Recent studies suggest that a complex, distributed neural network underpins semantic cognition. This article reviews our contribution to this emerging picture and traces the putative roles of each region within this network. Neuropsychological studies indicate that semantic cognition draws on at least two interacting components: semantic representations [degraded in semantic dementia (SD)] and control processes [deficient in patients with multimodal semantic impairment following stroke aphasia (SA)]. To explore the first component, we employed distortion-corrected functional magnetic resonance imaging (fMRI) and transcranial magnetic stimulation (TMS) in healthy volunteers: these studies convergently indicated that the anterior temporal lobes (ATLs; atrophied in SD) combine information from different modalities within an amodal semantic “hub”. Regions of cortex that code specific semantic features (“spokes”) also make a critical contribution to knowledge within particular categories. This network of brain regions interacts with semantic control processes reliant on left inferior frontal gyrus (LIFG), posterior middle temporal gyrus (pMTG) and inferior parietal cortices. SA patients with damage to these regions have difficulty focussing on aspects of knowledge that are relevant to the current goal or context, in both verbal and non-verbal tasks. SA patients with LIFG and temporoparietal lesions show similar deficits of semantic control, suggesting that a large-scale distributed cortical network underpins semantic control. Convergent evidence is again provided by fMRI and TMS. We separately manipulated the representational and control demands of a semantic task in fMRI, and found a dissociation within the temporal lobe: ATL was sensitive to the number of meanings retrieved, while pMTG and LIFG showed effects of semantic selection. Moreover, TMS to LIFG and pMTG produced equal disruption of tasks tapping semantic control. The next challenges are to delineate the specific roles of each region within the semantic control network and to specify the way in which control processes interact with semantic representations to focus processing on relevant features of concepts.
as non-verbal, everyday skilled behaviours (e.g., using objects and sequencing actions to achieve a goal). Our semantic knowledge is multimodal: it allows us to determine the meanings of items encountered via any of our senses. We can recognise and understand pictures, faces, smells, environmental sounds, words and sentences and events, and establish the identity of objects through touch. However, multimodal semantic representations are not sufficient for successful semantic cognition because we store a wealth of information about the meanings of words/objects and typically only a subset of this knowledge is required for a task – other aspects of knowledge may actually be inappropriate and unhelpful. For example, thinking about “coins” and “loans” is probably not helpful when trying to understand the sentence “The bank was slippery” (since in this context, the word “bank” is likely to refer to a river). Similarly, playing the piano requires information about fine movements of the fingers to be retrieved, yet if your task is to move a piano across the room, it is necessary to retrieve very different actions (Saffran, 2000). Control processes therefore play an essential role in shaping the activation within the semantic system, such that context- and task-relevant aspects of meaning are brought to the fore. Although in some circumstances, it may be sufficient to retrieve dominant aspects of meaning relatively automatically, in many other situations, we need to retrieve distant semantic associations or weakly activated features in a more controlled way, and/or select pertinent aspects of knowledge whilst inhibiting irrelevant semantic features. We may also need to configure the components of the semantic network in line with our current goals or expectations and to monitor our semantic retrieval so that control processes can be adjusted if necessary.

Semantic representation and control are not encapsulated in single, modular brain areas but reflect the joint action of a widely distributed set of cortical regions (in common with other higher brain functions; see Fig. 1). To make progress in understanding this network of brain regions, we have conducted neuropsychological studies comparing the nature of semantic deficits that arise from different aetiologies and other higher brain functions; see Fig. 1). To make progress in understanding this network of brain regions, we have conducted neuropsychological studies comparing the nature of semantic deficits that arise from different aetiologies and areas of brain injury. (2) Patients can show deficits in comprehension that are specific to a particular modality: for example, patients with ‘pure word deafness’ have difficulty accessing semantic knowledge from spoken words, while those with visual agnosia have difficulty accessing knowledge from vision (e.g., Farah, 2004; Franklin et al., 1996). The fact that comprehension from other modalities is intact in such patients indicates that the central store of conceptual knowledge is preserved. (2) Individuals with semantic dementia (SD) show progressive degradation of central conceptual representations, while other aspects of language and cognition remain largely intact (e.g., Hodges et al., 1992; Snowden et al., 1989; Warrington, 1975). This erosion of semantic knowledge gives rise to poor comprehension across all input and output modalities (Bozeat et al., 2000; Patterson et al., 2007). (3) Multimodal semantic deficits can also occur in patients with stroke aphasia (SA), although they are associated with different areas of brain damage that do not overlap with the regions in SD (see below; Jefferies and Lambon Ralph, 2006). These patients inconsistently access the meanings of items: in particular, they have difficulty in semantic tasks with greater executive demands. This suggests that SA patients have an intact store of conceptual knowledge but damage to semantic control processes.

Following these case-series comparisons of patients with SD and SA, which have highlighted the effects of impairment to amodal semantic representations and control processes respectively, we have used complementary neuroscientific methods — functional neuroimaging and transcranial magnetic stimulation (TMS) — to seek converging evidence for our hypotheses about the neural basis of these two key components of semantic cognition in healthy volunteers.

1. Neural basis of semantic representation

Where is semantic knowledge represented in the brain? Many researchers propose an ‘embodied’ view in which semantic information draws on a distributed network of sensory and motor representations (e.g., Pulvermüller, 2005; Martin, 2007; Barsalou, 1999). According to this view, the meaning of an item like “scissors” is derived from links between neural assemblies that represent this object’s distinctive shape, the “snip” sound that it makes, information about how you hold and use scissors, linguistic properties of the word “scissors” and so on. These links allow all of the information you have about an object to be activated from a single modality — so that, on hearing the word “scissors”, you can easily imagine...
what they look, sound and feel like. Functional neuroimaging studies provide support for this proposal: the retrieval of semantic information, such as an object’s colour or action, elicits activation in cortical areas that are adjacent to (or overlapping with) perceptual/motor areas (Martin et al., 1995; Chao et al., 1999; Goldberg et al., 2006; Hauk et al., 2004). Patients who are unable to access semantic knowledge from a specific input, such as an object’s visual form or spoken name (as in visual agnosia or pure word deafness), are likely to have damage to these modality-specific aspects of meaning.

