



Research report

Discrimination of brief speech sounds is impaired in rats with auditory cortex lesions

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ABSTRACT

Auditory cortex (AC) lesions impair complex sound discrimination. However, a recent study demonstrated spared performance on an acoustic startle response test of speech discrimination following AC lesions (Floody et al., 2010). The current study reports the effects of AC lesions on two operant speech discrimination tasks. AC lesions caused a modest and quickly recovered impairment in the ability of rats to discriminate consonant–vowel–consonant speech sounds. This result seems to suggest that AC does not play a role in speech discrimination. However, the speech sounds used in both studies differed in many acoustic dimensions and an adaptive change in discrimination strategy could allow the rats to use an acoustic difference that does not require an intact AC to discriminate. Based on our earlier observation that the first 40 ms of the spatiotemporal activity patterns elicited by speech sounds best correlate with behavioral discriminations of these sounds (Engineer et al., 2008), we predicted that eliminating additional cues by truncating speech sounds to the first 40 ms would render the stimuli indistinguishable to a rat with AC lesions. Although the initial discrimination of truncated sounds took longer to learn, the final performance paralleled rats using full-length consonant–vowel–consonant sounds. After 20 days of testing, half of the rats using speech onsets received bilateral AC lesions. Lesions severely impaired speech onset discrimination for at least one-month post lesion. These results support the hypothesis that auditory cortex is required to accurately discriminate the subtle differences between similar consonant and vowel sounds.

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1. Introduction

Bilateral auditory cortex lesions cause long-lasting impairments in the discrimination of complex sounds. Lesioned animals are impaired in the discrimination of frequency patterns [3], vowel-like stimuli [4], and animal vocalizations [5–7], with little recovery even a month later. Japanese macaques are unable to discriminate between coo vocalizations even years after receiving bilateral auditory cortex lesions [7]. Rats' auditory cortex neurons encode speech sounds in a manner that is highly correlated with the rats' ability to discriminate these sounds [2]. These results suggest that the auditory cortex serves a critical role in the discrimination of complex sounds.

However, a recent study reported that bilateral auditory cortex lesions do not impair consonant discrimination by rats [1]. This result seems to conflict with earlier studies suggesting that the auditory cortex is necessary for discriminating complex stimuli. One possible explanation for the spared speech discrimination following auditory cortex lesions is that the rats were tested with

pre-pulse inhibition in an acoustic startle reflex paradigm, while previous lesion studies reporting deficits for complex stimulus discriminations used operant training methods. An alternate explanation is that the rats were able to quickly adapt their strategy to use some cue present in the speech sounds that does not require the auditory cortex to discriminate, such as amplitude [8–10] or duration differences [11]. To test these potential explanations, we conducted two experiments. Experiment 1 tested whether auditory cortex lesions impair rat performance on an operant speech discrimination task. Experiment 2 tested whether auditory cortex lesions impair performance on an operant speech discrimination task in which the speech stimuli were truncated to reduce potential acoustic cues that could be used to accomplish the task.

2. Methods

2.1. Experiment 1

2.1.1. General procedure

Rats learned to perform a Go/No-Go procedure in three phases: shaping, detection, and discrimination testing. During the shaping phase, the rats learned to press a lever for a food pellet reward. In the detection phase, the rats learned to press the lever only after a presentation of the target sound, “dad”. Pressing the lever without the presentation of a target sound resulted in a brief period during which the rats did not receive sugar pellets and the lights were extinguished to serve as negative

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reinforcement. Once the rats consistently pressed the lever as a response to the target stimulus, the discrimination testing phase began. In discrimination testing, the rats had to press the lever to the target stimulus, “dad”, and ignore presentations of the distracting stimulus, “tad”. A past study using the identical stimuli showed discriminating the voiced “dad” from the unvoiced “tad” was a moderately difficult but reliably accomplished task [2]. Discrimination testing occurred in quiet and in four levels of white noise (48 dB SPL, 60 dB SPL, 72 dB SPL, or 84 dB SPL) presented as blocks within a session. Addition of the background noise helped to avoid a ceiling effect in discrimination performance by creating multiple levels of difficulty. After learning the discrimination task, rats received bilateral aspiration lesions to the auditory cortex and continued to test on the same task for an additional fifteen days.

2.1.2. Subjects

This experiment used three female Sprague-Dawley rats averaging 363 g (standard deviation 21 g). Rats’ activity increases during the dark cycle of the day; therefore, they were housed in a 12:12h reversed light cycle environment to increase activity during daylight hours. Rats were food deprived to not less than 85% of their normal body weight for the purpose of motivation. Rats had free access to water at all times except during testing. The University of Texas Institutional Animal Care and Use Committee approved all handling, housing, surgical procedures, and behavioral testing of the rats.

