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Talker-specific learning in amnesia: Insight into mechanisms of adaptive speech perception

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Abstract

A hallmark of human speech perception is the ability to comprehend speech quickly and effortlessly despite enormous variability across talkers. However, current theories of speech perception do not make specific claims about the memory mechanisms involved in this process. To examine whether declarative memory is necessary for talker-specific learning, we tested the ability of amnesic patients with severe declarative memory deficits to learn and distinguish the accents of two unfamiliar talkers by monitoring their eye-gaze as they followed spoken instructions. Analyses of the time-course of eye fixations showed that amnesic patients rapidly learned to distinguish these accents and tailored perceptual processes to the voice of each talker. These results demonstrate that declarative memory is not necessary for this ability and points to the involvement of non-declarative memory mechanisms. These results are consistent with findings that other social and accommodative behaviors are preserved in amnesia and contribute to our understanding of the interactions of multiple memory systems in the use and understanding of spoken language.

Keywords

speech perception; eye-tracking; accent; amnesia; episodic memory

1. Introduction

Human speech perception is remarkable in that it extracts meaning from a highly variable signal with speed and precision that defy even the most advanced speech recognition software. The variability inherent in speech is a classic example of a many-to-many

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mapping problem: Depending on the talker and context, different acoustic signals can map onto the same linguistic category; likewise, a particular acoustic signal can map onto different linguistic categories (Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967; Peterson & Barney, 1952). For example, native English speakers would consider the vowels in *tab* and *tack* to fall under the same linguistic category (e.g., the /æ/ sound), but when spoken, these vowels have different acoustic signatures because they precede different consonants. Conversely, a particular acoustic pattern might be categorized as either an *a* or an *eh* sound, depending on the speaker's identity and the way he or she pronounces these vowels. In principle, this many-to-many mapping should render the speech perception process impossible *unless* listeners learn how sound-to-meaning mappings change across talkers and contexts. The fact that human listeners rapidly adapt to new talkers suggests we do indeed learn these mappings. The key unanswered question is: *How?*

Although multiple theories of talker adaptation during speech perception have been proposed, their relationship to various memory systems has yet to be fully explored. Specifically, the respective roles of the declarative and non-declarative memory systems are not specified in these theories. One prominent theory of speech perception is the episodic theory, which proposes that every instance of speech is stored as a separate memory trace. These traces provide a record of how a particular acoustic signal was used by a particular talker in a particular context. When a new instance of speech is heard, contextually similar traces are activated proportionate to their similarity to the new sound, guiding comprehension of the unfolding speech sounds (Goldinger, 1998; Goldinger & Azuma, 2003). Although the name of this theory – and its proposal that “episodic memory traces” (Goldinger, 1996; 1998) of speech events are created– may lead one to believe that episodic memory, a form of declarative memory for autobiographical events and experiences (e.g., time-, place-, and person-specific information), underlies the storage of talker-specific information, this is not specified. One could imagine a version of this theory in which low-level, non-declarative associations accumulated with each new episode support this ability.

A second class of theories, broadly referred to as normalization theories, proposes that listeners compute talker-specific mappings or algorithms that support the ability to match the variable input received from multiple talkers onto invariant mental representations of phonological categories (Magnuson & Nusbaum, 2007). This class of theories posits reliance upon learned mappings or routines, which, to a first approximation, implicates procedural, non-declarative memory as the underlying mechanism. However, no specific memory mechanism has been proposed, and a version of these theories in which declarative memory subserves the learning of these mappings could also be imagined.

Currently, there is no direct evidence linking the learning of talker-specific mappings to either the declarative or non-declarative memory system, and evidence from healthy individuals is equivocal, as both systems may support learning. Here, we provide a test of the role of declarative memory by examining speech perception in amnesic patients who have severe declarative memory impairment following medial temporal lobe damage that prevents them from learning new episodic information while leaving non-declarative forms of memory intact.

In the present study, five amnesic and five healthy, demographically-matched comparison participants heard the speech of two unfamiliar talkers with different regional accents of American English. Crucially, the male talker pronounced words like *tack* and *tag* with different vowels, whereas the female talker used the same vowel. After a brief exposure to the two talkers' voices, each participant followed a series of instructions such as *Click on tack* while viewing a display containing images of a tack and a same-onset competitor word, such as tag (Dahan, Drucker, & Scarborough, 2008; Trude & Brown-Schmidt, 2012). We assessed participants' ability to distinguish between the initial sounds of the words *tack* and *tag* by monitoring eye-gazes to the corresponding images in the display. Previous research using this paradigm with neurologically intact, college-age participants has shown that listeners are able to learn and apply their knowledge about the male talker's accent and identify the target word more easily when listening to the male talker than the female talker due to the difference in the target and competitor words' vowels (Trude & Brown-Schmidt, 2012). Thus, successful learning predicts more looks to the target on male-talker trials, compared to female-talker trials. If declarative memory is necessary for how human speech perception adapts to variability across talkers, amnesic patients should fail to learn the male talker's accent, and therefore should perform equally with both talkers. However, if declarative memory is *not* necessary for this adaptation process, amnesic participants should perform like healthy participants.

