6. Warren, J.E., Wise, R.J., and Warren, J.D. (2005). Sounds do-able: auditory-motor transformations and the posterior temporal plane. Trends Neurosci. *28*, 636–643.
7. Hickok, G., and Poeppel, D. (2007). The cortical organization of speech processing. Nat. Rev. Neurosci. *8*, 393–402.
8. Foxton, J.M., Dean, J.L., Gee, R., Peretz, I., and Griffiths, T.D. (2004). Characterization of deficits in pitch perception underlying 'tone deafness'. Brain *127*, 801–810.
9. Hyde, K.L., Peretz, I., and Zatorre, R.J. (2008). Evidence for the role of the right auditory cortex In fine pitch resolution. Neuropsychologia *46*, 632–639.
10. Mandell, J., Schulze, K., and Schlaug, G. (2007). Congenital amusia: an auditory-motor feedback disorder? Restor. Neurol. Neurosci. *25*, 323–334.

Newcastle University Medical School, Framlington Place, Newcastle upon Tyne, NE2 4HH, UK.
E-mail: t.d.griffiths@newcastle.ac.uk

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Numerosity Perception: How Many Speckles on the Hen?

How do we tell how many objects there are in a visual scene? A recent study has shown that the numerousness of objects is a 'primary visual property' of the scene, just like the objects' colour, shape or location.

Brian Butterworth

It has long been known that we can 'see' how many objects there are up to about four without counting; for more than four, some kind of sequential enumeration process was assumed to determine the number [1,2]. We can, however, also make an estimate. In a study published recently in *Current Biology*, Burr and Ross [3] show our estimates are based on "a primary visual property of the scene" that can be radically modified by visual adaptation.

Suppose you glance at a speckled hen, and notice that it has quite a lot of speckles. You have the impression that each speckle has a definite shape and a definite location in space, but your glance leaves no time to count them. Does your percept have a definite number of speckles, even though you don't know the exact number? This is a problem that has troubled philosophers since it was first formulated by A.J. Ayer in 1940 [4]. Ayer's solution was that the percept (sense data) does not have a definite number unless you actually enumerate the speckles. This is unsatisfactory. The hen has a definite number of speckles, and one could count, say, exactly 48 in a photo that corresponded to your percept. But on Ayer's account, there are not 48 speckles in your percept, nor 47 nor 49, nor any other number [5]. This is rather like saying that there will be a test next week, but on no particular day. Nevertheless, we might still have an estimate of the number of speckles — for example, that there are more than 10 but less than 100. The question then becomes, how is this achieved.

Burr and Ross [3] have produced a remarkable new demonstration that the numerousness of the speckles is just as much a 'primary visual property' of a scene as their location, their colour, their size, their spatial frequency or their orientation: "just as we have a direct visual sense of the reddishness of half a dozen ripe cherries so we do of their sixishness". Like other primary visual properties, numerousness is susceptible to adaptation. In the new experiments, an adapting patch viewed for 30 seconds with a large number of spots (rather than speckles) made the test patch which followed seem to have fewer elements. The size of the effect is extraordinary. After an adaptor of 400 dots, the 'point of subject equality' (PSE) was three times as great as for the control: that is, the test needed three times as many dots to be regarded as numerous as the probe. Control experiments that manipulated the dot size and the contrast in the adaptor scarcely affected the PSE, indicating that it is indeed numerosity that is being adapted.

This discovery has implications beyond the narrow confines of visual psychophysics, for it provides evidence that the human brain is set up to extract the numerosity parameter from a visual scene, just as it extracts colour. One implication of this, though not one Burr and Ross [3] mention, is that we are born with this capacity.

This is not uncontroversial. On the one hand, Starkey and Cooper [6] and many others (for example [7–9]) have shown that infants appear to respond discriminatively to the numerosity of visual arrays, even to quite large arrays of dots provided the ratio difference is large enough [10]. On the other hand, Mix *et al.* [11] claim that "infants start out with a sensitivity to approximate quantity based on overall amount" (p45) not numerosity, and some studies show that infants respond to the total area covered by the dots, rather than numerosity, when the two are in conflict [12]. In Burr and Ross's [3] adults, the control for overall amount rules this out as an explanation.

The remarkable adaptation effect reported by Burr and Ross [3] can be seen in the on-line demonstration. This uses an adapting patch in each visual hemifield followed by an identical test patch in the corresponding spatial locations. Strikingly, the test patch that follows the more numerous adaptor seems to have fewer dots than its identical counterpart, without any particular dots seeming to have disappeared. So how can the apparent numerosity of the dots be decreased without annihilating any of them?

Burr and Ross's [3] proposed solution is that the visual system does not record speckles on the hens, but rather extracts a statistical description of the speckledness of the scene. This is in line with the 'sparse coding' hypothesis that, although we have the impression of great richness and detail, our conscious percept records only the important features and then fills in the rest [13]. This makes intuitive sense. The hen will have a definite number of speckles, and we can count them if we want to, but if we don't, there is no point making each and every one of them available for further cognitive processing.

This leaves two problems. First, what is the mechanism that constructs a statistic describing the

speckledness of the hen? It cannot simply take an average of some visual property of the scene (as is the case with orientation [14]) without first normalising the size and shape of each speckle. Second, which brain system implements this mechanism? Burr and Ross [3] cite evidence that the intraparietal sulcus responds to the number of objects in a display [15] even when the total continuous extent of the objects is taken into account [16]. But the intraparietal sulcus represents numerosity quite abstractly: independently of whether the objects are distributed in space or in time [16] and independently of modality [15]. Because the adaptation phenomenon described here is retinotopic, earlier stages in neural visual processing are implicated as well.