2.1.3. Apparatus

All training and testing sessions occurred in double-walled sound booths designed to reduce outside noise. Booths were 67 cm × 67 cm × 67 cm at the outer dimension with a 20 cm × 20 cm × 20 cm wire cage inside. A pellet dispenser provided 45 mg sugar pellets through a tube into the booth that attached to a pellet dish on the inside of the cage. The dispensing mechanism was attached to the outside of the sound booth to reduce the noise associated with the delivery of a pellet. A lamp located above the pellet dish indicated the beginning of a trial and a lever located to the right of the pellet dish provided the rats a means to respond. Roughly 20 cm to the left of the rats’ estimated head position was a Tucker-Davis Technologies FF1 free-field, flat spectrum speaker that played stimuli to the rats. A lamp located in the booth provided additional light during sessions. Programs created in MATLAB were used to run sessions and collect data via a Tucker-Davis Technologies RP2.1 real-time processor connected to an Hewlett Packard personal computer.

2.1.4. Stimuli

Two of the exact stimuli used by Engineer et al. [2], “dad” and “tad” were chosen for the discrimination task. These words differ only in the presence or absence of voicing in the initial consonant, and not the place of articulation of the initial consonant, vowel, or terminal consonant sound. An ACO Pacific microphone taking samples every 10 μs with 32-bit resolution recorded a native English-speaking female saying the words. The STRAIGHT vocoder program was used to shift the fundamental frequency and spectrum envelope of each speech sound higher in frequency by a factor of two to approximate the rats hearing range [12,13]. Amplitude levels from the most intense 100 ms of each sound were calibrated to 60 dB. The stimuli were presented randomly within blocks consisting of six target “dad” stimuli, six distracting “tad” stimuli, and one silent catch trial.

During behavior, a program created in MATLAB generated a white-noise background for each block of trials. The same microphone used in creating the speech stimuli was also used to calibrate noise levels at 48, 60, 72, and 84 dB SPL. Linear ramps using one-fifth steps occurred between noise levels to reduce stress to the rats by removing sudden amplitude increases. Pilot studies had revealed that the rats quickly habituate to the noise and demonstrate no behavioral aversions to even the loudest intensities. The first block of trials occurred in quiet. Afterwards, the silent background block was randomly interleaved with the four noise blocks throughout the remainder of the session to create five background noise conditions.

2.1.5. Behavioral testing procedure

Rats began by training twice daily for 30 min, five days a week on a go/no-go procedure. Initial training focused on shaping the rats to press the lever for a food reward. After being introduced to the cage, rats explored the area and received a sugar pellet reward whenever they approached the lever. Any time a pellet dispensed during the shaping phase, the target “dad” sound played over the speaker. Requirements for a reward were gradually restricted in regards to the rats’ proximity to and actions involving the lever so the rat had to be close to the lever, then touching the lever, and finally pressing the lever to receive a sugar pellet. Rats generally started to press the lever for a sugar pellet within a single training session and trained to a criterion of two consecutive sessions of 100 sugar pellets earned by lever press within four one-half hour training sessions.

After reaching the criterion for learning to press the lever, training was increased to two 60-min sessions per day (five days per week), during which the rats learned to press the lever only in response to a presentation of the target “dad” sound. The target sound was no longer played in response to a lever press, but acted as the cue for the rats to press the lever. Silent trials were interleaved with presentations of the target sound as catch trials. Catch trials accounted for 25% of the trials, and ensured the rats were responding to the target sound and not randomly pressing the lever. Responses to the silent catch trials resulted in a time out, during which lamps in the

booth were extinguished and the beginning of the next trial delayed. Rats did not receive any food during time out periods. Sessions started with either a target sound or a silent catch trial occurring every 10 s, allowing 8 s for the rats to respond, and with an 8-s timeout period for false alarms. As the rats’ performance progressed, trial spacing was eventually reduced from every 10 s to 8 s, the hit window reduced from 8 s to 3 s, and the timeouts reduced from 8 s to 6 s to allow for optimal training efficiency. Rats were unable to respond in less than 0.3 s from the beginning of a trial, therefore the hit window did not begin until after this period.

