Linking individual differences in human primary visual cortex to contrast sensitivity around the visual field

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A central question in neuroscience is how the organization of cortical maps relates to perception, for which human primary visual cortex (V1) is an ideal model system. V1 non-uniformly samples the retinal image, with greater cortical magnification (surface area per degree of visual field) at the fovea than periphery and at the horizontal than vertical meridian. Moreover, the size and cortical magnification of V1 varies greatly across individuals. Here, we used fMRI and psychophysics in the same observers to quantify individual differences in V1 cortical magnification and contrast sensitivity at the four polar angle meridians. Across observers, the overall size of V1 and localized cortical magnification positively correlated with contrast sensitivity. Moreover, greater cortical magnification and higher contrast sensitivity at the horizontal than the vertical meridian were strongly correlated. These data reveal a link between cortical anatomy and visual perception at the level of individual observer and stimulus location.

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**Results**

The size of V1 varies substantially across observers. First, we assessed the distribution of V1 surface area across 29 observers. Here, we report V1 surface area per hemisphere, from 0° to 8° eccentricity, which comprises almost half of V1. We limit the eccentricity range to 8° because the functional data are less reliable near the edge of the retinotopic mapping stimulus (12.4°) and to match the eccentricity extent of the wedge-ROIs used in the cortical magnification analysis. Consistent with previous reports, the surface area of V1 varied twofold (specifically by 110%, the largest V1 being 1776 mm² and the smallest being 832 mm²) (Fig. 1a). The variability in V1 surface area is substantial, especially when compared to the total surface area of the cortex (Fig. 1b), which varied by 50% (the largest hemisphere of the cortex being 0.12 m² and the smallest being 0.08 m²). Figure 1d shows visualizations of polar angle and eccentricity maps from the left V1 for the largest and smallest V1s. Both had clear, full representations of the right visual hemifield, indicating that the visual field maps were clear and complete, and there were no errors in delineating their boundaries. Instead, the retinotopic representations are simply compressed for the observer with a smaller V1. These large individual differences are not a result of sex differences. There was little difference in the size of V1 between females and males, regardless of whether we normalize the size of V1 to total cortical surface area (p > 0.1 for both comparisons, unpaired two-tailed t tests).

Although V1 surface area varies across individuals, the surface areas of left and right V1 are relatively similar within individuals. Here, too, we found that the surface area of left and right V1 were highly correlated (r = 0.67, p < 0.001, Fig. 1c).

Replicating group-level polar angle asymmetries for contrast sensitivity and V1 surface area. We tested whether the expected polar angle asymmetries existed in the psychophysical and cortical data at the group level. First, to calculate contrast sensitivity along the horizontal meridian, we averaged contrast sensitivity measurements from the left and right horizontal meridians. Similarly, to calculate contrast sensitivity along the vertical meridian, we averaged together contrast sensitivity measurements from the upper and lower vertical meridians. To calculate the amount of V1 surface area dedicated to processing the horizontal meridian, surface area measurements from the wedge-ROIs...
Fig. 1 Variability of human primary visual cortex. a Surface area (mm²) for the left and right hemispheres of V1 (n = 29) and b the total surface area for left and right hemispheres of the cortex (n = 29). The y-axes are matched so that the scaling of the values in b are 100× greater than those in a. Individual data are plotted in red. The horizontal line represents the median. Top and bottom bounds of each box represent the 75th and 25th percentiles, respectively. The whiskers extend to the minima and maxima data points not considered outliers. c The surface area of the left and right hemispheres of V1 are strongly correlated within individuals (two tailed Pearson’s correlation, r₁₄ = 0.67, p < 0.001). d Polar angle and eccentricity maps on the inflated left hemisphere for the individuals with the largest and smallest V1s. The border of V1 is defined by the black lines and data are shown out to 8° of eccentricity. Source data for a, b and c are provided as a Source Data file.

Fig. 2 Group-level polar angle asymmetries for contrast sensitivity and V1 surface area. a Group-level contrast sensitivity measurements from the four polar angle meridians (paired samples t-tests, two sided) and b group-level V1 surface area measurements from wedge-ROIs (±15°) centered on the four polar angle meridians (paired samples t-tests, two sided) (n = 29). Colored data points indicate individual measurements. The black datapoint represents the group average and the colored horizontal line represents the group median. Gray error bars on the horizontal brackets represent ±1 standard error of the difference. Top and bottom bounds of each box represent the 75th and 25th percentiles, respectively. The whiskers extend to the minima and maxima data points not considered outliers. *p < 0.01, **p < 0.001. Source data are provided as a Source Data file.