Despite these findings, there is ongoing controversy about the extent to which sensory and motor activation is necessary and sufficient for semantic processing (e.g., Météyand et al., 2012; Bedny et al., 2008a). Functional neuroimaging studies cannot conclusively show that sensory and motor activation is essential for computing meaning, since this could reflect spreading activation from modality-specific semantic regions and, in any case, such activation is unlikely to be essential for processing the meaning of abstract concepts, such as "truth", which do not have sensory or motor correlates. Some theorists have suggested that there are “convergence zones” where different types of information are combined into more abstract, multimodal semantic representations (Damasio, 1989; Patterson et al., 2007); by this view, modality-specific activation on its own is insufficient to explain conceptual processing. Multiple regions distributed across the cerebral hemispheres may underpin the convergence of different types of information — for example, one convergence zone to link vision to language, and another to link environmental sounds to language (Damasio, 1989, 1990). However, distributed convergence zones and/or direct links between sensory, motor and language-specific regions may not provide an adequate account of central semantic disorders that affect all modalities equally, at least when these occur in the absence of widespread brain injury (Patterson et al., 2007). As a consequence, Patterson, Lambon Ralph and colleagues have proposed that all knowledge pertaining to a single concept is combined within semantic representations in the anterior temporal lobes (ATLs; Patterson et al., 2007; Lambon Ralph et al., 2010; Rogers et al., 2004a; Pobric et al., 2010b). According to this “hub and spokes” model, conceptual categorisation requires central amodal representations in addition to sensory, motor and language areas. This is because objects that are semantically similar can nevertheless be very different in many of their features — for example, pineapples and grapes are different in shape, colour, size and texture, yet are highly related conceptually. By this view, the assignment of meaning always involves the ATL semantic hub, irrespective of whether the items are pictures, sounds, smells, objects, faces, actions or words denoting concrete or abstract concepts.

1.1. Degradation of conceptual knowledge in semantic dementia

Much of the evidence for a central, amodal semantic store in the ATL comes from patients with SD, who have atrophy and hypometabolism that is predominately focussed on the anterior inferior temporal lobes bilaterally (Mummery et al., 2000; Galton et al., 2001; Studholm et al., 2004; Rosen et al., 2002; Desgranges et al., 2007; Diehl et al., 2004; Nestor et al., 2006; Mion et al., 2010). These patients display progressively worsening semantic deficits that affect all modalities, including words (concrete nouns, verbs and abstract concepts — which show equivalent deficits as long as frequency/familiarity are matched), pictures, sounds, smells and actions (Bozeat et al., 2000, 2002, 2003; Luzzi et al., 2007; Coccia et al., 2004; Garrard and Carroll, 2006; Ikeda et al., 2006; Hoffman and Lambon Ralph, 2011; Jefferies et al., 2009; Bird et al., 2000). Despite this all-encompassing conceptual deficit, other aspects of cognition — for example, phonology and syntax, digit span, visuospatial processing and executive control — are largely preserved (Jefferies et al., 2004, 2005; Kramer et al., 2003; Hodges et al., 1999).

Several features of SD support the view that these patients have degradation of central amodal semantic representations (see Patterson et al., 2007; Lambon Ralph et al., 2010; Lambon Ralph and Patterson, 2008). Patients exhibit a systematic decline in their knowledge, which affects less familiar, atypical, and specific-level concepts more severely and at an earlier stage of the disease (Hodges et al., 1995; Bozeat et al., 2003; Woollams et al., 2008; Patterson et al., 2007; Rogers et al., 2004a). General information that is shared across many semantically-related concepts (e.g., animals have four legs) is better preserved than specific information that is not shared (e.g., giraffes have long necks). SD patients show this specific-to-general decline across a wide variety of semantic tasks, involving verbal or non-verbal input, differing output (production vs comprehension) and a variety of task demands (e.g., matching, similarity and association judgements) — for example, this pattern is evident in picture naming, word—picture-matching, picture copying and object decision (Bozeat et al., 2003; Woollams et al., 2008; Patterson et al., 2006, 2007; Rogers et al., 2004a). In object decision tasks, patients are likely to accept pictures in which a relatively unique feature has been replaced by an incorrect shared feature (such as a giraffe with a short neck) but can readily reject picture in which a shared feature has been replaced by a unique one (such as an elephant with a long neck) (Rogers et al., 2004b). In addition, when the same concepts are probed using different input/output modalities or different types of semantic task, SD patients are highly consistent in the concepts they can demonstrate knowledge of and the concepts that are impaired, even when frequency/familiarity is taken into account (Bozeat et al., 2000; Jefferies and Lambon Ralph, 2006). This suggests that SD produces degradation of a central store of amodal semantic representations: the degree of damage to a particular concept in an individual patient therefore predicts their performance on any given semantic task.

1.2. Convergent support for an amodal semantic hub in the ATLs

Despite the story emerging from studies of patients with SD, the view that the ATL plays a central role in the representation of semantic knowledge has proved controversial. This partly reflects the fact that, until recently, there was relatively little corroborating information from other neuroscience methods, such as functional magnetic resonance imaging (fMRI) and transcranial magnetic stimulation (TMS). Convergent evidence from healthy participants using these methods
is essential because while SD patients have relatively focal atrophy of bilateral ATL, many also have mild atrophy and hypometabolism in mid-inferior temporal and/or orbitofrontal areas. In addition, SD patients have bilateral ATL damage and this makes it difficult to assess the roles of left and right ATL separately. In contrast, functional neuroimaging and TMS studies can explore the roles of smaller anatomical regions, and separately consider the roles of left and right ATL. This work can therefore determine which portions of ATL are critical for semantic processing, and if there is any differentiation of function across this region.

1.2.1. Functional neuroimaging
While some functional neuroimaging studies, particularly those employing positron emission tomography (PET), have highlighted the role of ATL in semantic processing (e.g., Devlin et al., 2000; Bright et al., 2004; Rogers et al., 2006; Scott et al., 2000; Crinion et al., 2003; Noppeney and Price, 2002), many fMRI studies have failed to observe activation in this area, and instead have reported that key semantic regions fall within inferior frontal, posterior temporal and inferior parietal cortex (e.g., Wible et al., 2006; Gold et al., 2006; Demb et al., 1995; Mechelli et al., 2006). Given the pre-eminence of fMRI within cognitive neuroscience, this probably explains why some influential models of the neural basis of semantic cognition do not include a key role for the ATL (Martin, 2007; Thompson-Schill, 2003; Catani and Ffytche, 2005).

