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# Unconscious Touch Perception After Disruption of the Primary Somatosensory Cortex

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## Abstract
Brain damage or disruption to the primary visual cortex sometimes produces blindsight, a striking condition in which patients lose the ability to consciously detect visual information yet retain the ability to discriminate some attributes without awareness. Although there have been few demonstrations of somatosensory equivalents of blindsight, the lesions that produce "numbsense," in which patients can make accurate guesses about tactile information without awareness, have been rare and localized to different regions of the brain. Despite transient loss of tactile awareness in the contralateral hand after transcranial magnetic stimulation (TMS) of the primary somatosensory cortex but not TMS of a control site, 12 participants (six female) reliably performed at above-chance levels on a localization task. These results demonstrating TMS-induced numbsense implicate a parallel somatosensory pathway that processes the location of touch in the absence of awareness and highlight the importance of primary sensory cortices for conscious perception.

## Keywords
conscious, awareness, sensation, brain, human

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Humans primarily sense the environment through vision and audition but sometimes also use touch to create an accurate representation of the world. For example, we may use somatosensation to navigate in the dark, identify objects in opaque containers, or determine the ripeness of fruit. Although the neural correlates of touch perception are less understood than vision and audition, the primary somatosensory cortex (S1) has been shown to play an important role in conscious tactile perception (Penfield & Rasmussen, 1950). Indeed, lesions specific to S1 (Brochier et al., 1994), as well as transient and reversible disruptions to it with transcranial magnetic stimulation (TMS; Andre-Obadia et al., 1999; Cohen et al., 1991; Pascual-Leone et al., 1994; Seyal et al., 1992), result in the loss of conscious touch perception. Furthermore, tactile illusions in which there are conscious sensations of touch despite the absence of tactile stimulation evoke activity in S1 (Blakemore et al., 2005; Chen et al., 2003; Schwartz et al., 2004; Valenza et al., 2004). Tactile sensations in phantom limbs in patients with amputations also correlate with activity in corresponding somatotopic regions of S1 (Lotze et al., 2001; Roux et al., 2001).

Strikingly, a few patients with damage to somatosensory brain regions, such as S1 or the thalamus, have demonstrated *numbsense*, the ability to unconsciously discriminate some attributes of touch despite the inability to consciously report it. For example, these patients may deny feeling any sensations on the affected regions of their bodies but may nevertheless accurately discriminate the location of a tactile stimulus (Paillard et al., 1983; Rossetti et al., 1995, 2001) or whether it was static or dynamic (Brochier et al., 1994). Other patients without numbsense have demonstrated the ability to unconsciously process other tactile characteristics, including

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vibrotactile frequency (Knecht et al., 1996), stimulus-type categories (Aglioti et al., 1998), and three-dimensional information (Berti, 2002; Berti et al., 1999; Maravita, 1997). These studies suggest distinct neural mechanisms for the unconscious processing of different attributes, but these mechanisms remain poorly understood because of the rarity of numbsense and the variability in the lesions that produce it.

In the current study, we assessed the role of S1 in numbsense by applying single-pulse TMS over S1 during conscious touch perception and unconscious touch-discrimination tasks. After obtaining informed consent, we functionally localized the hand area of the right S1 in 12 neurologically healthy participants who were eligible for TMS (see the Method section and Fig. 1a). Subsequent to localizing S1, we delivered a near-threshold electrocutaneous tactile stimulus to either the left index or the left ring finger on 50% of the trials. No tactile stimulus was delivered on the remaining 50% of the trials, which allowed us to assess using signal detection analyses whether the application of single-pulse TMS may have produced response biases (see Fig. 1b). Participants first reported whether or not they felt the tactile stimulus (i.e., detection task) and then reported or guessed on which finger the tactile stimulus was delivered (i.e., two-choice location-discrimination task). The critical trials for demonstrating numbsense were those on which TMS over S1 suppressed conscious somatosensory perception. Performance on tactile-stimulus localization from these trials was compared with performance on trials on which the TMS over a control site (four of the 12 participants) or TMS over S1 (all 12 participants) did not produce touch suppression.