Additionally, we identified slightly more V1 surface area dedicated to the right than the left horizontal meridian (p = 0.028, d = 0.43). In a supplementary analysis we tested this left-right horizontal meridian asymmetry using an extended dataset centered on the left and right horizontal meridians were summed. Similarly, to calculate the amount of surface area dedicated to processing the vertical meridian, the V1 surface area measurements from the wedge-ROIs centered on the upper and lower vertical meridians were summed. See Methods section Defining wedge-ROIs for further details.

As expected, both the HVA and VMA emerged at the group-level in the contrast sensitivity data. Contrast sensitivity was significantly greater at the horizontal than the vertical meridian (HVA) (t(28) = 15.88, p < 0.001, d = 2.95; paired samples t test; Fig. 2a). Similarly, contrast sensitivity was greater at the lower than the upper vertical meridian (VMA) (t(28) = 4.18, p < 0.001, d = 0.78; Fig. 2a). Both the HVA and VMA were well-correlated across subsampled blocks of the behavioral data from each observer, indicating that the contrast sensitivity measurements were reliable within each observer and supporting the use of contrast sensitivity measures as reflecting genuine individual differences (Supplementary Fig. 1).

Likewise, we confirmed the HVA and VMA at the group level in the V1 surface area data. There was significantly more V1 surface area dedicated to the horizontal than vertical meridian (HVA) (t(28) = 16.40, p < 0.001, d = 3.05; Fig. 2b). Similarly, there was more V1 surface area dedicated to the lower than upper vertical meridian (VMA) (t(28) = 3.30, p = 0.002, d = 0.61; Fig. 2b). Neither the effect of sex nor its interaction with location (HVA or VMA) for contrast sensitivity or surface area was significant (p > 0.1 for main effect of sex and interactions; 2 × 2-way (2 locations × 2 sexes) repeated measures ANOVAs).
(n = 54; the 29 observers here and 25 additional observers for whom we have retinotopy measurements but no psychophysics data) and found the slight bias for more surface area along the right than left horizontal meridian was diminished and became only marginally significant (p = 0.065, d = 0.26; Supplementary Fig. 2).

Overall V1 surface area predicts contrast sensitivity. After confirming the polar angle asymmetries at the group level, we measured the relation between contrast sensitivity and V1 surface area at different scales, across individual observers. The relation between these two variables is non-linear; an increase in V1 surface area does not necessarily correspond to a proportional increase in contrast sensitivity. However, the relation between these two variables is hypothesized to be monotonic and positive; as surface area increases, so will contrast sensitivity. Thus, the following analyses were conducted using one-tailed Spearman’s rho; \( r_p \).

First, we asked whether contrast sensitivity averaged across the four polar angle locations correlated with overall surface area (i.e., the size) of V1 (summed across hemispheres and restricted to 0°–8° of eccentricity). Averaged contrast sensitivity across location was positively correlated with V1 surface area (\( r_p = 0.47, p = 0.005 \); Fig. 3). The surface area of a cortical region depends on the cortical depth used to define the surface. The superficial surface is an overall larger surface area than a surface defined at a deeper layer. Moreover, the superficial layer has greater surface area for gyri and the deeper surfaces have greater surface area for sulci. To reduce these biases, we defined surface area on the midgray surface, half-way between the white matter and the pial surface. We also measured all effects relative to the white matter surface and the pial surface and found a consistent pattern of results; significant correlations were also found when V1 surface area was calculated using the pial (Supplementary Fig. 3a) or white matter surfaces (Supplementary Fig. 3b), rather than the midgray (see Methods: Midgray, pial, and white matter surfaces).

Overall, observers with a larger V1 tended to have greater contrast sensitivity, whereas those with a smaller V1 tended to have relatively lower contrast sensitivity.

When we computed the relation between contrast sensitivity and V1 surface area, we did not normalize V1 surface area by the total cortical surface per observer. In rodents, animals with larger brains have larger neurons, so the number of neurons is approximately constant despite differences in brain size\(^42\). Were this also the case for the human brain, it would be appropriate to normalize each observer’s V1 surface area by their total cortical surface area, as in this case surface area would be a good proxy for neural count. When we normalize by total cortical surface area and then correlate with contrast sensitivity, a positive correlation between the variables remains (\( r_p = 0.33, p = 0.038 \); Fig. S4). Further, average contrast sensitivity did not correlate with overall cortical surface area (p = 0.106), indicating that these correlations are specific to V1.