With Maya Visser, we recently conducted a meta-analysis of over 160 functional neuroimaging studies which explored (1) the factors that influence the likelihood of observing ATL activation during semantic tasks and (2) the location of reliable activation across studies (Visser et al., 2010b). ALE analyses showed reliable activation within ATL for both words and pictures, from mid-temporal lobe sites to the pole (e.g., y = 2–22; although there was some variation in function across this large region, detailed below). ATL activation was found more often in studies (1) with a large field of view, ensuring data acquisition from even the most inferior parts of the ATL, (2) when a demanding baseline task was used to prevent semantic processing during ‘non-semantic’ periods (see also Binder et al., 1999) and (3) when the ATL was selected as a region of interest (which is unlikely for researchers adopting one of the models above that do not emphasise the role of the ATL). (4) There was also a very strong influence of imaging modality, with 53% of PET studies revealing ATL activation compared with only 30% of fMRI studies. In line with our meta-analysis, Devlin et al. (2000) directly compared PET and fMRI using the same category judgement task: substantial ATL activation was detected with PET, but none with fMRI. This difference is likely to reflect susceptibility artefacts caused by variations in magnetic field strength at the interface between brain, bone and the air-filled sinuses: in fMRI studies, these produce signal loss and distortion in ATL and orbitofrontal cortex (Ojemann et al., 1997; Gorno-Tempini et al., 2002; Weiskopf et al., 2006; Visser et al., 2010a). PET investigations are not subject to these difficulties.

Karl Embleton, Geoff Parker, Matt Lambon Ralph and colleagues at the University of Manchester have developed a distortion-corrected spin echo fMRI method in order to try to maximise signal acquisition from the ATL (Embleton et al., 2010; Visser et al., 2010a). We used this method to address questions about the role of the ATL in semantic processing that emerge directly from studies of SD patients – for example, (1) which area(s) within ATL are important in semantic processing (since the patients’ atrophy encompasses several potentially functionally-independent regions), and (2) do left and right ATL contribute to semantic processing for both words and pictures (a question that is difficult to address in studies of SD patients with bilateral atrophy). In our first distortion-corrected fMRI study (Visser et al., 2010a), we used the category decision task employed by Devlin et al. (2000): this fMRI method revealed significant ATL activation in line with Devlin et al.’s PET results. Distortion-corrected fMRI has also revealed significant ATL activation in several tasks adapted from investigations of patients with SD (see Binney et al., 2010; Visser et al., 2012), including synonym judgement (Jefferies et al., 2009) and verbal and picture tests of semantic association (Bozeat et al., 2000; Jefferies and Lambon Ralph, 2006). These studies point to a distributed semantic network, involving left inferior frontal cortex (LIFG), posterior middle temporal gyrus (pMTG) and ATL (Fig. 1). The ATL activation extended to the lateral surface of MTG and ITG but was centred on the anterior fusiform gyrus, which is the site of maximal atrophy and hypometabolism in SD (Binney et al., 2010; Mion et al., 2010). These findings suggest that the key site for the semantic hub may not be the temporal pole (TP), but instead inferior and medial parts of the ATL which are more posterior (i.e., within the anterior portions of the mid-temporal lobe).

We have used distortion-corrected fMRI to confirm that left and right ATL show activation to both word and picture tasks (along with LIFG and pMTG, which are also multimodal; Visser et al., 2012). This study therefore confirmed the bilateral amodal pattern seen previously in ATL with PET (Vandenberghe et al., 1996; Bright et al., 2004). In contrast, modality-specific patterns of activation were found in posterior temporal areas: bilateral occipital–temporal cortex was specifically recruited during picture tasks, while left posterior/mid STG/MTG showed greater activation in the verbal task (Visser et al., 2012). These findings are highly consistent with the hub and spoke model of Patterson et al. (2007), which proposes that posterior temporal regions correspond to modality-specific ‘spokes’, while the anterior temporal cortex forms an amodal semantic ‘hub’.

These results do not rule out some differentiation of function within ATL. For example, ATL has been associated with social concepts (Olson et al., 2007; Skipper et al., 2011) and combinatorial semantics/narrative comprehension (Ferstl et al., 2008; Baron and Osherson, 2011), in addition to amodal conceptual representations. It is likely that different regions within ATL make a greater or lesser contribution to these different aspects of conceptual processing. Moreover, even within an amodal hub, there may be graded specialisation which reflects the strength of connection of different ATL regions with posterior temporal modality-specific areas. One possibility is that left and right ATL have somewhat different functions, following stronger connections between (i) left ATL and left-lateralised speech production and phonological processes and (ii) right ATL and right-lateralised face recognition areas. In line with this view, researchers have noted
that while SD patients always show bilateral ATL atrophy, the degree of tissue loss can be lateralised: patients with left > right atrophy have disproportionate problems in picture naming, while those with right > left atrophy have greater difficulty recognising faces than names (Lambon Ralph et al., 2001; Snowden et al., 2004). Similar associations between left versus right-sided ATL atrophy and verbal semantic versus object recognition deficits have also been reported across a broader range of patient groups (e.g., Acres et al., 2009; Vandenbulcke et al., 2006). A second (not mutually exclusive) possibility is that superior ATL may show greater involvement in word semantics, while inferior regions of ATL are more strongly focussed on picture semantic tasks, reflecting the differential connectivity of these areas with modality-specific regions associated with processing verbal inputs (posterior STG) and picture inputs (fusiform cortex).