## Statement of Relevance

Although much of the information bombarding our sensory systems, such as a light breeze on one's arm, often goes unnoticed, it may nonetheless influence our behavior by being unconsciously processed in the brain. In this research, we found that transient and reversible disruption of the primary somatosensory cortex (S1) with transcranial magnetic stimulation (TMS) can induce numbsense, a striking ability in which one can make accurate guesses about tactile information without awareness. These findings provide evidence that brain areas beyond S1, such as subcortical regions and the secondary somatosensory cortex, may play a role in the unconscious processing of numbsense. More importantly, they demonstrate the necessity of primary sensory cortices in perceptual awareness and highlight the importance of early brain areas in consciousness.

## Method

On the basis of the sample sizes, effect sizes, and standard deviations from previous studies on TMS-induced blindsight that used similar designs and dependent variables (i.e., Boyer et al., 2005; Jolij & Lamme, 2005; Ro et al., 2004), we calculated the sample size needed to achieve a power of 80%, which was between four and nine participants. After obtaining informed consent approved by the City University of New York Institutional Review Board, we successfully localized S1 in 12 eligible participants (six female, six male; age: $M$ = 25.8 years, range = 21–34) who completed the main experiment. Four of these 12 participants returned for a second control session with TMS over a site 5 cm posterior to the S1 stimulation site to confirm that stimulation sites outside of S1 would not produce any somatosensory suppression.

### *TMS*

A Magstim Rapid stimulator (The Magstim Company, Whitland, United Kingdom) with a 70-mm figure-eight coil was used to briefly and reversibly disrupt S1. S1 was localized by first determining the location and intensity of TMS that produced visible twitches of the left hand on three out of five trials. After finding the hand area of the right motor cortex, we moved the TMS coil caudally in 0.5-cm increments and adjusted the TMS intensity to 110% of motor threshold until there was suppression of tactile sensations on three out of five trials. For seven of the participants, TMS at 110% of motor-threshold intensity was sufficient to induce tactile suppression, whereas for the remaining five participants, TMS was increased to an average intensity of 117% of motor threshold (range = 115%–119%).

### *MRI*

To confirm that our functional-mapping procedure reliably localized S1, we acquired high-resolution structural MRI scans from four participants using a Siemens Skyra 3.0T whole-body scanner (Siemens Medical Solutions, Malvern, Pennsylvania). We used a magnetization-prepared rapid acquisition with gradient echo (MPRAGE) sequence optimized for gray-matter/white-matter contrast with 0.8-mm-thick sagittal slices and an in-plane resolution of 0.8 mm × 0.8 mm. After assessing tactile detection and tactile discrimination in the main experiment, we digitized each participant's head using a Polhemus Fastrak digitizer (Polhemus, Colchester, Vermont) and coregistered standard anatomical landmarks (i.e., nasion, inion, left and right preauricular points, and vertex), as well as the location of the center of the TMS coil, with their MRI scans using MRIcro (Version 1.40) and MRIreg (Version 0.995; Rorden & Brett, 2000). FreeSurfer (Version 6.0; Dale et al., 1999) was used to