Rats demonstrated a reliable response to the target sound within 40 sessions by reaching a d' value of 1.5 for 10 consecutive sessions, after which they advanced to discrimination testing. All the parameters remained the same in discrimination testing as they were in detection training, except for the addition of the distracting sound, “tad”, and white noise. Stimuli were presented in blocks of thirteen trials, with each block occurring in one of five, randomly presented noise conditions (silence, 48 dB SPL, 60 dB SPL, 72 dB SPL, 84 dB SPL). The rats were previously used in experiments using other noise levels in unrelated experiments before entering the 98 pre-lesion testing sessions reported on here (one rat tested for ninety-six sessions). Evaluation of the rats’ performance used percent correct, which averaged the percent of responses to the target sound and the percent of correct rejections for the non-target sound (excluding the catch trials).

2.1.6. Auditory cortical lesions

Bilateral lesions were administered to determine the role of AC in the operant discrimination of speech sounds varying in the initial consonant. All surgeries used sterilized instruments in a clean environment. After rats received an initial anesthetic dose of sodium pentobarbital (50 mg/kg, i.p.) they were placed in a Kopf Stereotaxic device. Doses of atropine (0.54 mg, s.c.), dexamethasone (2 mg, s.c.), cefotaxime sodium (20 mg, s.c.), and 10 ml of dextrose/Ringer’s solution (s.c.) given to the rats before and during the surgery reduced inflammation, prevented infection, and provided nourishment and fluids throughout the surgery and recovery. Bupivacaine (1 ml, s.c.) injections into the scalp acted as a local anesthetic at the beginning and end of the surgeries to ensure the rats felt no pain. Body temperature was maintained at 37 °C and the application of ophthalmic ointment prevented corneal drying. The initial incision was made mid-line on top of the skull. After resecting the temporalis muscle, micro-rongeurs were used to remove a piece of the skull, as delineated by the space between the temporal ridge, coronal suture, and the lateral suture. The exposed area of the brain is known from neurophysiology to contain primary auditory cortex (A1) and most of the surrounding auditory fields. After removing the dura mater, an aspiration lesion was used to remove the cortex to the approximate level of the hippocampus as judged by visual inspection of the tissue and a rough depth estimate. Removal of the entire exposed auditory cortex ensured that A1 was lesioned. Bleeding stopped through natural clotting and the skull piece was replaced. The procedure was then replicated on the other side of the brain within the same surgical session. After the completion of both lesions and the replacement of both skull pieces, a loose stitch was made connecting the two temporalis muscles that helped to hold the skull pieces in place. The skin was sutured and a topical antibiotic ointment applied. Rats recovered for one week with free access to food before testing began again. Rats received amoxicillin (5 mg) and carprofen (1 mg) in tablet form for the three days immediately following the surgery.

2.2. Experiment 2

2.2.1. General procedure

Behavioral methods for Experiment 2 closely approximated Experiment 1 with two changes: no background noise was delivered and a greater number of distracting stimuli were delivered. In Experiment 2, rats discriminated “dad” from “bad”, “sad”, “tad”, “dood”, “deed”, and “dud”. Half of the rats were tested on speech sounds that were limited to the first 40 ms of the speech sounds and the other half tested on the full-length speech sounds. After 20 days of discrimination testing, the rats that tested using the shortened stimuli received either bilateral auditory cortex lesions or sham lesions. The rats were allowed to recover for one week following the surgical procedure and then continued testing for an additional 15 days.

2.2.2. Subjects

Experiment 2 used 24 female Sprague-Dawley rats averaging 264 g (standard deviation 15 g). Housing conditions and food deprivation protocols were the same as Experiment 1.

2.2.3. Stimuli

Two lists of stimuli were created for comparing the effects of onset speech sound discrimination following lesioning, and onset speech discrimination compared to full-length speech sound discrimination before lesioning. Creation of both lists followed the same initial steps. A native English speaking female was recorded saying the consonant–vowel–consonant sounds “dad”, “bad”, “sad”, “tad”, “dud”, “deed”, and “dood”. Engineer et al. [2] used four of the same stimuli varying in the initial consonant place (“dad”, “bad”, “sad”, and “tad”). Three additional stimuli varying in the vowel place introduced a greater range of testing to the lists (“dud”, “deed”, and “dood”). The speaker and recording procedures were the same as Experiment 1. Following amplitude calibrations, one list remained at full-length and the second list was truncated to the first 40 ms following sound onset (see Fig. 1). The duration

