have accumulated within a single allele in the short time window since house mice populations have been exposed to anticoagulant pesticides. The fact that all amino acid substitutions in the introgressed *vkorc1* gene strongly inhibit vitamin K epoxide reduction [18], and thus are likely to contribute to resistance, would support this hypothesis. Whether the overall fitness advantage of acquiring resistance through introgression is greater than from *de novo* mutations or standing genetic variation remains to be determined.

While Kohn and colleagues' study of introgression in European mice [12] does not by itself answer the overall question about how frequent adaptive introgression is in nature, it does make several important contributions. First, it provides an example of the multiple lines of evidence required to convincingly document adaptive introgression, ranging from identification of the causative genes and traits to documentation of their fitness effects to reconstruction of their molecular evolutionary history. Second, it suggests that introgression may play an important evolutionary role through the simultaneous transfer of multiple advantageous mutations within genes, in addition to the exchange of favorable sets of genes as previously theorized. Lastly, the study implies that human-mediated changes in selection pressures and dispersal patterns may frequently create conditions where introgression is adaptive. The latter two insights enlarge the circumstances under which introgression is likely to facilitate adaptive evolution, suggesting that close to 40 years after Heiser's seminal review [1] the time may have arrived to re-examine introgression once again.

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Cognitive Neuroscience: Swapping Bodies in the Brain

A recent study has found that activity in multisensory brain areas, namely the premotor cortex, intraparietal cortex and the putamen, mirrors the vividness of ownership over a mannequin, induced by the body-swap illusion.

G. Lorimer Moseley

Rene Descartes was a mind-body dualist. Yet he was painfully aware of how closely united we are with our body: "*I am not merely lodged in my body as a pilot in a ship, but…. I am so closely united to it that I seem to compose with it one whole. For if that were not the case, when my body is hurt, I, the thinking thing, should not feel pain, but would perceive the wound just as the sailor perceives something damaged in his vessel*" (1641). Almost five centuries later, we are beginning to understand how this sense we have that our body is ours is produced by the human brain. An important paper by Petkova *et al*. [1], published recently in *Current Biology*, suggests a possible neuroanatomy of this sense of full-body ownership. In three separate studies involving the experimental manipulation of whole body ownership in healthy human volunteers, the authors show that activity in premotor cortex, intraparietal cortex and putamen, mirrors the self-reported vividness of the full-body ownership illusion. The authors argue that two mechanisms underpin our sense of owning our entire body — the integration of visual, tactile and proprioceptive information in body-part-centered frames of reference, and the perceptual binding of the separate body parts into a unified percept of whole-body ownership.

That multisensory illusions can be relatively easily induced experimentally has been appreciated for some time — Tastevin [2] first reported illusory ownership over an artificial finger over 70 years ago. Botvinick and Cohen [3] reignited interest with their account of the rubber hand illusion and, since then, a great deal has been uncovered about the nature, extent and neural substrate of limb ownership in healthy and clinical populations (see [4] for review). Petkova *et al.* [1] used an

established method to induce the illusion that one has adopted a mannequin for a body — the 'body swap illusion' — while participants underwent functional brain imaging. To induce the illusion, the participant is tapped on the chest while they watch, through a head mounted display, a first person perspective of a mannequin being tapped on the chest (i.e. a view from the perspective of the mannequin's head). The taps are synchronised.

These illusions exploit the brain's predilection for congruence between multiple sensory modalities. Most studies have used synchronous tactile and visual input, which led to the suggestion that multisensory cells underpin the illusion [5]. That is, visual input of the stimulus occurring on the artificial hand or body activates multisensory mechanisms, and tactile input of the stimulus occurring on the actual hand or body activates the same multisensory mechanisms. Together, they signal a single multisensory event.

Visual input is not critical to induce illusory ownership. For example, the illusion can be induced without visual input by the participant stroking a rubber arm while the experimenter simultaneously strokes the participant's other arm [6]. Once illusory ownership is in place, closing one's eyes does not break it [7]. Tactile input is not critical either — passive or active movement of an anaesthetised finger combined with visual input of a fake finger undergoing the same movement induces a vivid sense of ownership over the fake finger [8]. Indeed, the 'fake finger' experiments [8] suggest that precise synchrony of bimodal inputs induces a stronger illusion than less precise multisensory inputs — it is the synchrony of inputs, not the multisensory extent of those inputs, that seems most important.

It has been proposed that different brain areas underpin the induction and the maintenance of illusory limb ownership [9]: intraparietal and premotor cortices initiate the illusion and the right insula and frontal operculum subserve the sense of ownership that follows. That damage to the right insula is an important determinant of 'disturbed sensation of limb ownership' after stroke [10], and that the rubber hand illusion is associated with limb-specific disruption of autonomic control [7], lend support to the importance of the insula. Petkova *et al.* [1], however, report significant illusion-related activation in insula cortex in only one of their three studies and even then, activation was only apparent at lower statistical thresholds. Perhaps insula cortex activity does not fluctuate with experimental condition because during the 'off' condition the participant still has a sense of ownership, it is just over their own actual body, not the artificial one.

Animal studies appear consistent with the role of premotor and intraparietal areas in the sense of body ownership, insofar as they are clearly critical for multisensory integration. Both areas receive projections from visual association and somatosensory areas [11], and both contain neurons that respond to visual and tactile stimulation [12] and have visual receptive fields that are anchored to the limb. As such, these receptive fields move with the limb, ensuring consistent visuotactile coupling [12]. That the intraparietal activation associated with ownership was lateralised to the left in the Petkova *et al*. study [1] appears consistent with the established role of this brain area in motor and body-related attention [13,14].