Local V1 surface area measurements predict contrast sensitivity at the polar angle meridians. Next, we assessed the relation between contrast sensitivity and V1 surface area at a finer granularity. Across observers, we asked whether contrast sensitivity at each polar angle meridian correlated with V1 surface area measurements taken from the spatially corresponding wedge-ROI. Each wedge-ROI was ±15° in width, extended from 1° to 8° eccentricity, and was centered along a polar angle meridian corresponding to the contrast sensitivity measurements. Spearman’s rho was used to assess the relation between contrast sensitivity measurements taken at each of the left and right horizontal, upper, and lower vertical meridians and the surface area of the ±15° wedge-ROI centered on the corresponding meridian.

Across polar angle locations and observers, contrast sensitivity measurements were strongly correlated with the corresponding V1 surface area (\( r_p = 0.60 \); Fig. 4a). Note that this correlation relies on the variability across two factors: polar angle and individual observer. Thus the data points were not independent, with a data point from each observer per polar angle location. To assess whether each of these factors significantly contributed to the correlation, we computed two null distributions (Supplementary Fig. 5). One removed variability across observers and the other removed variability across polar angle. Each distribution was generated by bootstrapping 10,000 Spearman’s rho correlations to the contrast sensitivity and localized surface area measurements. The first null distribution removed variability across observers: on each iteration we shuffled the assignment of the four contrast sensitivities and four local surface area values across observers, while maintaining the tie of a given quadruple of contrast sensitivity and local surface area measurements at each location. The second null distribution removed variability across polar angle: on each iteration we shuffled the assignment of the four contrast sensitivities and four local surface area values across locations, while maintaining the tie of a given quadruple of contrast sensitivity and local surface area measurements to an observer. We then calculated the \( r_p \) values at the 95th percentile (\( x_{0.95} \))\(^43\) of these bootstrapped \( r_p \) distributions.

For the null distributions, the \( r_p \) values at the 95th percentile (\( x_{0.95} \)) were 0.56 (first distribution, removing the effect of individual observer) and 0.24 (second distribution, removing the effect of polar angle). Both of these values are less than the \( r_p \) value of 0.60, obtained from the unshuffled distribution, thereby demonstrating a significant contribution of both polar angle and individual observer to the correlation. A similar correlation between contrast sensitivity and local surface area was found using measurements on the pial surface (Supplementary Fig. 6a) and white matter surface (Supplementary Fig. 6b). Thus, local V1 surface area (or equivalently, cortical magnification) predicted contrast sensitivity measurements taken from different polar angle locations.

To visualize the contribution of variability across polar angle and individual observer, we factored out between-observer variability or within-observer variability from the data. We did this by subtracting the contrast sensitivity/surface area value averaged across the four polar angle locations separately for each observer (Fig. 4b) or by subtracting the contrast sensitivity/surface area...
area value averaged across the 29 observers separately for each of the four polar angles (Fig. 4c). In both cases, a positive correlation remained, supporting the statistical comparisons described above. In particular, these results show a robust effect of polar angle on the data ($r_\rho = 0.78$; Fig. 4b) supporting the statistical comparison, and a modest effect of individual observer variability on the data ($r_\rho = 0.27$; Fig. 4c).

Quantifying the relation between the strengths of the behavioral and cortical HVA and VMA. Next, we calculated a summary metric to describe the strength of the HVA and VMA for contrast sensitivity and local surface area. We then assessed the relation between the strength of behavioral and cortical HVA and VMA across observers.

For each observer, we calculated an asymmetry index for the HVA. The HVA index was calculated as the difference in contrast sensitivity or local V1 surface area between the horizontal and vertical meridian, divided by the mean of the two, multiplied by 100.

$$HVA = \frac{(\text{horizontal} - \text{vertical})}{\text{mean(horizontal, vertical)}} \times 100 \quad (1)$$

An HVA index of 0 indicates no difference in contrast sensitivity or surface area between the horizontal and vertical meridian. As the HVA index increases, the asymmetry increases, with greater contrast sensitivity, or surface area, at the horizontal than vertical meridian.

Correspondingly, the VMA index was calculated as the difference in contrast sensitivity or local V1 surface area between the lower and upper vertical meridian, divided by the mean of the two, multiplied by 100.

$$VMA = \frac{(\text{lower vertical} - \text{upper vertical})}{\text{mean(lower vertical, upper vertical)}} \times 100 \quad (2)$$

A VMA index of 0 indicates no difference in contrast sensitivity or surface area between the lower and upper vertical meridian. As the VMA index increases, the asymmetry increases, with greater contrast sensitivity, or surface area, at the lower than upper vertical meridian.

Importantly, these HVA and VMA strengths reflect differences in contrast sensitivity, or V1 surface area, between locations, after dividing out the mean contrast sensitivity or local V1 surface area, per observer. Therefore, one could have low contrast sensitivity measurements, but a strong HVA.