2. Methods

2.1 Participants

Participants included five amnesic participants (1 female; mean age =51.8) with bilateral hippocampal damage (Figure 1)² due to anoxia (n=3), resulting in damage that was hippocampal circumscribed, or herpes simplex encephalitis (n=2), resulting in more extensive bilateral medial temporal lobe damage affecting hippocampus, amygdala, and surrounding cortices (Table 1). Neuropsychological testing confirmed a severe and selective declarative memory impairment (mean WMS-III GMI =61.4) in the context of preserved intelligence (mean WAIS-III FSIQ =97.6). Previous work with the same amnesic participants studied here revealed sparing of non-declarative memory across a broad range of perceptual-motor tasks (Cavaco, Anderson, Allen, Castro-Caldas, & Damasio, 2004). Participants were free of aphasia. Comparison participants were five healthy individuals matched pair-wise to amnesic participants on age, sex, education, and handedness. All were native, monolingual American English speakers with normal or corrected-to-normal hearing and vision. Although participants were not explicitly queried about their familiarity with the critical accent, none had spent a significant amount of time in any region where the critical accent is common.

2.2 Stimuli

Acoustic stimuli were produced by two native English speakers who did not interact with the participants: a male from Oregon with a regional accent in which /æ/ raises to [eɪ] only

²Patient 2563 wears a pacemaker and was unable to undergo the structural magnetic resonance imaging examination. Anatomical analyses from a computerized tomography scan revealed damage only to the hippocampal region bilaterally.

before /g/, and a female from the Chicago area who did not exhibit the critical vowel shift (Trude & Brown-Schmidt, 2012).

Participants heard the two talkers at training and test. The test stimuli were eleven pairs of same-onset, monosyllabic English words ending in /æg/ and /æk/ (e.g., *tag* and *tack*). Each pair was matched with two same-onset filler words that ended in /g/ and /k/, but did not contain /æ/ or /eɪ/ vowels (e.g., *wig* and *wick*), for a total of 44 stimuli (see Appendix for a complete list of stimuli). The training stimuli consisted of a four-minute scripted dialogue in which each talker produced the 11 *-ag* words in sentence contexts. Images used during the eye-tracking task were color drawings chosen to provide the clearest possible depiction of each word.

2.3 Equipment and Procedure

Participants completed three experimental sessions. Time between sessions varied across participants. Training was repeated if more than 10 minutes elapsed between sessions for amnesic participants or if more than 24 hours elapsed between sessions for comparison participants. The experiment was programmed in Matlab using the Psychophysics Toolbox (PTB-3, Brainard, 1997; Pelli, 1997).

2.3.1 Training—Participants were first familiarized with the word-picture pairings to be presented at test. Each picture was displayed on screen with its name above it and a context-providing sentence below it. After viewing each picture-name pairing once, participants completed a recognition task in which four pictures appeared on screen, along with one picture's name. Participants were instructed to click the picture that matched the name. All participants correctly identified the pictures to at least a 90% criterion on their first attempt. Participants then listened to the training dialogue. No additional task was imposed on the participants while listening.

2.3.2 Test—Participants' eye movements were recorded using an Eyelink 1000 desktop-mounted eye-tracker, which sampled eye movements monocularly at 1000 Hz. Participants viewed a fixation cross for 1000 ms, then viewed a display containing pictures of one of the eleven word sets. Each display contained an *-ag* word, *-ack* word, and two fillers (Figure 2). After 2000 ms, participants heard one talker say *Click on*, followed by the target word, and then clicked the named picture. Participants were given unlimited time to respond. All audio was played through desktop speakers. Each session consisted of 352 trials³. Each word was the target eight times (produced four times by each talker). The trial order, as well as the position of the 4 images on each display screen, was randomized separately for each participant. The randomization of the trial order ensured that participants could not predict which talker would speak next.

³Due to a technical problem, Patient 1951 received only 118 trials during the first experimental session. Sessions 2 and 3 contained the full 352 trials.