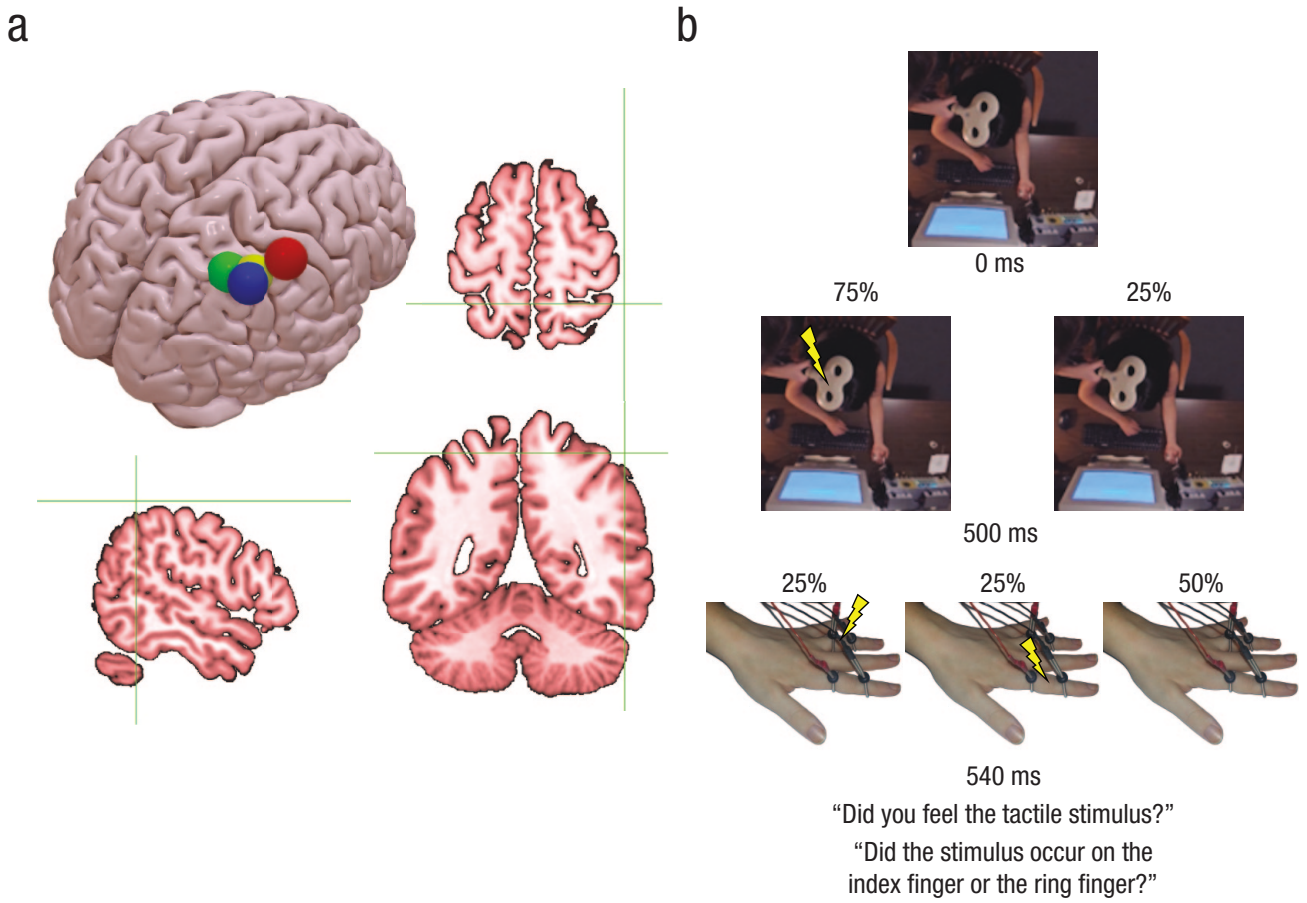


**Fig. 1.** Sites of cortical stimulation and stimuli and procedures. An MRI surface reconstruction of the normalized transcranial magnetic stimulation (TMS) sites (a) is shown for four participants. Each colored spherical node represents the position of the center of the TMS coil for a given participant. The mean axial, sagittal, and coronal position of stimulation, indicated by the intersection of the green lines (Montreal Neurological Institute coordinates: $x = 52.3$, $y = -49.2$, $z = 61.4$, respectively), was over the caudal portion of the primary somatosensory cortex (S1). The timeline and procedures of the experiment are shown in (b). At 500 ms after the start of 75% of the trials, a single TMS pulse (indicated by the lightning bolt) was delivered over S1. On 50% of the trials, a tactile stimulus (indicated by lightning bolts) was delivered to the left index finger or left ring finger 40 ms after the TMS pulse. Participants indicated whether or not they felt the tactile stimulus and then reported or guessed on which finger it was presented.

create a cortical surface reconstruction, and Surf Ice (Version 1.0.20190720; Rorden, 2019) was used to visualize the scalp and cortical surface reconstructions with the site of stimulation during the main experiment.