Spearman’s rho was used to assess the relation between the HVA index for contrast sensitivity and local V1 surface area across observers. There was a significant, positive correlation.
between the two measurements ($r_p = 0.60, p < 0.001$; Fig. 5a). A similar correlation was found when the wedge-ROIs were used to calculate V1 surface area measurements on the pial (Supplementary Fig. 7a) and white matter surfaces (Supplementary Fig. 7c). Thus, observers with a stronger asymmetry in contrast sensitivity measurements between the horizontal and vertical meridians had a stronger asymmetry in dedicated V1 surface area between the horizontal and vertical meridians.

Following this, Spearman’s rho was used to test the relation between the VMA index for contrast sensitivity and local V1 surface area. There was a non-significant correlation between the two measurements ($r_p = −0.30, p = 0.942$; Fig. 5b). Thus, the individual asymmetry for contrast sensitivity between the lower and upper vertical meridian was not associated with the individual amount of V1 surface area dedicated to the lower and upper vertical meridian. There was also no systematic relation when the local V1 surface area measurements were made on the pial surface (Supplementary Fig. 7b) and white matter surface (Supplementary Fig. 7d). In a supplemental analysis, we found no significant correlation between the behavioral HVA and VMA (Supplementary Fig. 8a) and only a marginal correlation between the cortical HVA and VMA (Supplementary Fig. 8b).

**Discussion**

We quantified the relation between contrast sensitivity and V1 surface area at a global scale (i.e., the surface area of V1 itself) and a local scale (i.e., the local surface area of V1 processing the polar angle meridians) across 29 observers. We confirmed group-level polar angle asymmetries in the contrast sensitivity and V1 surface area data. We then quantified individual differences across observers, leading to three major findings. First, contrast sensitivity averaged across the four polar angle locations positively correlated with the size of V1. Second, contrast sensitivity measurements taken at the four polar angle locations positively correlated with localized V1 surface area measurements taken from the spatially corresponding polar angle meridian in the visual field. Third, the extent of the HVA for contrast sensitivity was correlated with the extent of the HVA for V1 surface area, whereas the VMA was not.

**Group-level reproduction of polar angle asymmetries.** The data showed clear group-level polar angle asymmetries for contrast sensitivity and V1 surface area. Contrast sensitivity was roughly 50% higher for the horizontal than vertical meridian and 20% higher for the lower than upper vertical meridian. Likewise, the V1 surface area measurements also showed group-level polar angle asymmetries; V1 surface area was around 60% greater along the horizontal than vertical meridian and around 25% greater along the lower than upper vertical meridian, consistent with data from our previous study, the Human Connectome Dataset and other work. Polar angle asymmetries have also been found in the surface areas of non-human primate V1, in the amplitude of the BOLD response in human V1, and in spatial frequency preference in human. The existence of these polar angle asymmetries in multiple, large datasets for both the behavioral and cortical data indicates that they are robust at the group-level, and speaks to the high level of reproducibility reported for fMRI-derived retinotopic maps.

The present group-level data showed a small bias towards more V1 surface area dedicated to the right than left horizontal meridian. It might be that a left-right horizontal meridian asymmetry relates to visual tasks in which an advantage along the right horizontal meridian exists, such as crowding and letter recognition. A larger left than right hemisphere of V1 has been previously reported but see refs. 2,28. However, none of these studies examined the surface area of the horizontal meridian specifically.

**Variation in the size of V1 surface area across observers.** The surface area of V1 varied substantially across observers, whereas within observers, the surface area of V1 in the left and right hemispheres was relatively consistent, in line with previous studies. V1 size is only weakly correlated with overall cortical surface area, which is less variable in size, as shown here and in prior reports. Neither does V1 size differ between males and females after normalization to total cortical surface area, again shown here and elsewhere. Why is there so much variability in the size of V1? One hypothesis is that variation in the size of V1 depends on the amount of detail encoded in earlier stages of the visual system: cone density varies by about threefold across observers and the size of the LGN and optic tract also vary substantially. Indeed, the size of V1 correlates with the size of the lateral geniculate nucleus and the optic tract, suggesting that these components of the early visual system, all of which are important for visual perception, develop interdependently. This finding lends credence to the possibility that the size of V1 is important for perceptual tasks.