3. Results

Analyses of the eye-tracking data focused on a time window of 200–1200 ms following critical word onset (e.g., *tack*). The analysis window was offset by 200 ms due to the time needed to program and launch an eye movement (Hallett, 1986). The target advantage score (the proportion of fixations to the target, e.g., *tack*, minus the proportion of fixations to the competitor, e.g., *tag*) was calculated by item for each participant. Data were analyzed with multilevel modeling using the lmer function in the lme4 software package in R (Bates, 2007). The best-fitting model contained group (patient vs. comparison) and talker (male vs. female) as independent variables and crossed random intercepts for subjects and items. Backwards model comparison determined that random slopes for the fixed effects did not improve model fit ($\chi^2_{(3)} < 3.6$, p 's > 0.3). Significance of fixed effects was determined based on 10,000 samples of the posterior distribution (Monte Carlo Markov Chain sampling, pMCMC).

Eye-gaze analyses showed that both groups rapidly learned and applied information about the two talkers' speech in time to guide speech perception (Figures 3 and 4). A significant effect of talker was found ($t = 3.88$, $p\text{MCMC} < .001$, Coef. $\beta = .09$, $SE(\beta) = .02$), such that, as predicted, participants fixated the target image (*tack*) more and the competitor image (*tag*) less when spoken by the male talker, for whom *tack* and *tag* were pronounced with different vowels, compared to the female talker who pronounced the words with the same vowel ($M = .28$ and $M = .19$, respectively). The interaction between talker and participant group was *not* significant ($t = -.051$, $p\text{MCMC} = .61$, Coef. $\beta = -.02$, $SE(\beta) = .05$), indicating that the magnitude of the effect was similar for patients (mean diff = .08) and comparisons (mean diff = .10). Further supporting the conclusion that the participant groups did not differ was the finding that random slopes by participant for the talker effect did not improve model fit ($\chi^2_{(1)} = .61$, $p = .74$). The time-course graphs (Figure 4) indicate that both groups' eye movements followed a similar pattern. Participants gradually fixated the target more and the competitor less as more of the word was heard. In the case of the male talker, participants were able to disambiguate the words earlier and at a more rapid rate due to the male talker's differing vowels in -ack and -ag words.

Interestingly, while amnesic patients successfully learned to capitalize on the talker-specific accent characteristics, they experienced slightly more difficulty in the eye-tracking task than comparison participants, evidenced by a lower overall target preference regardless of talker ($t = -2.70$, $p\text{MCMC} < .01$, Coef. $\beta = -.08$, $SE(\beta) = .03$)⁴. The fact that the amnesic patients successfully learned the talker-specific information despite being more challenged by the task makes the learning effect even more impressive, and suggests that while the declarative memory system may not play a critical role in accent adaptation, it may play a role in more basic processes such as the real-time integration of spoken language with information from the visual world (Duff & Brown-Schmidt, 2012). In summary, our results demonstrate that declarative memory cannot be the critical mechanism supporting the learning of talker-

⁴Inspection of the data pattern of individual patients revealed that four out of five patients showed the learning effect; the fifth patient, 2308, did not show the learning pattern. Note that the lower across-condition performance by the patient group is not driven by this patient; a post-hoc analysis removing this patient and his matched comparison shows the same pattern of data ($t = -1.92$, $p\text{MCMC} < .08$, Coef. $\beta = -.08$, $SE(\beta) = .04$).

specific mappings, as amnesic patients, who have severe episodic memory impairments, were as successful as comparison participants in learning to distinguish the accents of two unfamiliar talkers.

4. Discussion

Despite significant impairment in episodic memory, amnesic patients showed rapid, talker-specific adaptation to a novel accent of an equivalent magnitude as healthy comparison participants. That this learning was successful shows that amnesic individuals and healthy comparison participants alike learned arbitrary talker-accent pairings and used this knowledge of which accent went with which talker to guide on-line speech perception. This learning effect is particularly striking given that amnesic patients exhibited more difficulty in resolving the lexical competition overall. The following discussion highlights the significance of these results with respect to models of speech perception, the relationship to social adaptation, and the memory-language interface.