## *Stimuli and procedures*

Electro-cutaneous tactile stimuli were 0.03-ms square-wave electrical pulses delivered by a Grass Astro-Med electrical stimulator (AstroNova, West Warwick, Rhode Island) through pairs of two ring electrodes. Each pair was attached to the left index and the left ring fingers. Prior to the main experiment, each participant's tactile-intensity threshold for each finger was found using the method of limits, whereby ascending and descending series of tactile-stimulus intensities were delivered until three of five stimuli could be detected and verbally reported. Tactile-stimulus intensity was set at 120% of each finger's respective tactile threshold, and participants rested their supine left hand on a table in front of them for the remainder of the experiment.

Prior to the main experiment, participants completed two practice blocks, one without and then one with TMS. Each participant completed a total of 320 trials, divided into five blocks of 64 trials each, with the order of conditions randomized within each block. On 75% of the trials, a single TMS pulse was applied over S1. On 50% of the trials, a tactile stimulus was delivered to the left index or left ring finger at an interval of 40 ms after the TMS pulse to maximize somatosensory suppression.

## *Data analyses*

We used signal detection analyses to compute a bias-free measure of tactile sensitivity in the TMS-present

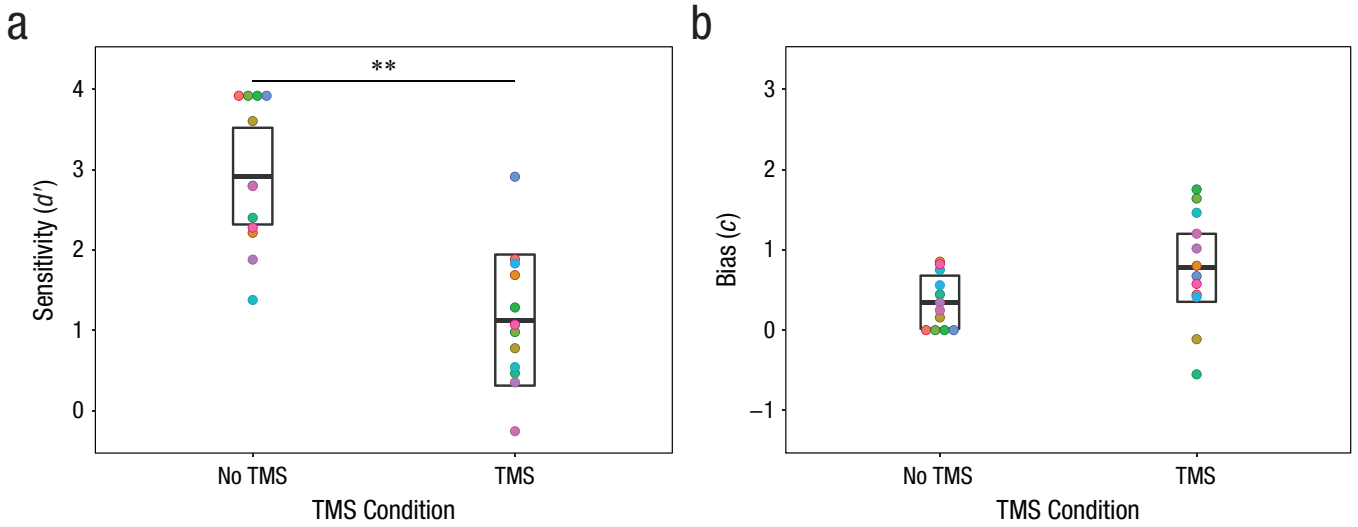


**Fig. 2.** Conscious tactile-detection performance. Tactile sensitivity (a), as measured by bias-free $d'$ rates, is shown separately for trials with and without transcranial magnetic stimulation (TMS) of the primary somatosensory cortex. Response bias (b), as measured by criterion ($c$) values, is shown separately for the TMS and no-TMS conditions. Colored dots are data points for each participant, and the boxplots illustrate the condition means (center lines) and within-participants standard errors (top and bottom edges of boxes). Asterisks indicate a significant difference between TMS conditions ($p < .001$).

and the TMS-absent trials. Paired-samples $t$ tests were used to compare the sensitivity ($d'$) and criterion ($c$) differences between the TMS and no-TMS conditions. To assess the presence of numbsense, we used a one-sample $t$ test to compare accuracy rates for location discrimination on trials in which participants reported not feeling the tactile stimulus with a chance-performance level of 50%.