Other brain areas are important in multisensory integration and have been implicated in body ownership, most notably the temporoparietal junction. Temporoparietal junction activity mirrored the out-of-body sensation induced in healthy volunteers [15]; stimulation of the temporoparietal junction in a patient undergoing brain surgery evoked out-of-body sensation [16], and, in a series of neurological patients who reported out of body experiences, lesion analysis revealed damage at the temporoparietal junction across the group [17]. In fact, the temporoparietal junction is implicated in other disorders of embodiment and self-location, which are attributed to different kinds of disruption of multisensory integration [18]. One might suggest then that the temporoparietal junction is critical for the feeling of disembodiment rather than embodiment.

Petkova *et al.* [1] extend the vast body of work on illusory limb ownership to illusory whole-body ownership, in which the left ventral premotor cortex seems to be particularly important. They substantiate this claim in two ways. First, they designed an experiment to specifically interrogate neural activity associated full body versus partial body ownership: the mannequin's hand was either attached to, or detached from, the mannequin's body, and the mannequin's hand and the participant's hand were stroked in an asynchronous or synchronous manner. Activation in the three multisensory areas was greater during the attached synchronous trials than it was during the other trials. However, this finding may simply reflect the reduced vividness of the illusion in these conditions — certainly such factors as congruence of felt and seen position and orientation are known to affect the rubber hand illusion [19]. Second, and perhaps more compelling, multivoxel pattern analysis [20] revealed, in all twenty participants, a cluster of voxels, activity of which was specific to the full-body illusion across multiple studies. This is in contrast to the majority of voxels, in which activity varied according to the body part used to induce the illusion.

We are still short of fully understanding the unity we have with our body, eloquently described by Descartes centuries ago. In the first instance, it would seem prudent to clarify that the key role of the ventral premotor cortex in full body ownership is truly about ownership and not about the induction of the illusion [9]. More importantly, if we are to move forward we need new paradigms and new experimental approaches. We need a method of experimentally modulating ownership over our own body. Although elegant, the illusory ownership studies really only reveal the neural substrate of owning another body, not our own.

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Tissue Remodeling: Making Way for Cellular Invaders

Cellular invasion through protein matrices is a critical process during epithelial–mesenchymal transitions. A recent study of *Caenorhabditis elegans* vulval development reports a novel invasive mechanism in which cells coordinate spatially restricted degradation and sliding of a basement membrane during cellular ingression and tissue formation.

Mark Schramp and Jeff Hardin

The invasion of cells through the basement membrane is a critical process during animal development, when mesenchymal cells detach from their resident epithelia to migrate within the embryo [1]. Additionally, the re-acquisition of invasiveness is one of the earliest steps in metastasis [2]. The 'standard' model of cellular invasion through an underlying basement membrane involves two premises: decreased or altered expression of genes whose protein products are essential to the structural integrity of the extracellular matrix (ECM), leading to a more porous or labile matrix; and/or the localized and regulated secretion of proteases to degrade ECM components to form a hole in the matrix through which cells can move. Examples of each of these processes have been well documented, and include the loss of certain basement-membrane-associated proteins and increased secretion of the glycoprotein fibronectin during tumor cell invasion [3] and the enhanced secretion and activity of matrix metalloproteases during neurulation [4]. Studies of other invasive events, however, such as leukocyte invasion into endothelial-based tissues, suggest that collaborative processes between multiple cell types are critical to form basement membrane gaps [5]. Recent work by Ihara *et al.* [6] indicates that another, novel mechanism exists to promote cell invasion. In this case, migrating cells expand a previously formed gap in the basement membrane by sliding the perforated ECM apart, allowing them to move through it.

During development of the vulva in *Caenorhabditis elegans* hermaphrodites, epithelial cells known as vulval precursor cells (VPCs) are born on the ventral surface of the animal. The VPCs then invaginate, giving rise to a stack of seven toroids (vulA, ventral-most, through vulF, dorsal-most; Figure 1) that form an epithelial lumen through which mating and the passage of fertilized eggs or embryos occurs [7]. Vulval invagination is preceded by the localized secretion of proteases from the anchor cell (AC) and its subsequent movement adjacent to the 1°-fated VPCs, which form direct attachments with uterine epithelial cells [8]. This invasive event creates a gap in both the gonadal and ventral basement membranes, through which the invaginating cells will ultimately pass (Figure 1). Thus, *C. elegans* vulval development provides a unique *in vivo* system to further define the molecular mechanisms of cell invasion, and its consequences for other concurrent morphogenetic events.

Ihara *et al.* [6] began their analysis by using a tried-and-true approach in *C. elegans* — laser ablation — to identify which cells are involved in widening the perforation that normally forms at the site of AC invasion and found that both VPCs and ventral uterine cells are required. Ihara *et al.* [6] went on to use several important technical approaches to identify how regulated formation of basement membrane perforations occurs during AC invasion, and which cells are involved. One is the use of Dendra — a stable, photoconvertible, fluorescent protein [9] — fused to components of the basement membrane (such as laminin) to track ECM movement. Using this technology, Ihara *et al.* [6] showed that the basement membrane adjacent to the ECM gap induced by AC invasion remains intact while the diameter of the gap increases. Furthermore, photobleached basement membranes proximal and distal to the expanding gap had similar rates of fluorescence recovery, suggesting that decreased membrane deposition does not