4.1 What memory systems support talker adaptation?

Our findings demonstrate that declarative memory is not necessary for adaptation to talker-specific variability in speech perception. This result therefore rules out versions of accounts of talker-specific adaptation that point exclusively to declarative (episodic) memory as the underlying memory mechanism. How, then, do humans rapidly adapt speech perception to new talkers and new contexts? One explanation is that a fast, non-declarative learning process such as priming could be responsible. Indeed, previous research has demonstrated that, similar to healthy participants, amnesic patients can be primed to produce a particular syntactic structure (Ferreira, Bock, Wilson, & Cohen, 2008). Similarly, in the current experiment, patients may have been primed to associate the male talker's pronunciation of the raised [eɪ] vowel with the words (or images depicting) *bag*, *rag*, *tag*, etc., and associate his pronunciation of the /æ/ vowel with the words *back*, *rack*, *tack*, etc. Whether the learning effect would generalize to unstudied words that follow the pattern of the accent (e.g., *crag*, *diagonal*, *agriculture*) may speak to whether the underlying mechanism primarily reflects abstract learning about phonological structure (e.g., Ferreira et al., 2008) or is primarily driven by item-specific associations (e.g., the *lexical boost*; Branigan, Pickering, & Cleland, 2000).

Additional learning mechanisms may have supported the amnesic patients' success as well. Healthy comparison participants and patients alike may have rapidly adjusted to the male talker's accent through a re-mapping of their existing phonological knowledge (Davis, DiBetta, Macdonald, & Gaskell, 2009). Because such a re-mapping process could rely on procedural memory and would not require the learning of completely novel information, this may be a plausible mechanism for successful learning in amnesia (see Duff, Hengst, Tranel, & Cohen, 2006). The fact that learning could draw on existing phonological knowledge, together with the fact that talkers are rich contextual cues used in every-day life may have contributed to the successful learning of arbitrary talker-accent pairings here. By contrast, learning of completely novel information may be more dependent on declarative memory (e.g., Chun & Phelps, 1999).

More generally, our findings fall naturally from the complementary learning systems account of memory, which proposes that in healthy individuals, the declarative and non-declarative memory systems complement each other, with each system demonstrating some specialization in the acquisition and processing of different forms of knowledge over distinct time scales (McClelland, McNaughton, & O'Reilly, 1995). Consistent with our findings, complementary learning systems theory proposes that learning about a language's sound system may take place independently of the declarative memory system because it is involves reliable rules and mappings (Gupta, 2012). Conversely, learning the meanings of new words may rely more heavily on the declarative memory system due to the arbitrary relationship between the way a word sounds and its meaning. On such a view, amnesic participants were successful in the present task precisely because it drew on regularities in the sound system of the language. By contrast, learning of arbitrary mappings between talkers and words (Creel, Aslin, & Tanenhaus, 2008) would likely present these patients with significantly more difficulty, as it would rely on arbitrary semantic mappings, rather than systematic phonological rules.

In more recent years, a number of additional neurocognitive models of memory and category learning have emerged that could also explain our results. One such model implicates the striatum in the processes of working memory maintenance, as well as context-based learning (Ashby & Crossley, 2011; Ashby, Ell, Valentin, & Casale, 2005), while another theory suggests that the cerebellum is responsible for fast category learning (Wolpert, Diedrichsen, & Flanagan, 2011). Both of these brain regions are intact in the patients in the current study, making them possible loci for learning processes required during perceptual adaptation. However, neither of these theories has been directly tested in the domain of speech perception; thus, more research is necessary in order to evaluate the roles of the striatum and cerebellum in this area.

Why did one amnesic patient (2308) fail to learn the talker-specific accents? Patient 2308, like patient 1951, has more extensive bilateral medial temporal lobe damage (including to the amygdala) than the anoxic patients. Given the intact learning of 1951, however, we can rule out the notion that 2308 failed to learn because of the amygdala damage or extra-hippocampal MTL damage. That said, there are anatomical differences that may account for the aberrant performance of patient 2308. While both 2308 and 1951 have asymmetrical lesions (2308 has more extensive damage on the left extending to left temporal pole and lateral cortices; 1951 has more extensive damage on the right extending to right temporal pole and lateral cortices), 2308 is left-handed and 1951 is right-handed. An open question is the extent to which interactions between language lateralization/handedness and side/extent of lateral temporal lobe damage, specifically those areas involved in phonological processing (e.g., STG, Hickok & Poeppel, 2007), may account for the lack of learning in 2308. Future exploration of this possible link is warranted.