## Results

When TMS was applied over S1 prior to a tactile stimulus, there was a significant decrease in tactile sensitivity compared with the no-TMS trials, $t(11) = 6.87$, $p < .001$, Cohen's $d = 2.03$, $0.96 \leq d^* \leq 3.1$ (see Fig. 2a; Howell, 2011). Participants reported being unaware of the target tactile stimulus on 55.8% ($SEM = 8.3\%$) of the TMS trials, compared with 18.9% ($SEM = 5\%$) of no-TMS trials. Importantly, this suppression of somatosensory processing after TMS of S1 was due to a change in tactile sensitivity rather than a change in criterion (see Fig. 2b). There was no difference in $c$ values between the TMS trials and the no-TMS trials, $t(11) = 1.83$, $p > .05$, Cohen's $d = 0.8$, $-0.24 \leq d^* \leq 1.84$, indicating that the TMS of S1 was effective at decreasing conscious tactile-perception sensitivity and not at influencing response biases.

To assess the effects of S1 TMS on unconscious tactile discrimination, we calculated the percentage of correct localization responses on TMS trials in which participants reported that they did not feel a tactile stimulus. Despite participants being unaware of the tactile stimulus, performance on the two-choice location-discrimination task was significantly above chance levels, $t(11) = 2.53$, $p < .03$, Cohen's $d = 0.73$, $-0.58 \leq d^* \leq 2.04$ (see Fig. 3). In fact, in some participants, location discrimination was near ceiling levels even though participants were not able to consciously report feeling these stimuli. This result suggests that even in the absence of normal S1 functioning that affects conscious tactile perception, an alternative means for discriminating these tactile stimuli remains available.

Further analyses on discrimination performance using a binomial generalized linear mixed-effects model (GLMM) with participant as a random effect yielded estimated log odds ratios of −2.08 ($SEM = 0.30$) for the fixed effect of TMS ($z = -7.05$, $p < .001$), −2.82 ($SEM = 0.37$) for the fixed effect of detection ($z = -7.72$, $p < .001$), and 2.05 ($SEM = 0.39$) for the TMS × Detection interaction ($z = 5.22$, $p < .001$). This significant interaction likely reflects the smaller change in discrimination performance between detected and undetected trials with TMS than without TMS, as one would expect with the presence of numbsense after TMS disruption of S1. A Bayesian sequential analysis showed that the numbsense effect was moderate after 11 participants (Bayes factor favoring the alternative over the null hypothesis, or $BF_{10} = 5.097$). Ten of the 12 participants had unconscious-discrimination rates numerically above chance levels, and four of these 10 participants showed statistically reliable numbsense in single-participant analyses (all $\chi^2$s $> 9.8462$, all $p$s $< .002$).

To rule out nonspecific effects of the TMS on tactile detection, we measured detection sensitivity following