Our own findings support the second but (so far) not the first of these possibilities. In our meta-analysis (Visser et al., 2010b), there were no differences between verbal and picture tasks in the distribution of peaks across left and right ATL. However, the ATL peaks for picture-based tasks were, on average, more inferior than the peaks for word-based tasks. This pattern was confirmed with distortion-corrected fMRI: word and picture judgements both produced significant activation of left and right ATL, with no clear hemispheric differences, but the anterior fusiform showed greater activation to pictures, while the anterior STG showed greater activation to words (Visser et al., 2012; see also Skipper et al., 2011). It seems, therefore, that even within the ATL, there is a graded pattern of modality-specificity; in regions just posterior to the TP, responses in MTG are wholly amodal (i.e., equivalent for words and pictures), while STG and fusiform cortex show a preference for words and pictures respectively (although fusiform cortex in Visser et al.’s study showed a significant response to both modalities). Computational modelling work by Plaut (2002) reveals how functional specialisation within the semantic system could be driven by connection strengths with modality-specific inputs: this proposal fits well with our data, since anterior STG is strongly connected with posterior superior temporal regions associated with phonology and auditory processing, while anterior fusiform receives input from posterior inferior temporal and fusiform areas associated with object recognition. As a consequence of this graded specificity, it is conceivable that a patient could show an impairment of semantic representation which disproportionately affected words, leaving pictures more intact, or vice versa (although such a pattern is highly unlikely in SD). Verbal > non-verbal impairment would be expected following damage to STG in the mid-temporal lobe, while mid-to-posterior fusiform lesions might elicit the reverse pattern – i.e., semantic deficits for pictures > words. Since other neuropsychological and functional neuroimaging research has suggested that right fusiform gyrus is crucial for knowledge of visual attributes (Vandenbulcke et al., 2006), it is also possible that two gradients of functional specialisation co-exist within anterior temporal cortex (i.e., graded differences between left vs right ATL and fusiform vs lateral cortex in both hemispheres). This would explain why lesions of right fusiform have a particularly catastrophic impact on knowledge of visual (not functional) attributes (Vandenbulcke et al., 2006).

1.2.2. TMS

We have also been employing TMS to establish whether the ATL makes a critical contribution to semantic cognition in healthy participants. TMS generates magnetic pulses over the scalp that induce electrical activity in the underlying neurons: when this is done repeatedly, TMS can be used to temporarily disrupt cortical activity, making it possible to explore the consequences of a ‘virtual lesion’ in an otherwise normal brain (Cowey and Walsh, 1998). This corroborating evidence is essential because, as noted above, the fMRI literature has underemphasised the importance of ATL in semantic cognition. Moreover, while SD patients support the proposal of an amodal semantic hub in ATL, their semantic deficits could reflect subtle atrophy or hypometabolism in posterior temporal cortex (Martin, 2007). TMS, in contrast, produces relatively focal and reversible effects, making it possible to (i) compare the contribution of several sites in the same participants and (ii) differentiate between cortical regions which are typically damaged together in patients – such as left and right ATL in SD.

We used an offline inhibitory TMS procedure (low frequency stimulation for 10 min) which disrupts tasks reliant on the underlying cortex for approximately 10 min after stimulation. Post-TMS performance was compared with ‘baseline’ testing either before TMS was applied or 30 min after the end of stimulation, by which time the effects of TMS have washed out. In a series of studies, we have demonstrated that (i) ATL–TMS disrupts semantic tasks involving a range of input and output modalities (e.g., picture naming; semantic judgements about words and pictures; see Fig. 2); (ii) TMS over this region produces more disruption of specific than basic-level picture naming (e.g., “Dalmatian” vs “dog”); and (iii) both left and right ATL are involved in verbal and non-verbal

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**Fig. 2 – Effects of TMS to left and right ATL on semantic judgements to words and pictures (reproduced from Pobric et al., 2010a, with permission).**

Footnote: each bar represents the reaction time (RT) change caused by TMS. For example, the white bar above the Left TP site indicates that rTMS over left TP caused an RT delay of 177 msec for picture association judgements, when compared to the no-TMS condition. Negative figures indicate RTs on TMS trials were faster than no-TMS trials. Error bars indicate standard error of mean (SEM) adjusted to reflect the between-condition variance used in repeated-measure designs (Loftus and Masson, 1994). Occ Pole = Occipital Pole (control site not thought to be involved in semantic cognition). TP = temporal pole.
semantic processing (Pobric et al., 2007, 2009, 2010a,b; Lambon Ralph et al., 2009). These effects were specific to semantic tasks: response times on number judgement and number naming tasks, which do not rely on ATL, were unchanged. Our findings simulate (in milder form) the impairment seen in SD patients and suggest that the ATL plays an essential role in a wide range of semantic judgements in healthy volunteers.

We used the offline 1 Hz paradigm primarily because this allowed us to undertake behavioural testing in the absence of the jaw contractions and eye blinks that are elicited via facial nerve stimulation when TMS is applied to ATL. However, a caveat should be noted: the behavioural effects we observed do not necessarily show highly focal involvement of ATL, because this TMS method is thought to not only suppress activation directly below the coil, but also produce distal effects, influencing processing in other parts of the network. These distal effects can be compensatory in nature (i.e., increased responsiveness following TMS) or alternatively can reflect reduced activity in connected brain regions. Although these considerations pose a potential problem for the interpretation of our previous studies, they provide an opportunity to study interactions between regions within the distributed neural network underpinning semantic cognition. Binney (2010) used a combined fMRI–TMS approach to chart the effects of 1 Hz TMS to left ATL: this stimulation (1) suppressed activation beneath the coil, (2) reduced activity in other connected regions in the left-hemisphere (posterior temporal and LIFG), yet (3) increased activation in right ATL, supporting our hypothesis that ATL is a bilateral system which supports semantic processing along with posterior temporal and frontal regions.

Finally, we have also directly compared the contribution of the ATL ‘hub’ and feature-specific ‘spokes’ to semantic processing using TMS. This approach overcomes the problem that functional neuroimaging studies are correlational and cannot be used to make causal inferences. If the application of TMS to regions of cortex associated with visual processing or praxis (i.e., the hand shapes and movements critical to tool use) is shown to disrupt semantic tasks (and if this effect is specific to relevant classes of stimuli), then it is possible to make a stronger case for the ‘spokes’ being necessary for semantic processing. In our experiment, 1 Hz TMS over the ATL slowed picture naming for all categories equally, while stimulation of an inferior parietal (IPL) site, involved in coding praxis, induced a category-specific deficit only for manipulable objects (Pobric et al., 2010b). These findings suggest that the ATL semantic ‘hub’ and the modality-specific ‘spokes’ both make a critical contribution to the representation of semantic knowledge. This was apparent even in a picture naming task that did not explicitly require access to these praxis features. In future work, it would be interesting to extend this approach to primary sensory/motor cortex, since the site we stimulated is likely to integrate somatosensory and motor aspects of praxis.