**Greater contrast sensitivity is a perceptual consequence of greater V1 surface area.** Here, we have shown that contrast sensitivity measurements derived from an orientation discrimination task positively correlate with V1 surface area;
observers with greater contrast sensitivity tend to have a larger V1 and those with lower contrast sensitivity tend to have a smaller V1. This relation holds irrespective of the cortical depth used to compute surface area (gray/pial boundary, gray/white boundary, or half-way between them). V1 surface area has been shown to correlate with a few measurements of visual performance, such as perceptual acuity thresholds $^{35,56}$, measurements of subjective object size $^{52}$, and orientation discrimination thresholds $^{57}$, but not with contrast discrimination thresholds $^{57}$. Such thresholds are different from those measured here; they depend upon the range of the contrast response function being measured $^{38,59}$, but not upon stimulus orientation. Nonetheless, performance on most visual tasks has not been compared to V1 size and there is not yet a computational account that would enable one to predict to what extent, if any, performance on different tasks would be affected by V1 size.

We focused on the relation between performance on one visual measure—contrast sensitivity—and the size of the one cortical map—V1. Although V1 size has been linked to a few perceptual measures $^{52,55-57,60-62}$, it is likely that there will be other measures for which performance is better explained by the size of other visual maps. Such an outcome is possible because the sizes of different maps are at least partially independent $^{2,4}$. For example, an observer might have a large V1 and high contrast sensitivity, but a small hV4 and poor performance on visual crowding tasks $^{63}$.

An open question is whether and how the variation in the size of V1 relates to neural circuitry. Smaller V1s have a full, but relatively compressed representation of the visual field. Does this compression represent fewer overall neurons or is neural count similar across individuals and instead this compression represents increased neural density? The fact that performance on some tasks correlates with V1 surface area $^{52,55-57}$ suggests that a smaller V1 likely has fewer overall neurons, but the histological measures to directly assess this do not yet exist. Differences in V1 neural counts among individuals and across polar angle raise the question of how the neural code varies across individuals and visual field location. One interesting observation is that the size of V1 is inversely correlated with the size of its population receptive fields (pRFs), suggesting that a larger V1 enables finer sampling of visual space $^{64}$.

**Local contrast sensitivity is linked to local V1 cortical magnification across the visual field.** Virsu and Rovamo $^{36}$ hypothesized that the mechanism underlying contrast sensitivity is a central integrator that pools the activity of V1 neurons; contrast sensitivity should increase in proportion to local cortical surface area (i.e., cortical magnification) and thus the number of neurons activated by a visual stimulus. This hypothesis was derived from group-level behavioral measurements taken as a function of eccentricity. We have tested whether this hypothesis holds for individual, localized V1 surface area measurements taken as a function of polar angle, as well as for individual measurements of the size of V1. We found that observers with more local cortical surface area dedicated to processing some polar angle location had greater contrast sensitivity at the corresponding location, and that individuals with larger V1 had overall higher contrast sensitivity. Therefore, our data support the hypothesis that contrast sensitivity varies as a function of the number of stimulated visual neurons.

Perceptual measurements have been related to the surface area of entire visual maps $^{52,55-57}$. Advances in computational neuroimaging provide the tools to precisely delineate visual maps and assess their internal layout $^{85}$, enabling the assessment of the relation between cortical anatomy and performance as a function of location in the visual field. Indeed, three studies have related individual differences in localized measurements of cortical magnification to perceptual outcomes. Local V1 cortical magnification positively correlates with visual acuity measured as a function of eccentricity $^{35}$, position discrimination ability at different angular locations $^{35}$, and subjective object size for different visual field quadrants $^{60}$.

**Performance field asymmetries.** Perceptual polar angle asymmetries (i.e., performance fields) have been established across a broad range of visual tasks. The HVA and VMA are found in tasks involving contrast sensitivity $^{12,22,23,63-73}$, perceived contrast $^{74}$, spatial resolution $^{24,75,76}$, crowding $^{49}$, temporal information accrual $^{77}$, illusory motion perception $^{25}$, and visual short term memory $^{78}$. Further, these polar angle asymmetries are pervasive across a range of conditions; they exist across luminance levels $^{22}$, binocular and monocular stimulation $^{22,24}$, different stimulus orientations $^{22,68,73}$ and sizes $^{12}$, eccentricities and spatial frequencies $^{12,22-24,68}$, number of distractors $^{22,27,77}$, and covert attentional conditions $^{22,25,27}$.