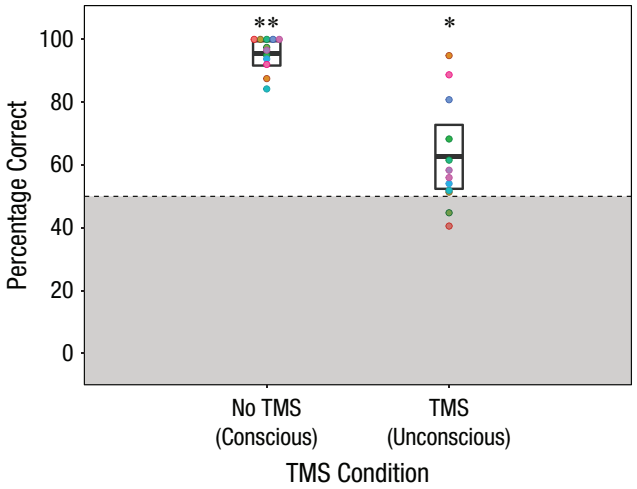


**Fig. 3.** Unconscious location discrimination in transcranial magnetic stimulation (TMS)-induced numbsense. The percentage of correct localization responses is shown separately for no-TMS trials, in which participants reported feeling the tactile stimulus, and TMS trials, in which participants reported not feeling the tactile stimulus. Colored dots are data points for each participant, and the boxplots illustrate the condition means (center lines) and within-participants standard errors (top and bottom edges of boxes). The dashed line marks chance performance (50% correct). Asterisks indicate significant differences between mean performance and chance (*$p < .05$, **$p < .001$).

TMS over a control site that was 5 cm caudal to S1 in four participants who previously participated in the main experiment. As expected (see Fig. 4a), there was no suppression of the tactile stimuli when TMS was located over a region 5 cm caudal to S1. Tactile-sensitivity levels after TMS of this control site were statistically similar to those in the no-TMS conditions, $t(3) = 0.61$, $p = .587$, Cohen's $d = 0.04$, $-0.11 \le d^* \le 0.18$, and significantly greater than when TMS was applied over S1, $t(3) = 3.60$, $p = .037$, Cohen's $d = 0.41$, $0.12 \le d^* \le 0.69$. Because we were able to retest only a minority of the original 12 participants, we also conducted Bayesian statistical analyses to ensure that this smaller sample size was sufficient to rule out any nonspecific effects of the TMS in producing tactile suppression and the associated numbsense results in the main experiment. A Bayesian *t* test comparing whether detection sensitivity on trials with TMS over the control site was any less than on trials without any TMS showed moderate evidence for the lack of a difference (Bayes factor favoring the null hypothesis over the alternative hypothesis, or $BF_{01} = 0.305$). Bayesian sequential analysis showed that this moderate evidence for a lack of a difference was already apparent after the third participant. These expected results demonstrate that TMS suppresses tactile perception when delivered over S1 but not over the posterior parietal cortex.

We also measured above-chance location discrimination on trials in which participants missed the near-threshold tactile stimulus even though there was no TMS. Figure 4b shows that discrimination performance was significantly greater than chance for the no-TMS trials, $t(9) = 3.16$, $p = .012$, Cohen's $d = 0.99$, $-0.52 \le d^* \le 2.52$, demonstrating numbsense even without somatosensory cortical disruption, as has also been shown with blindsight in normal observers (Kolb & Braun, 1995; but see Morgan et al., 1997; Robichaud & Stelmach,