1.3. Summary of brain regions involved in semantic representation

This review, focused on our own research, suggests that the key regions involved in semantic representation include both bilateral ATL (amodal ‘hub’) and ‘spokes’ representing specific modalities or features of objects; for example, posterior inferior temporal cortex (linked to visual object recognition) and inferior parietal lobule (linked to praxis representations). This account of the neural basis of semantic knowledge overlaps substantially with several recent meta-analyses that also implicate large swathes of temporal cortex in semantic processing (Price, 2010; Binder et al., 2009). However, there are detailed differences between these accounts: we suggest that ATL is crucial for all aspects of knowledge — for example, knowledge of semantic features, categories and associations, and representations of both animals and artefacts (in line with the absence of category-specific semantic impairment in SD; Lambon Ralph et al., 2007, 2003). In contrast, other researchers have assigned a more category-selective role to the ATL for animals and/or faces and specific entities (e.g., Warrington and Shallice, 1984; Damasio, 1990), while tool/action knowledge is captured within posterior temporal lobe areas (in particular, pMTG; e.g., Martin et al., 1995; Chao et al., 1999; Perani et al., 1999). The ‘hub and spokes’ account is consistent with the notion of category effects in posterior temporal and parietal areas — for example, pMTG and IPL may be crucial for the representation of actions/tools/verbs, given that these regions are thought to represent visual motion and praxis features that are particularly pertinent to these classes of stimuli. In contrast, the role of ATL in animals and faces may reflect general processing principles that apply to all domains of knowledge — anterior fusiform regions may be critical for semantic judgements with a high degree of specificity, or that require discrimination between highly similar concepts that share a lot of features (such as faces and animals) (Rogers et al., 2005).

In addition, some researchers have suggested that there might be two separate amodal semantic “hubs”, which represent taxonomic and thematic relationships. Using a voxel–lesion symptom mapping approach to examine picture naming errors in patients with SA, Schwartz et al. (2011) found that “taxonomic” errors (e.g., ‘pear’ for apple) were associated with damage to ATL, while “thematic” errors (e.g., ‘worm’ for apple) were associated with damage to temporoparietal cortex. Schwartz et al. suggested that temporoparietal regions (including pMTG and IPL) might be involved in simulating events, capturing action, causal and spatial relations, and that this function might be linked to the role of these regions in action understanding. An alternative view (although not mutually exclusive, and potentially connected to this idea), is that pMTG and IPL form part of a distributed semantic control network (see below).

2. Deregulated semantic cognition in semantic aphasia

Studies of patients with SD have been enormously important in advancing our understanding of the role of the ATL in semantic memory. However, deficits of semantic cognition are seen more frequently following left-hemisphere stroke. These patients typically implicate a different set of regions in semantic processing, most notably left posterior temporal, inferior parietal and inferior frontal regions (Chertkow et al.,
The SA patients additionally made associative errors (e.g., producing “dog” or “animal” for squirrel). Picture naming, SD patients made coordinate and superior same concepts, while SA patients were inconsistent. (ii) In consistent across different semantic tasks that tapped the between these groups: (i) SD patients were strikingly beyond this point (Borden, 2006).

In a series of studies, we have contrasted the nature of the semantic impairment in SD and SA. We selected stroke patients who showed deficits across both verbal and nonverbal semantic tests (as in SD), yet we found these patients had qualitatively different patterns of impairment (see also Ogar et al., 2011). In contrast to SD (which produces gradual degradation of core amodal conceptual knowledge following bilateral ATL atrophy), multimodal semantic impairment in SA was associated with left prefrontal and temporoparietal infarcts, and poor executive control over conceptual processing (Corbett et al., 2009a; Jefferies et al., 2007, 2008; Jefferies and Lambon Ralph, 2006; Noonan et al., 2010). There were a number of qualitative differences between these groups: (i) SD patients were strikingly consistent across different semantic tasks that tapped the same concepts, while SA patients were inconsistent. (ii) In picture naming, SD patients made coordinate and superordinate errors (e.g., producing “dog” or “animal” for squirrel). The SA patients additionally made associative errors (e.g., “nuts” for squirrel), suggesting they had difficulty directing activation towards the target and away from irrelevant prepotent associations. This is related to the distinction between taxonomic and thematic errors noted by Schwartz et al. (2011) but we have suggested an alternative interpretation — patients with left prefrontal and temporoparietal lesions have deficits of semantic control, and as a result, they retrieve task-irrelevant but strong associations (i.e., thematic errors). In contrast, patients with ATL lesions have degraded semantic representations; consequently, they are unlikely to make thematic errors (which require the retention of associations). Instead, they largely make high frequency coordinate errors (e.g., ‘dog’ for goat) and superordinate errors (e.g., ‘animal’ for goat), reflecting their poor knowledge of less familiar and specific-level concepts. (iii) SA patients showed stronger benefits of cues (e.g., phonemic cues in picture naming) that reduced the need for internally-generated semantic control (see Fig. 3; Jefferies et al., 2008). The SA patients were also highly sensitive to miscues designed to activate semantic competitors (e.g., picture of a cat plus/d/; Noonan et al., 2010). (iv) The SD patients showed strong effects of frequency/familiarity, consistent with the view that less frequent concepts are more vulnerable to damage. In contrast, the SA patients showed negligible or even reverse frequency effects. This fits with the prediction that high frequency words have greater semantic selection demands because they appear in more contexts and have more variable meanings (Robinson et al., 2010; Almaghyuli et al., 2012; Hoffman et al., 2011a,b). (v) In tasks designed to directly manipulate the requirement for semantic control, the SA patients were poor at (a) inhibiting strongly associated distracters and (b) focussing on less dominant aspects of meaning (Noonan et al., 2010). (vi) Semantic impairment in SA correlated with executive dysfunction (see also Baldo et al., 2005; Wiener et al., 2004), while executive functions were largely spared in SD.