We found no relation between the behavioral HVA and VMA, consistent with previous reports $^{12,24}$, and only a weak, non-significant relation between the cortical HVA and VMA. These results suggest that the HVA and VMA are independent of each other at the level of perception and at the level of the cortex. It is likely that these two behavioral asymmetries develop with age independently $^{79}$ and may have separate neural substrates. Cortical magnification changes as a function of eccentricity in a similar fashion for children and adults $^{80}$; However, perceptual polar angle asymmetries vary between children and adults $^{81}$ and how cortical magnification changes as a function of polar angle in children still needs to be determined. Furthermore, the HVA, but not VMA, exists in photoreceptor cone density $^{82,83}$, whereas both the HVA and VMA exist in retinal midget ganglion cell density $^{84,85}$. A computational model has shown that the HVA and VMA for contrast sensitivity cannot be fully explained by these retinal factors. Asymmetries in optics and cone sampling only accounted for a small fraction of contrast sensitivity asymmetries $^{86}$. Including midget retinal ganglion cells in the model explained a larger fraction of the contrast sensitivity asymmetries but did not account for the extent of the asymmetries reported in human visual behavior $^{87}$. Thus, these retinal asymmetries did not account for perceptual polar angle asymmetries at the group level. It is unlikely that retinal asymmetries could account for individual polar angle asymmetries.

**Individual differences in perceptual polar angle asymmetries are rooted in individual variation in cortical anatomy.** We have shown that the HVA for contrast sensitivity can be predicted from the cortical HVA in individual observers. Thus, the perceptual asymmetry between the horizontal and vertical meridian for contrast sensitivity is strongly reflected by the relative distribution of V1 surface area between the horizontal and vertical meridian. The findings that the behavioral and cortical HVA can be linked across individual observers, and is stronger in the visual cortex than the retina $^{10,87}$, suggest that this perceptual asymmetry can be predominantly explained by the asymmetric distribution of cortical surface (and thus neurons) in V1. Here, we found group-level VMA measurements for contrast sensitivity and surface area, and it has been shown that group-level VMA measurements for spatial acuity thresholds and V1 surface area correlate $^{40}$. However, we did not find a relation between the VMA for contrast sensitivity and the cortical VMA at the level of individual observers. Why might this be? One possibility is statistical power. The VMA was computed using half
Extending the link between brain and behavior. Here, we have linked V1 cortical magnification to contrast sensitivity for a particular stimulus configuration (a $3^\circ$ vertically oriented Gabor with a spatial frequency of 4 cpd centered at 4.5$^\circ$ eccentricity on a uniform gray background). Would these results generalize to other orientations, spatial frequencies, and stimulus sizes? Perceptual polar angle asymmetries are robust across modulations of stimulus content for which V1 neurons are tuned. They persist across different stimulus orientations, sizes, eccentricities and spatial frequencies, and in the presence of distractors. Likewise, cortical polar angle asymmetries have been reproduced across several independent datasets that differ in their experimental design, including differences in the pRF stimulus carrier image. The cortical asymmetries are robust to experimental differences because they rely on polar angle pRF measurements that have shown to be highly reproducible across retinotopy experiments. As these behavioral and cortical asymmetries are preserved across an array of stimulus conditions, we predict that the link between brain and behavioral measurements would also be preserved, albeit with modulations to the strength of the correlations.

What other visual properties might correlate with cortical magnification around the visual field? It is likely that properties for which perceptual polar angle asymmetries exist, and for which V1 neurons are tuned, could also correlate with cortical magnification; for example, acuity and spatial frequency preference.

We have quantified the relation between contrast sensitivity and V1 surface area, measured as a function of polar angle, across the same observers. Our data showed that: First, observers with greater contrast sensitivity tended to have a larger V1, and vice versa. Second, local contrast sensitivity can be predicted by local V1 surface area using measurements taken from the polar angle meridians. Third, a stronger horizontal-vertical asymmetry in contrast sensitivity correlated with the corresponding asymmetry in the distribution of local V1 surface area. The vertical meridian asymmetry in contrast sensitivity did not correlate with the corresponding asymmetry in the distribution of local V1 surface area, likely due to fMRI measurement constraints. Overall, these findings show that individual differences in contrast sensitivity can be linked to individual differences in V1 surface area at global and local scales and reveal that perceptual polar angle asymmetries are rooted in the cortical anatomy of V1. More broadly, our findings show that there is a tight link between visual perception and the idiosyncratic organization of V1.
The pRF stimulus, MRI, and fMRI acquisition parameters, MRI and fMRI preprocessing, and the implementation of the pRF model are identical to those in our prior work⁹. The retinotopic data for 17 of the 29 observers reported here are the same as reported in that study.

**fMRI stimulus display.** Observers viewed a pRF stimulus from inside the MRI scanner bore using a ProPixx DLP LED Projector (VPixx Technologies Inc., Saint-Bruno-de-Montarville, QC, Canada). The pRF stimulus was projected onto an acrylic back-projection screen (60 cm × 36.2 cm) within the scanner bore. The projected image had a resolution of 1920 × 1080 and a refresh rate of 60 Hz. The display was calibrated using a linearized lookup table and the display luminance was 500 cd/m². Observers viewed the screen at a distance of 83.5 cm (from eyes to the screen) using an angled mirror that was mounted on the head coil.