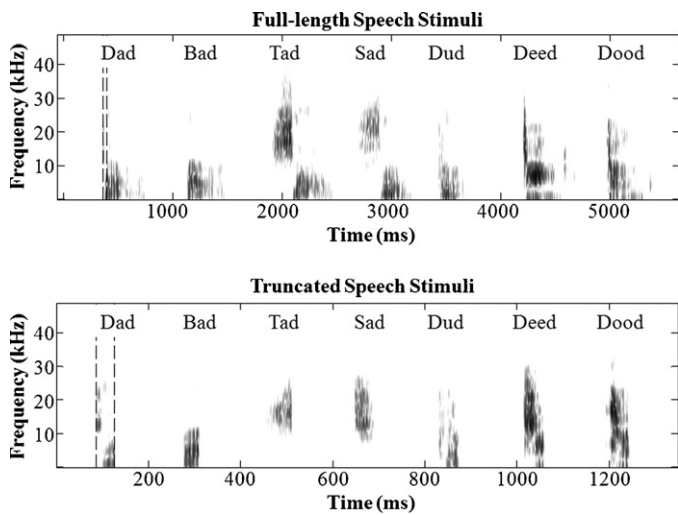


Fig. 1. Spectrograms of the speech stimuli used. Spectrograms on the top represent the full-length stimuli and spectrograms on the bottom represent the same stimuli truncated to the first 40 ms of the sound. In Experiment 1, the full-length “dad” was discriminated from the full-length “tad” stimuli. In Experiment 2, the full-length trained group discriminated the full-length “dad” from the full-length versions of “bad”, “tad”, “sad”, “dud”, “deed”, and “dood”. The onset trained group in Experiment 2 discriminated the first 40 ms of “dad” from the first 40 ms of “bad”, “tad”, “sad”, “dud”, “deed”, and “dood”. The space between the two dotted lines represents a 40 ms duration. The speech sound represented is listed above each spectrogram.

of cortical activity that correlated with behavioral discrimination in an earlier study served as the basis for the 40 ms duration [2]. This duration is also similar to the duration limits suggested by psychophysical studies in humans and tests two of the consonants and two of the vowels used by Blumstein and Stevens [14].

2.2.4. Behavioral testing procedure

Behavioral testing in Experiment 2 followed the same basic procedures and used the same apparatus as Experiment 1. Rats were trained using stimuli limited to the speech onsets, referred to as the “onset trained group”, or the full-length versions of the sounds, referred to as the “full-length trained group”. During the discrimina-

tion testing phase, rats heard full-length or truncated stimuli in blocks of thirteen, with the target version of “dad” presented six times, versions of the six distracting sounds “bad”, “sad”, “tad”, “dud”, “deed”, and “dood”, presented once, and one silent catch trial. Stimuli were presented in random order within each block. Unlike the first experiment, no noise was presented in the background. The full-length trained group tested for 20 sessions after which most rats demonstrated a plateau in their performance. Rats in the onset trained group tested for an additional 20 sessions to reach a consistent level of discrimination. Calculations for percent correct were the same as Experiment 1 using all the distracting sounds in place of the single “tad” sound (excluding the catch trials). The same formula was used to calculate performance scores for individual contrasts, e.g. responses to “dad” compared to “sad”.

After the last testing session, the onset trained group was randomly divided into two smaller groups of six. To test the effects of auditory cortex lesions on speech onset discrimination, one group received bilateral auditory cortex lesions focused on destroying A1, the lesion group, while the second group received sham surgeries, the sham group. Following a week of recovery, all rats began testing again for an additional 15 days using the 40 ms speech stimuli.

2.2.5. Auditory cortical lesions

Six rats from the onset trained group received bilateral auditory cortex lesions (Fig. 2) while the other six received sham surgeries. All surgical procedures for lesioned rats were the same as Experiment 1. Previous research has indicated that penetrating the skull can result in neurochemical changes in the underlying brain regions [15]. Sham surgeries were similar to the lesion surgeries with the exception that the skull was not penetrated to avoid unwanted cortical damage.

2.3. Histological procedures

Following the last testing session, the rats from Experiments 1 and 2 were deeply anesthetized with sodium pentobarbital (50 mg/kg, i.p.). Once the rats were unresponsive to toe pinch, they were transcardially perfused with 250 ml of 0.1 M PB solution with 0.02% heparin, followed by 500 ml of 4% formalin solution in 0.1 M PB. Brains were cryoprotected in a 30% sucrose solution for 2 days after removal from the skulls. Sections were taken at 40 μ m intervals and stained with Cresyl Violet. Four sections used to define the lesion were spaced at 1 mm intervals and matched to reference sections in a widely used brain atlas for the rat [16]. Sections were then scanned into a computer, where a recreation of the lesion was created using Adobe Photoshop CS3 and the selected atlas slides. Several studies have suggested that the defined area for A1 in the cited atlas does not accurately represent A1 based on the neuronal response characteristics [1,17]. The coordinates from Polley et al. [18] agree well with the coordinates for A1 defined in an earlier lesion study [1]. These coordinates provided the approximate location of the neurophysiologically defined A1 used in this study.