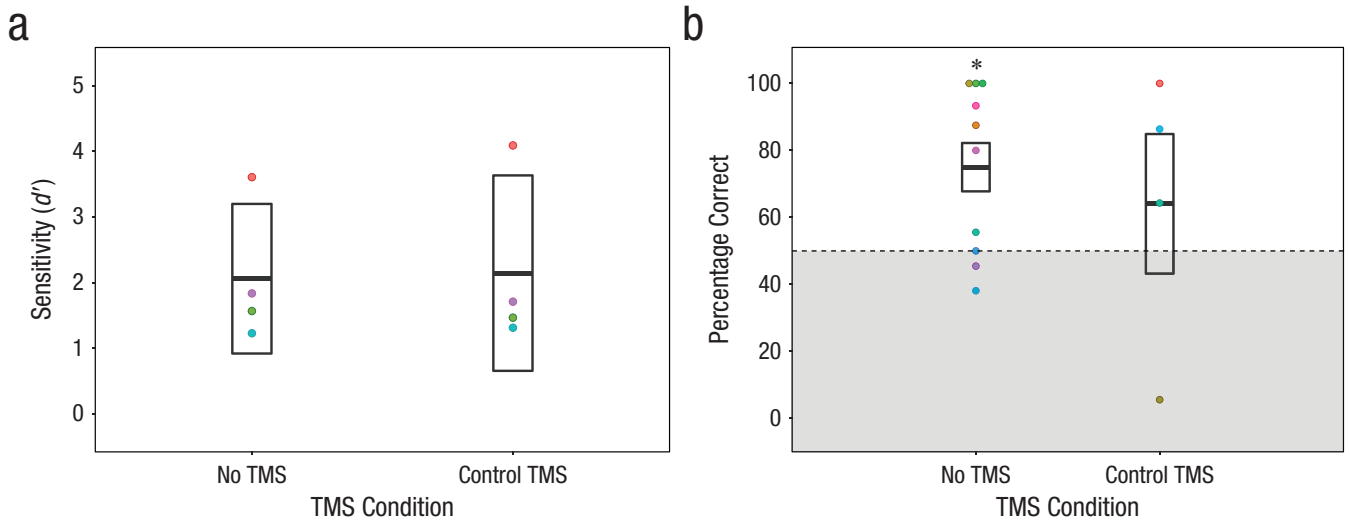


**Fig. 4.** Tactile sensitivity and location discrimination under control conditions. Tactile sensitivity (a), as measured by bias-free $d'$ rates, is shown separately for trials with transcranial magnetic stimulation (TMS) of a control site 5 cm caudal to the primary somatosensory cortex and trials with no TMS. The percentage of correct localization responses (b) for trials on which the participants reported not feeling the tactile stimulus is shown for both the no-TMS and control TMS conditions. Colored dots are data points for each participant, and the boxplots illustrate the condition means (center lines) and standard errors of the mean (top and bottom edges of boxes). In (b), the dashed line marks chance performance (50% correct). The asterisk indicates a significant differences between mean performance and chance ($p < .05$).

2003). When TMS was over the cortical control site that did not produce tactile suppression, discrimination performance was not significantly greater than chance, $t(3) = 0.67$, $p = .549$, Cohen's $d = 0.34$, $-2.87 \leq d^* \leq 3.54$.

## Discussion

These results provide the first demonstration of numbsense in normal participants using the noninvasive brain-stimulation technique of TMS. The disruption of S1 with TMS, but not a control site, caused an inability to consciously detect tactile stimuli on the contralateral hand. These results are consistent with those of previous studies using TMS to disrupt somatosensory perception (Andre-Obadia et al., 1999; Cohen et al., 1991; Pascual-Leone et al., 1994; Seyal et al., 1992) and demonstrate a critical role of S1 in conscious touch perception. These results also add to evidence showing that S1 contributes to somatosensory processing involved in one-interval forced-choice detection tasks (Tamè & Holmes, 2016).

As suggested by findings of TMS-induced blindsight after stimulation of the primary visual cortex (Boyer et al., 2005; de Graaf et al., 2011; Jolij & Lamme, 2005; Ro et al., 2004), the current results demonstrating TMS-induced numbsense suggest alternative somatosensory pathways that process different attributes of touch and highlight the importance of S1 for conscious perception. One candidate pathway that may be responsible for numbsense is a direct projection from the thalamus to the secondary somatosensory cortex (S2) in the parietal operculum. The existence of a thalamocortical pathway projecting from the lateral posterior nucleus of the thalamus to S2 in rats, rabbits, cats, and marmoset monkeys has been extensively described in anatomical studies (Chakrabarti & Alloway, 2006; Kwegyir-Afful & Keller, 2004; Murray et al., 1992; Turman et al., 1992; Zhang et al., 2001). Although the existence of an equivalent pathway has not been demonstrated in humans, neuromagnetic measurements (Karhu & Tesche, 1999; Raij et al., 2008) and disruption of S1 and S2 with TMS (Raij et al., 2008) have shown simultaneous coactivation of S1 and S2 from median nerve stimulation, providing further evidence for parallel processing in the somatosensory cortex.