The pRF stimulus consisted of image patterns presented within a bar aperture that swept across the screen for the duration of each scan. The image patterns consisted of colorful objects, faces, and scenes at multiple scales that were superimposed on an achromatic pink noise (1/f) background⁹⁰,⁹¹. The image pattern was windowed within a circular aperture that had a radius of 12.4°. The image pattern was revealed through a bar aperture (3.1° wide, or 1/8th of the full stimulus extent) that swept across the screen in 24 equal steps (once per second). Each step was synchronized to the MR image acquisition (TR 1 s). There were eight sweeps in total. Each sweep began at the edge of the circular aperture. Horizontal and vertical sweeps covered the entire diameter of the circular aperture. Diagonal sweeps only traversed half of the circular aperture; the second half of these sweeps were replaced with a blank gray screen. Each directional sweep lasted 24 s. The full stimulus run lasted 192 s. The stimulus image updated three times per second without intermediate blanks (3 Hz).

The bar aperture was superimposed on a polar fixation grid placed upon a uniform gray background, with a red or green dot at the center (3 pixels, or 0.07°). Fixation (1000 ms) 

Observers were instructed to maintain fixation throughout the entire scan and completed a fixation task in which they were required to respond, using a button box, when the fixation dot changed from green to red, or vice versa.

**Anatomical and functional data acquisition.** Anatomical and functional data were acquired on a 3T Siemens MAGNETOM Prisma MRI scanner (Siemens Medical Solutions, Erlangen, Germany) using a Siemens 64-channel head coil. A T1-weighted (T1w) MPRAGE anatomical image was acquired for each observer (TR, 2400 ms; TE, 2.4 ms; voxel size, 0.8 mm³ isotropic; flip angle, 8°). This anatomical image was auto-aligned to a template to ensure a similar slice prescription for all observers. Between 4 and 12 (9× had 12 scans, 2× had 11 scans, 2× had 10 scans, 5× had 8 scans, 1× had 7 scans, 8× had 6 scans, 1× had 5 scans, and 1× had 4 scans) functional echo-planar images (EPIs) were acquired for each observer using a T²*-weighted multiband EPI sequence (TR, 1000 ms; TE, 37 ms; voxel size, 2 mm³; flip angle, 68°; multiband acceleration factor, 6; phase-encoding, posterior-anterior)¹⁰⁰,¹⁰¹. Two distortion maps were also acquired to correct susceptibility artifacts. The full stimulus sequence was completed once per functional scan. The identical aperture sequence was shown in each of the scans.

**pRF stimulus.** Retinotopic maps were measured using pRF mapping⁹⁰. The pRF stimulus was generated on an iMac computer using MATLAB 2017a and was projected onto the fMRI stimulus display in the scanner bore using the Psychophysics Toolbox v3⁹² and custom visalisp software⁹⁰.

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distortions in the functional images: one spin-echo image with anterior-posterior (AP) phase encoding and one with posterior-anterior (PA) phase encoding.

Preprocessing of structural data. fMRIPrep v2.0.1\textsuperscript{19,20} was used to pre-process anatomical and functional data. For each observer, the T1w anatomical image was corrected for intensity inhomogeneity and then skull stripped. The anatomical image was automatically segmented into cerebrospinal fluid, cortical white matter, and cortical gray matter using fast\textsuperscript{194}. Cortical surfaces were reconstructed using FreeSurfer's recon-all\textsuperscript{195} and an estimated brain mask was refined using a custom version of the method.

Preprocessing of functional data. The following preprocessing was performed on each observer’s functional data. First, a reference volume (and a skull stripped version) was created using custom methodology of fMRIPrep. The two spin echo images with opposing phase-encoding directions (i.e., AP and PA distortion maps) were used to create a nonuniformity map. Nonuniformity was estimated by filtering each functional image with a 2D-Gaussian. The 2D-Gaussian was multiplied pointwise by the stimulus contrast aperture and was standard deviation of the 2D-Gaussian, specifying a contrast map. These maps were then summed across all TRs and the nonuniformity map was then used to generate a corrected functional reference image. This corrected functional reference was co-registered to the anatomical image using six degrees of freedom.

Next, head-motion parameters with respect to the functional reference were estimated before any spatiotemporal filtering. Each functional image was slice-time corrected with all slices realigned to the middle of each TR. The slice-time corrected functional data were then resampled to the T1w anatomical space via a one-shot interpolation consisting of all the pertinent transformations (i.e., head-motion transform matrices, susceptibility distortion correction). These preprocessed time-series data were then resampled to the fsaverage surface by averaging across the cortical ribbon.