Fig. 3 – Qualitative differences between SD and SA patients in the effect of progressive phonemic cueing on picture naming (reproduced from Jefferies et al., 2008, with permission)

Footnote: patients with SD and multimodal semantic impairment following SA attempted to name black and white line drawings. If they were unable to provide the correct name without a prompt, they were provided with progressive phonemic cues. Phonemes were added to cue one at a time until the patient produced the correct response or the cue consisted of the whole word minus the last phoneme (referred to as the ‘final cue’). Cueing produced small but significant benefits for SD patients and more dramatic benefits in SA: even the most impaired SA patients were able to retrieve almost all of the target names by the final cue, consistent with the proposal that their semantic knowledge remains intact.
These findings suggest that semantic cognition is underpinned by a large-scale neural network comprising at least two interacting principle components – (i) semantic knowledge, including amodal representations in the ATL (degraded in SD) and (ii) semantic control processes that allow task- and context-relevant aspects of knowledge to be brought to the fore, while irrelevant information is suppressed (disrupted by left prefrontal and temporoparietal lesions in SA) (Jefferies and Lambon Ralph, 2006; Noonan et al., 2010). However, further studies were needed to establish the underlying nature of the semantic control deficit in SA – for example, whether the impairment is specific to verbal semantics (since the tasks above all involved words), or if it extends to non-verbal conceptual tasks, even though the key presenting symptom in this group is aphasia. Studies led by Faye Corbett have demonstrated semantic control deficits across a wide range of verbal and non-verbal tasks (Corbett et al., 2009a,b, 2011). In one study, we compared SD and SA patients on a battery of object use tasks and found qualitative differences that resembled the pattern in the verbal domain. While the SD patients performed consistently across tasks that tapped different aspects of knowledge and object use for the same items, the SA participants’ performance reflected the control requirements of the tasks. They were able to complete straightforward item matching tasks, such as word–picture-matching, but performed more poorly on associative picture-matching involving the same items, and on complex mechanical puzzles (Corbett et al., 2009a). The SA group also showed strong sensitivity to manipulations of semantic control in the domain of object use: they had difficulty using their knowledge of specific semantic features to flexibly support the non-canonical uses of objects (e.g., using a newspaper as a fly swat), they showed poor inhibition of semantically-related distractors, and their object use improved with the provision of verbal and pictorial cues designed to constrain the task (Corbett et al., 2011).

These findings have important consequences for how we approach aphasia rehabilitation. If semantic knowledge is largely intact in SA but these patients have difficulty retrieving task- and context-relevant aspects of meaning following executive deficits that are correlated with the degree of semantic impairment, interventions that minimise the executive demands of semantic tasks are likely to be effective in improving comprehension. Indeed, cues that are consistent with the target response and inconsistent with distractors, and which therefore minimise the requirement for internally-generated semantic control, boost the performance of SA patients in both verbal and non-verbal tasks (Corbett et al., 2011; Jefferies et al., 2008). Longer-lasting gains in comprehension may follow from a combination of training on executively-demanding semantic tasks, plus brain stimulation techniques such as transcranial direct current stimulation (tDCS) to augment neural plasticity.

2.1. Convergent support for a distributed network underpinning semantic control

The prefrontal cortex has long been associated with cognitive control and there is strong neuropsychological evidence linking left inferior frontal gyrus (LIFG) to deficits in the control of linguistic and semantic processing (Schnur et al., 2009; Thompson-Schill et al., 1998; Novick et al., 2009; Robinson et al., 2010). However, as noted above, our research suggests that semantic control impairment in SA follows from both left prefrontal and temporoparietal lesions. SA cases with LIFG and temporoparietal lesions have almost indistinguishable deficits: both groups have difficulty rejecting strongly related distractors, retrieving the less frequent meanings of ambiguous words and overcoming the effects of miscues (Noonan et al., 2010). This suggests that anterior and posterior brain regions form a large-scale distributed cortical network underpinning semantic control.

Nevertheless, patients with large and variable lesions are not ideally suited to producing a detailed neural model of semantic control: SA patients’ deficits could conceivably follow damage to white matter tracts and/or their cortical damage could span several functionally-separable regions – for example, temporoparietal patients often have damage to both posterior temporal and inferior parietal cortex, which might make separate contributions to semantic control. Similarly, frontal SA patients might have damage encompassing several functionally discrete regions, such as LIFG and dorsomedial PFC (which is associated with executive control beyond the semantic domain; Duncan, 2010). Although SA patients show deficits on standard measures of executive function which correlate with their degree of comprehension impairment (Jefferies and Lambon Ralph, 2006; Baldo et al., 2005; Wiener et al., 2004), the large lesions in this group may mask functional specialisation, potentially within both the frontal and posterior components of the semantic control network. Some regions may be selectively involved in linguistic control, some may underpin conceptual control for both verbal and non-verbal stimuli, and others may particulate in executive control irrespective of task (semantic vs non-semantic) or modality (verbal vs non-verbal). Once again, it is therefore important to seek convergent evidence from neuroimaging and TMS methods that have better spatial resolution. The key questions are: (1) is there convergent evidence for a large-scale distributed neural network underpinning semantic control that encompasses areas beyond LIFG; (2) which specific sites within the areas of brain-injured in SA are crucial for semantic control and (3) what is the functional organisation of the semantic control network – i.e., how does semantic control relate to linguistic control and domain-free executive control?

2.1.1. Functional neuroimaging

fMRI studies of healthy participants partially converge with the conclusions of our neuropsychological investigations, although their primary focus has been on LIFG (BA44,45,47) (Thompson-Schill et al., 1997; Wagner et al., 2001; Badre et al., 2005). This region responds strongly to experimental manipulations of semantic control demands: it shows more activation for (i) ambiguous words (with multiple meanings) compared with unambiguous words (Bedny et al., 2008b; Rodd et al., 2005; Whitney et al., 2009), (ii) associations based on specific semantic features (i.e., colour, shape) as opposed to associations based on global semantic relatedness, (iii) decisions involving many versus few response options and (iv) cue-target pairs when associative strength is low versus high.
(Badre et al., 2005; Thompson-Schill et al., 1997; Wagner et al., 2001). However, while SA patients typically have widespread damage in LIFG, fMRI suggests a possible functional subdivision, with anterior regions (BA47) acting to bias semantic retrieval according to current goals, and posterior regions (BA45/44) underpinning semantic selection when several competing representations are active (Badre et al., 2005).