Another candidate mechanism may involve the posterior parietal cortex. In monkeys, there is a pathway, likely an ascending somesthetic projection, between the lateral posterior nucleus of the thalamus to Areas 5 and 7 of the posterior parietal cortex (Jones et al., 1979; Pearson et al., 1978). Although an analogous pathway has yet to be demonstrated in humans, the role of the posterior parietal cortex, and specifically of the left inferior parietal lobule and the bilateral precuneus, was demonstrated for tactile finger identification (Rusconi et al., 2014). Future studies should aim to measure the differential contributions of these pathways to the unconscious localization of touch.

Importantly, the original reports of numbsense (Paillard et al., 1983; Rossetti, 1998, 1999; Rossetti et al., 1995) demonstrated the loss of touch detection despite the ability to unconsciously point to the location of the stimulus on the body. In these case studies, the ability to verbally report the location of the stimulus was at chance. Brochier et al. (1994), however, demonstrated the loss of touch detection with above-chance verbal reports of location. These different cases highlight a range of residual abilities preserved in numbsense that may rely on different neural mechanisms and are reminiscent of the distinction between action blindsight and perceptual blindsight (Danckert & Rossetti, 2005). Assessing whether pointing responses might be more preserved after disruption of S1 would be an interesting follow-up study that would provide further insights into the neural mechanisms of this TMS-induced numbsense.

Using near-threshold tactile stimuli, we also measured unconscious touch-location discrimination on trials without TMS (see Fig. 4b), similar to results demonstrating blindsight in normal observers (Kolb & Braun, 1995; but see Morgan et al., 1997; Robichaud & Stelmach, 2003). Despite this loss of tactile awareness without TMS, touch discrimination in the two-choice task was significantly higher than chance performance. This may reflect a more general dissociation in perception between detection and discrimination tasks that is independent of the numbsense induced by TMS of S1 (e.g., differences in thresholds for detection vs. discrimination tasks). In fact, the significant TMS × Detection interaction in the GLMM analysis of discrimination performance suggests that these two distinct unconscious-discrimination phenomena may reflect unique neural and perceptual mechanisms. The above-chance unconscious tactile discrimination without TMS may reflect a general unconscious-perception effect that includes S1, whereas the numbsense from S1 disruption with TMS likely reflects neural mechanisms that encode tactile location without S1 and awareness. Further research will be necessary to more precisely determine how unconscious location discrimination without S1 may overlap and differ from unconscious location discrimination in general.

It is important to note that we used a functional-localization procedure to delineate an optimal site in each participant to produce an attenuation in somatosensory sensitivity in the main condition of this experiment. Unlike in studies using structural or functional MRI, this functional-localization procedure provides a more definitive, causal delineation of the somatosensory cortex. Thus, even though our localization procedure positioned the TMS coil in a more caudal and dorsal location than in other studies (Holmes & Tamè,

2018; Holmes et al., 2018), our functional TMS coil-positioning procedure ensured adequate suppression of tactile stimuli for both the index and ring fingers. The MRI scans of four of our participants validated that our coil was positioned over the caudal regions of the post-central gyrus and ensured that the induced current from the TMS was affecting S1.

In conclusion, after we disrupted neural processing in S1 using single-pulse TMS, thereby suppressing tactile awareness of a stimulus on the contralateral hand, participants were nevertheless able to accurately discriminate the location of the stimulus on the hand at significantly above-chance levels. This is the first demonstration of numbsense in normal participants with transient and reversible virtual lesions of S1. These results suggest that somatosensory regions beyond S1 can process location of touch on the body in the absence of conscious awareness.

## Transparency

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*Author Contributions*

The idea for the study was conceptualized by T. Ro and L. Koenig. The methodology was developed by T. Ro. Software was programmed by T. Ro and L. Koenig. Data were collected by L. Koenig. Study materials were prepared by T. Ro; data were cleaned and analyzed by T. Ro and L. Koenig. The project was supervised and administered by T. Ro, who also acquired funding. The manuscript was written by T. Ro and L. Koenig, and the figures were prepared by T. Ro. Both authors approved the final manuscript for submission.

*Declaration of Conflicting Interests*

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

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*Open Practices*

Data and materials for this study have not been made publicly available, and the design and analysis plans were not preregistered.

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