomic rescue construct. Homozygous mutant clones in the wing exhibit strong notching and occasional ectopic wing margin formation (fig. 1, D and E). We did not observe any bristle density or cell fate defects (fig. 1F and figs. S2 and S3). Hence, \textsuperscript{n}\textsuperscript{jigsaw} displays a specific phenotype for a lethal N allele as inductive signaling is impaired, whereas lateral inhibition and lineage decisions remain unaffected.

Because the phenotypes associated with \textsuperscript{n}\textsuperscript{jigsaw} are similar to Ser loss of function (13, 14), we determined whether N-Ser signaling is compromised. In the early wing primordium, N is activated at the boundary between the dorsal and ventral compartments (Fig. 1G). The dorsal domain expresses both Ser and Fringe (Fng) (fig. 15), whereas DI is mainly expressed ventrally (16). Fng, a β3-N-acetylgalactosaminyltransferase that adds N-acetylgalactosamine (GlcNAc) to O-fucosylated EGF\textsubscript{R}s, modifies N so that it can be activated by DI but not Ser (17, 18). As a result, DI activates N in the dorsal domain, and Ser activates N in the ventral domain in cells flanking the dorsal-ventral boundary (Fig. 1, G and H). In \textsuperscript{n}\textsuperscript{jigsaw} hemizygous discs, N activation is severely reduced or lost (Fig. 1I). Furthermore, cells that activate N signaling are present in the dorsal but not ventral compartment (Fig. 1I’). These data indicate that \textsuperscript{n}\textsuperscript{jigsaw} is defective in N-Ser but not N-DI signaling. This was confirmed in mosaic experiments (fig. S4). Last, \textsuperscript{n}\textsuperscript{jigsaw} mutant clones can be ectopically activated by overexpression of DI but not by Ser (fig. S5).

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References

11. Information on materials and methods for all experiments is available on Science Online.