4.2 Social adaptation

A particularly surprising aspect of our results was that patients were successful despite the fact that our task introduced an element of arbitrariness, in that the male talker spoke with an accent and the female talker did not. After all, a hallmark of the hippocampal declarative

memory system is its ability to support the learning of arbitrary mappings. To succeed in the task, participants had to learn the (arbitrary) fact that the *male* talker had the accent, and not the female talker. The patients' ability to learn this talker-to-accent mapping is consistent with preserved learning of person-specific social information in amnesia (e.g., who is a good or bad guy; Coronel et al., 2012; Johnson, Kim, & Risse, 1985; Todorov & Olson, 2008; Tranel & Damasio, 1993). Indeed, the learning of indexical social and linguistic information may share common mechanisms. Indexical features of speech influence social interaction, with some evidence suggesting that an accent can affect how likely a talker is to converge his or her own speech toward the speech of an interlocutor (Giles, 1973; Willemyns, Gallois, Callan, & Pittam, 1997). Similarly, accents can affect how attractive or friendly a listener finds a talker to be (Raisler, 1976). The inherently social nature of language tasks may support learning through recruitment of additional learning mechanisms or routines. For example, whereas amnesic patients fail to learn arbitrary experimenter-generated object labels, they are successful in a collaborative language task (Duff et al., 2006) and show normal convergence with an interactional partner in conversation (Gupta, Tranel, & Duff, under review).

4.3 The memory-language interface

In conclusion, human speech perception is successful because it rapidly adapts to enormous variability between talkers and contexts. This type of learning supports communication and may share common mechanisms with other social and accommodative behaviors. By exploring the relationship between memory and language, we have provided critical information that can help to theoretically constrain models of speech perception, demonstrating that declarative memory is not necessary for learning talker-specific mappings between the acoustic signal and linguistic categories. Instead, rapid, non-declarative mechanisms such as priming and procedural learning likely support the accommodation of variability in speech perception. While talker-specific adaptation was not impaired in amnesia, overall speech perception was impaired. This result is consistent with our proposal (Duff & Brown-Schmidt, 2012) and recent empirical work (Kurczek, Brown-Schmidt, & Duff, 2013) suggesting that the ability to rapidly integrate the unfolding linguistic signal with contextually instantiated referential candidates draws on the hippocampal-dependent declarative memory system. This duality of spared and impaired function in language processing in amnesia highlights the significance of understanding how multiple memory systems, operating at different timescales and with different neural substrates, interface with linguistic representations during the real-time processing of language.

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Appendix. Complete list of stimuli used in the experiment

<i>-ack word</i>	<i>-ag word</i>	<i>-k filler</i>	<i>-g filler</i>
back	bag	lock	league
flack	flag	check	chug
jack	jag	dock	dog
lack	lag	smoke	smog
rack	rag	lick	lug
sack	sag	pluck	plug
shack	shag	pick	pug
snack	snag	buck	bug
stack	stag	tuck	tug
tack	tag	wick	wig
whack	wag	jock	jog

Figure 1.

Magnetic resonance scans of amnesic patients. Images are coronal slices through the midportion of the hippocampus from T1-weighted scans. Volume changes can be noted in the region of the hippocampus bilaterally.

Figure 2.

A sample display from the test portion of the experiment. All displays included a target word (*tack*), a same-onset competitor (*tag*), and two unrelated filler items (*wick* and *wig*).

Figure 3.

Patients' and comparisons' preference to fixate the target picture (e.g., *tack*) when listening to the (male) accented and (female) unaccented talkers. Error bars represent the standard error of the mean calculated by item.

Figure 4.

Time course of patients' (L) and comparisons' (R) fixations to targets (e.g., *tack*) and cohort competitors (e.g., *tag*) when listening to the (male) accented and (female) unaccented talkers, with 0 ms indicating the onset of the target word.

Table 1
Demographic, anatomical, and neuropsychological data characterizing amnesic patients.

Demographic				Anatomical			Neuropsychological					
Patient	Sex	Hand	Age	Edu	Etiology	HC Vol	WAIS-III FSIQ	WAIS-III Vocab	WAIS-III Similar	WAIS-III Inform	WMS-III GMI	CFT Copy
1846	F	R	49	14	Anoxia	-4.23	84	8	7	8	57	28
1951	M	R	60	16	HSE	-8.10	106	10	13	11	57	32
2308	M	L	56	18	HSE	N/A	98	11	9	8	45	32
2363	M	R	56	18	Anoxia	-2.64	98	12	12	13	73	26
2563	M	L	57	16	Anoxia	N/A	102	9	7	12	75	36

F, female; M, male; Hand, handedness; Age, years; Edu., education, years; HSE, herpes simplex encephalitis; HC Vol = studentized residual differences in hippocampal volume relative to a matched comparison group (see Allen et al., 2006; Buchanan et al., 2005); WAIS, Wechsler adult intelligence scale-III; FSIQ, full-scale IQ; Vocab, vocabulary; Similar, similarities; Inform, information; WMS-III, Wechsler memory scale-III; GMI, general memory index; CFT, complex figure test.