Unlike our patient studies, the fMRI literature does not highlight a role for temporoparietal areas in semantic control. This apparent discrepancy between methods is important because some researchers propose that posterior temporal cortex (as opposed to ATL) is the central site for semantic representation (e.g., Martin, 2007). In reality, there are likely to be dissociable regions within temporal cortex that support semantic control and the storage of knowledge. Many neuroimaging studies have, in fact, observed sites in posterior temporal and inferior parietal cortex that show activation modulated by semantic control demands (e.g., Badre et al., 2005; Nagel et al., 2008; Thompson-Schill et al., 1997; Wagner et al., 2001). We recently conducted a meta-analysis of functional neuroimaging studies that confirmed that, like LIFG, pMTG, intraparietal sulcus (Noppeney et al., 2004) and portions of the left inferior parietal lobule (particularly a site at the boundary of dorsal angular gyrus (AG) and supramarginal gyrus) are reliably influenced by manipulations of semantic control (Nooen et al., submitted for publication). In this study, we selected 53 neuroimaging studies which contrasted semantic tasks with high versus low executive requirements. Moreover, we were able to demonstrate a functional dissociation between ATL and pMTG within a single fMRI study utilising ambiguous words in a double-prime paradigm (Whitney et al., 2011a): ATL was sensitive to the number of meanings that were retrieved, consistent with a role for this region in semantic representation, while pMTG and LIFG showed greater activation when the dominant meanings of words had to be inhibited, supporting our hypothesis that these regions underpin semantic control.

2.1.2. TMS

Once again, we have used TMS to confirm a role for these sites in semantic control. First, in a convergent neuropsychological and TMS investigation, we focussed on the role of LIFG in semantic judgements for abstract words (Hoffman et al., 2010) – one of the brain regions most reliably activated by tasks involving abstract as opposed to concrete words (Binder et al., 2009). Abstract concepts are generally more difficult to process than concrete concepts but there are at least two alternative (although not mutually exclusive) explanations for this. One view, referred to as the “dual-coding account”, (Pavio, 1986) is that abstract concepts rely predominately on language areas: therefore, they do not benefit from broader ‘embodied’ activation within sensory and motor areas. An alternative approach suggests that abstract words are used in a broader range of contexts and this makes it more difficult to activate associations relevant to these words (Schwanenflugel and Shoben, 1983). From these accounts, we might predict that abstract concepts would require more semantic and linguistic control compared with concrete concepts because (i) they are used in a broader range of contexts and their meanings are therefore ambiguous (Hoffman et al., 2011b) and (ii) they have higher lexical retrieval or phonological working memory demands (Binder et al., 2005). First, we established that SA patients with LIFG damage had greater difficulty in a synonym judgement task for abstract than concrete items. Similar difficulties have been demonstrated before for patients with left perisylvian lesions, consistent with the view that linguistic processes are particularly critical for these concepts (e.g., Goodglass et al., 1969); however, as noted above, our SA patients showed comparable deficits in verbal and non-verbal semantic tasks, suggesting they had impairment of amodal control processes. Moreover, their problems were ameliorated by providing a sentence cue that placed the word in a specific context. Our interpretation of this finding is that the sentence cues reduced the need for internally-generated semantic and linguistic control by focussing activation on relevant aspects of meaning. When TMS was applied to LIFG (BA45) in healthy participants, we replicated this pattern: response times were slowed for abstract and not concrete words, but only in the absence of a sentence cue.

We have also used TMS to investigate the wider neural network underpinning semantic control for words, by contrasting the effects of stimulation of LIFG and pMTG (Whitney et al., 2011b). We found that TMS to these sites produced an identical pattern: there was no effect of TMS on semantic judgements to strongly associated pairs of words (i.e., SALT-PEPPER), which are thought to be underpinned by automatic spreading activation potentially within the language system itself (Simmons et al., 2008); in contrast, judgements about distantly-associated words, which are thought to require additional semantic control processes (e.g., SALT-GRAN), were equally slowed by TMS delivered to LIFG and pMTG. These findings suggest that semantic control effects in pMTG in fMRI studies cannot be ‘explained away’ in terms of, for example, strong coupling with LIFG; instead both LIFG and pMTG appear to play an essential role in a distributed neural network underpinning semantic control (however, it remains unclear from these results whether these control processes are specific to language or extend to conceptual processing more widely; see below).

Recently, in an as yet unpublished study, we explored the same tasks in a combined fMRI–TMS investigation. We applied 1 Hz TMS to LIFG (BA45) and used fMRI to measure the effect on neural activity in other putative components of the semantic control network. Additional activation was seen in pMTG, compared to a baseline scan with no stimulation, and these changes were restricted to situations where the demand on the semantic control network was high. These results therefore strongly suggest that LIFG and pMTG are both components of a distributed cortical system underpinning semantic control: when the contribution of LIFG is disrupted via TMS, pMTG shows compensatory increases in activation.

One important yet unresolved issue is whether the sites within the semantic control network – i.e., LIFG, pMTG and dorsal AG plus IPS – make different contributions to semantic control. Although SA patients with LIFG and temporoparietal lesions have highly similar deficits, cases with LIFG lesions have greater difficulty inhibiting previously relevant semantic information – they show (i) more perseverations of their earlier responses in tasks like picture naming and (ii) declining accuracy in ‘cyclical’ tasks that present a set of semantically-
related items repeatedly, leading to a build-up of competition between previous and current targets (Jefferies et al., 2007; Gardner et al., 2012). Similarly, pMTG and sites within parietal cortex may play distinct roles in the regulation of semantic processing although these regions do not easily dissociate in studies of brain-injured patients. In fMRI, IPS is activated in tasks requiring the top-down selection of specific semantic features (Badre et al., 2005; Thompson-Schill et al., 1997), while pMTG is sensitive to cue-target associative strength, which influences the degree of stimulus-driven semantic control that is required (Badre et al., 2005; Wagner et al., 2001).