References

1. Jevons, W.S. (1871). The power of numerical discrimination. Nature *3*, 363–372.
2. Kaufman, E.L., Lord, M.W., and Reese, V.J. (1949). The discrimination of visual number. Am. J. Psychol. *62*, 498–525.
3. Burr, D., and Ross, J. (2008). A visual sense of number. Curr. Biol. *18*, 425–428.
4. Ayer, A.J. (1940). The Foundations of Empirical Knowledge (London: Macmillan & Co Ltd).
5. Chisholm, R. (1942). The problem of the speckled hen. Mind *LI*(204), 368–373.
6. Starkey, P., and Cooper, R.G., Jr. (1980). Perception of numbers by human infants. Science *210*, 1033–1035.
7. Antell, S.E., and Keating, D.P. (1983). Perception of numerical invariance in neonates. Child Devel. *54*, 695–701.
8. Van Loosbroek, E., and Smitsman, A.W. (1990). Visual perception of numerosity in infancy. Dev. Psychol. *26*, 916–922.
9. Wynn, K., Bloom, P., and Chiang, W.C. (2002). Enumeration of collective entities by 5-month-old infants. Cognition *83*, B55–B62.
10. Xu, F., and Spelke, E. (2000). Large number discrimination in 6-month-old infants. Cognition *74*, B1–B11.
11. Mix, K.S., Huttenlocher, J., and Levine, S. (2002). Quantitative Development in Infancy and Early Childhood (New York: Oxford University Press).
12. Feigenson, L., Spelke, E., and Carey, S. (2002). Infants' discrimination of number vs. continuous extent. Cogn. Psychol. *44*, 33–66.
13. O'Regan, J.K., and Noë, A. (2001). A sensorimotor account of vision and visual consciousness. Behav. Brain Sci. *24*, 939–973.
14. Parkes, L., Lund, J., Angelluci, A., Solomon, J., and Morgan, M.J. (2001). Compulsory averaging of crowded orientation signals in human vision. Nat. Neurosci. *4*, 739–744.
15. Piazza, M., Mechelli, A., Butterworth, B., and Price, C. (2002). Are subitizing and counting: implemented as separate or functionally overlapping processes? NeuroImage *15*, 435–446.
16. Castelli, F., Glaser, D.E., and Butterworth, B. (2006). Discrete and analogue quantity processing in the parietal lobe: A functional MRI study. Proc. Natl. Acad. Sci. USA *103*, 4693–4698.

Brian Butterworth is at the Institute for Cognitive Neuroscience, University College London, London WC1N 3AR, UK.
E-mail: b.butterworth@ucl.ac.uk

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Cell–Matrix Adhesion: The Wech Connection

Integrins link the extracellular matrix to the cytoskeleton via a complex of proteins: the integrin–cytoskeleton link. A recent study in *Drosophila* has uncovered a new component of the link, Wech, and shown that it is essential for integrin-mediated adhesion.

Isabelle Delon and Nick Brown

Cell adhesion in multicellular organisms relies on highly conserved multi-protein complexes. Attachment of cell layers to each other is mediated by the integrin family of transmembrane receptors. Integrins connect to ligands in the extracellular matrix, and to the cytoskeleton inside the cell [1]. This connection is the basis of cellular junctions that mediate stable adhesion in tissues. Integrins are also essential for cell migration over the extracellular matrix. The assembly of the organism requires integrins to mediate attachment between cell layers, such as the attachment of the dermis to the epidermis in mice [2], or of muscles to the body wall in worms and flies [3]. Disrupting integrin function results in separation of these cell layers and impairment of migration, and the subsequent death of the animal. Integrins do not attach to the cytoskeleton directly, but via a complex of proteins, or the 'integrin–cytoskeleton link' (the link) [4]. Disrupting the function of one of these components can be as deleterious as disrupting integrins themselves, stressing their significance for integrin-mediated adhesion. A recent paper from Löer *et al.* [5] reports the identification of a new essential member of the integrin–cytoskeleton link.

The molecular composition of this link has been extensively studied in many systems, and 156 components have been collated so far that may contribute to it [6]. The multi-protein complex identified was called 'the adhesome', and includes the link as well as proteins involved upstream and downstream. Amongst the components of the adhesome are 90 'intrinsic' components which physically localise to adhesion sites, and 66 'peripheral' components affecting the activity of the intrinsic ones. Four functional families of adhesome components can be defined: adhesion receptors, adaptors and actin regulators, which form the physical structure of the adhesion site; and signalling molecules, consisting mostly of enzymes that modify the interactions and signal inside the cell. Löer *et al.* [5] report that mutation of the *Drosophila wech* gene mimics the absence of integrins in the embryonic muscles. The Wech protein is concentrated at sites of integrin adhesion, such as the muscle ends, and require talin to be positioned there. In absence of Wech, integrin-linked kinase (ILK) and tensin are reduced, but PINCH is still localised (Figure 1). These data suggest that Wech provides a link between talin and ILK, and this was confirmed by finding that Wech binds to both proteins. Mutation of the *wech* gene causes a stronger phenotype than that of *ilk,* suggesting that Wech does more than just recruiting ILK. From these data Wech can be classified as an adaptor molecule.

Given that so many proteins have been implicated as adhesome components already, why is it remarkable to find a new one? First of all, Wech is a member of a protein family that contains domains not so far documented in the 156 other known adhesome proteins. Second, the other members of the Wech family have very different functions, such as regulating cell proliferation and tumour suppression. Third, it is exciting that forward genetic