Implementing the pRF model to produce retinotopic maps. The pRF model was implemented on the fsaverage surface of each observer. For each fsaverage vertex, the time-series data across each functional scan were averaged together to generate an average time series. These average time-series were then transformed to BOLD percent signal change (i.e., % change at each TR from the mean signal across all TRs). The pRF model was fit to the BOLD signal change.

The pRF model was implemented using visalab\textsuperscript{196} (https://vistalab.stanford.edu/software/, Vista Lab, Stanford University) and customized code to run the model on the cortical surface. A pRF was modeled as a circular 2D-Gaussian that was parameterized by values for $x$, $y$, and $\sigma$. The $x$ and $y$ parameters specify the center position of the 2D-Gaussian in the visual field, whereas the $\sigma$ parameter, the standard deviation of the 2D-Gaussian, specifies the size of the receptive field. The 2D-Gaussian was multiplied pointwise by the stimulus contrast aperture and was then convolved with a hemodynamic response function (HRF) to predict the BOLD percent signal change. We parameterized the HRF by five values, describing a difference of two gamma functions\textsuperscript{96,106,207}.

The pRF model was implemented using a coarse-to-fine approach to find the optimal $x$, $y$, and $\sigma$ for each vertex by minimizing the residual sum of squares between the predicted time-series and BOLD signal\textsuperscript{196}. The $x$ and $y$ values were then used to calculate eccentricity and polar angle coordinates, reflecting the pRF center position in the 3D field. All analyses were completed using data with $r^2 > 10\%$.

Defining V1. V1 was defined as a region-of-interest (ROI) by hand using Neurospy\textsuperscript{11.9} (https://github.com/noahbenson/neurospy\textsuperscript{108}). We defined V1 from 0° to 8° eccentricity with the V1/V2 dorsal border falling through the center of the lower vertical meridian, and the V1/V2 ventral border falling through the center of the upper vertical meridian.

Cortical magnification as a function of polar angle analysis. To calculate local measurements of V1 surface area along the polar angle meridians, we defined ±15° wedge-ROIs that were centered along each of the four polar angle meridians in the visual field. We measured the amount of V1 surface area within these wedge-ROIs\textsuperscript{8,7}. The wedge-ROIs were defined by setting each meridian ±15° of visual space. Any differences in the amount of localized V1 surface area, and white matter surface. These maps are generated using FreeSurfer. The main analyses were conducted using the midgray cortical surface. The supplemental analyses used pial and white matter cortical surfaces. This is because the surface area changes as a function of cortical depth. The surface area of gyri at the pial surface and the sulci at the white matter surface tend to be large, whereas the reverse (sulci at the pial surface and gyri at the white matter surface) are smaller.

Summary to relate psychophysical and fMRI analyses. Figure 7 summarizes how the contrast sensitivity measurements relate to the wedge-ROIs measurements of local V1 surface area. Contrast sensitivity was measured at three different depths—midgray surface, pial surface, and white matter surface. These maps are generated using FreeSurfer. The main analyses were conducted using the midgray cortical surface. The supplemental analyses used pial and white matter cortical surfaces. This is because the surface area changes as a function of cortical depth. The surface area of gyri at the pial surface and the sulci at the white matter surface tend to be large, whereas the reverse (sulci at the pial surface and gyri at the white matter surface) are smaller.
measurements to be an exact measurement of the amount of V1 surface area dedicated to processing the Gabor stimulus itself (i.e., a 3° × 3° patch at 4.5° of eccentricity in V1 at each polar angle meridian). Measurements of cortical magnification are noisy, and the more data included in the calculation of the surface area of the wedge-ROI, the more accurate the output value. We chose to measure the surface area of wedge-ROIs ±15° of width in angle and extending out ±8° of eccentricity when the Gabor patches in the visual field extend ±1.5° either side of each polar angle meridian and were centered at 4.5° of eccentricity. This is because there is a tradeoff in the size of the wedge-ROI and the accuracy of cortical magnification measurements (especially along the vertical meridian where data is comparatively sparse when compared to the horizontal meridian)\(^9\).

**Reporting summary.** Further information on research design is available in the Nature Research Reporting Summary linked to this article.

**Data availability**

Source data are provided with this paper. The data generated for this study have been deposited in the OSF repository https://osf.io/de7zg. Source data are provided in this paper.

**Code availability**

Scripts used for data collection and analysis code to generate manuscript figures are available in the OSF repository at https://osf.io/de7zg and on GitHub at https://github.com/WinawerLab/vistadisp.

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