To develop a detailed understanding of the distinct roles of these regions, it is also imperative to examine the relationship between control over (i) amodal conceptual processing, (ii) semantic control specific to language and (iii) domain-general executive processes. SA patients show correlations between their scores on semantic and executive tests (Jefferies and Lambon Ralph, 2006) but their large lesions may mask potential dissociations between these functions. We have new and as yet unpublished data showing that patients with dys-executive syndrome (without aphasia) have semantic impairment that resembles SA: this suggests that ‘multiple-demand’ regions like inferior frontal sulcus, frontal operculum, dorsomedial prefrontal cortex and IPS, which are recruited during a wide variety of executively-demanding tasks (Duncan, 2010), make an important contribution to semantic control (see also Nagel et al., 2008). However, the involvement of some regions – e.g., LIFG and pMTG – may be more specific to semantic control (Nagel et al., 2008; Devlin et al., 2003; Gough et al., 2005). Therefore, in a recent TMS study (Whitney et al., 2012), we contrasted two different manipulations of semantic control (based on associative strength and feature selection respectively) with a non-semantic task involving the selection of specific visual features (based on Navon letters with opposing local and global features). We found an intriguing dissociation between LIFG/pMTG and IPS. While TMS to LIFG and pMTG disrupted both tasks requiring semantic control, leaving the non-semantic task unaffected, stimulation of IPS differed from this pattern in two ways: (i) it only influenced the selection of specific semantic features (e.g., matching ‘tomato’ with ‘stop sign’, based on shared colour), not the ability to detect global distant relationships (e.g., matching ‘salt’ with a relatively distantly-associated word, such as ‘grain’) and (ii) it disrupted both the semantic and non-semantic Navon trials, consistent with the notion that IPS forms part of the multiple-demand network. Our findings therefore provide support for a complex, distributed semantic control network that shows (i) some specialisation of function, such that different sites contribute to different aspects of semantic control and (ii) partial overlap with sites supporting domain-general executive control.

2.3. Summary of brain regions involved in semantic control

This review, focussed on our own research, suggests that the key regions involved in semantic control include LIFG, pMTG and dorsal AG/IPS. Binder et al.’s (2009) meta-analysis of neuroimaging studies of semantic cognition highlights similar regions, but provides a differing account of their functions. First, there is controversy about the role of the AG. Binder et al. (2009) propose that this region integrates multimodal semantic concepts into a single, coherent meaning, noting for example that this site shows late activation in sentence comprehension tasks, which is maximal at the point when the constituent words are linked together conceptually (Humphries et al., 2007). It is likely that AG contains several distinct functional regions (Seghier et al., 2010): for example, the dorsal AG bordering IPS might play a role in selection/inhibition in semantic and non-semantic tasks. In contrast, the more ventral AG might have a selective semantic integration role. Binder et al. (2009) note that “fluent conceptual combination” is an essential component of many tasks that require “problem solving and planning” and in this way, we might anticipate AG regions to contribute to executive aspects of semantic processing.

Our proposal that pMTG forms part of the semantic control network is perhaps the most controversial aspect of our account, since, as noted above, this area has been linked to the representation of tool, action and verb knowledge (Martin, 2007; Martin et al., 1995; Chao et al., 1999; Perani et al., 1999). A key issue for future research will be to determine whether the precise cortical areas that respond to tool/action knowledge and semantic control manipulations are overlapping or distinct, and to provide an explanatory framework for the overlap (should it exist). For example, it might be that both pMTG and IPL are involved in mental simulation of meaningful events/contexts (Schwartz et al., 2011), and that this type of simulation is helpful in many control-demanding semantic tasks.

Binder et al. (2009) additionally note that many fMRI studies yield significant and extensive activation in dorso-medial prefrontal cortex (BA8 and 9). This region has not been the focus of substantial research effort within the field of semantic cognition (including in our own studies), but nevertheless this site is linked to planning, attention and motivation and overlaps with the multiple-demand network of brain regions linked to domain-free executive function and fluid intelligence (Duncan, 2010).

3. Conclusion and future directions

Our research over the last 5 years has contributed to our understanding of the distributed neural network underpinning semantic cognition in several ways: (1) We have obtained convergent evidence from distortion-corrected fMRI and TMS studies of healthy volunteers that points to a role of both left and right ATL in the representation of word and picture meanings, in line with findings from investigations of patients with SD. (2) The distortion-corrected fMRI studies indicate graded specialisation of function within ATL, with more superior areas playing a greater role in semantic judgements about words, and inferior regions making a stronger contribution to semantic judgements about pictures. (3) We have shown that the degradation of semantic knowledge in SD is qualitatively different from the difficulties in semantic control shown by patients with multimodal semantic impairment following SA. This suggests that semantic cognition draws on
at least two interacting components—semantic representations (degraded in SD) and semantic control processes (deficient in SA). (4) SA patients have damage to different cortical regions beyond ATL—in particular, LIFG, posterior temporal and/or inferior parietal cortices. This suggests that a large-scale distributed cortical network underpins semantic control. We have again obtained convergent evidence for this view from fMRI and TMS. In our fMRI study, ATL was sensitive to the number of meanings retrieved, while pMTG and LIFG showed effects of semantic selection/inhibition. Moreover, TMS to LIFG and pMTG produced equal disruption of tasks tapping semantic control.

The next challenges are (i) to delineate the specific roles of each region within the semantic control network and (ii) to specify the way in which control processes interact with semantic representations. We have started to make progress on the first of these issues; however, many questions remain unanswered—for example, studies have not yet fully characterised the different functions of LIFG, pMTG and IPL and how they contribute to bottom-up and top-down aspects of semantic control, nor how these sites interact to give rise to control over semantic activation in different contexts. A related question is whether the semantic control network that underpins verbal tasks is identical to the network supporting control-demanding non-verbal semantic tasks; our theoretical account predicts that semantic control mechanisms are amodal but functional neuroimaging and TMS research has almost exclusively relied on verbal tasks, so the existence of separate linguistic control areas is currently hard to rule out.

We still know relatively little about how semantic control processes interact with the store of knowledge such that processing is focussed on task-relevant features, or how these task-relevant elements are integrated: this is likely to be an exciting avenue for research over the next few years. Progress may follow the use of experimental paradigms that manipulate control and representational demands simultaneously, and through the use of additional imaging methodologies, such as magnetoencephalography (MEG), that allow us to chart the interactions between brain regions with high temporal resolution.

Finally, we are starting to explore the practical implications of our research for the patients who initiated it. Our conclusion that multimodal semantic impairment in SA is linked to poor semantic/executive control has clear implications for speech and language therapy—in particular, it suggests that recovery may be facilitated by the use of training tasks that focus on strengthening semantic control processes, as opposed to attempts to retrain ‘lost’ semantic knowledge. We are starting to explore these ideas in studies that use tDCS to augment the effects of semantic and executive training.

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References


Noonan K, Jefferies E, Corbett F, Hopper S, and Lambon Ralph MA. Elucidating the nature of deregulated semantic cognition in semantic aphasia: Evidence for the roles of prefrontal and...