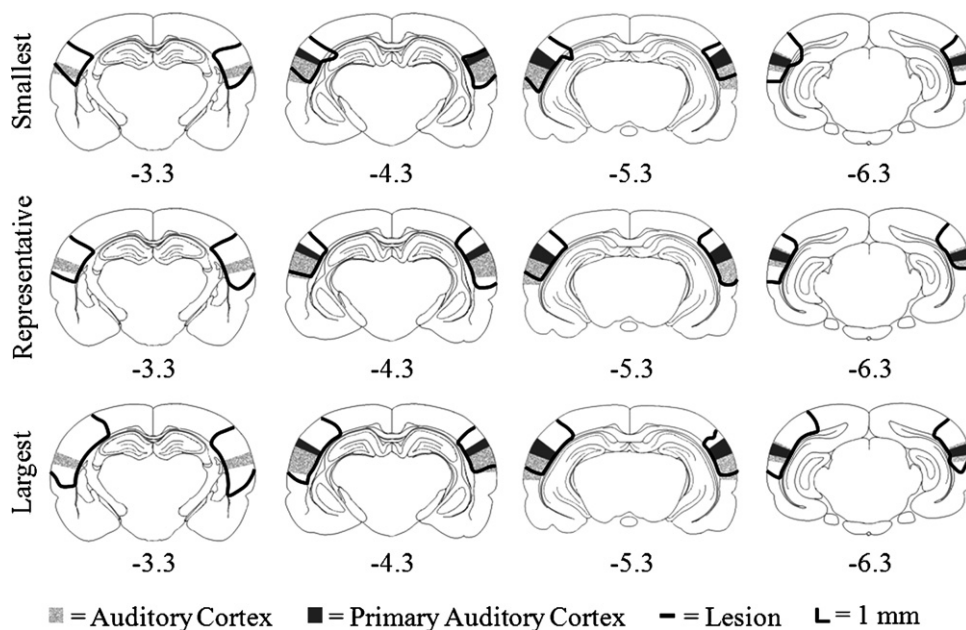


Fig. 2. Estimated lesion locations on horizontal sections at -3.3 , -4.3 , -5.3 , and -6.3 mm from bregma of a rat with a lesion representative of the groups in Experiment 1 and 2, and the two rats with the smallest and largest lesions. Five of the nine lesioned rats had their brains stained with Cresyl Violet for comparison. Visual inspection of the remaining four rats’ brains revealed a similar pattern of destruction. Stippled areas in light grey represent the location of auditory cortex and the dark grey shaded area represents primary auditory cortex determined using coordinates from Polley et al. [17]. Areas outlined in a bold, black line represent traces of the destroyed cortex. Scale bars located in the bottom right hand corner of the figure represent 1 mm.

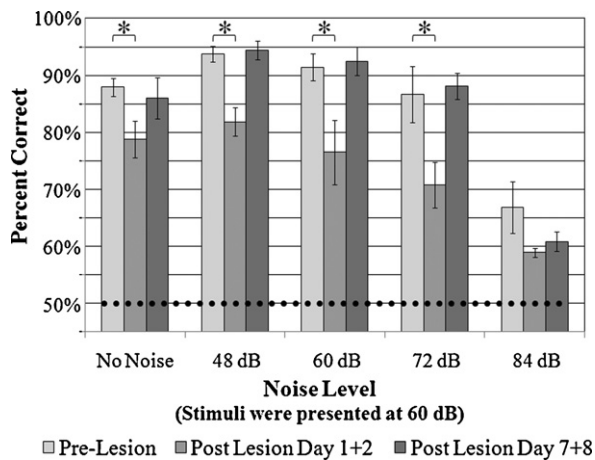


Fig. 3. Percent correct discrimination of “dad” and “tad” by three rats in Experiment 1 before and after bilateral auditory cortex lesions. Discrimination scores are reported in quiet and in four levels of white noise across the last two days of pre-lesion, first two-days post-lesion, and days seven and eight post-lesion behavior. One rat used slightly different noise levels (no noise, 42 dB, 54 dB, and 66 dB) during the first session of retesting; therefore, only her performance in silence was included in the analysis of this session. The dotted line represents chance performance at 50%. Asterisks represent significant differences between pre-lesion and post-lesion scores, $P < 0.05$.

3. Results

3.1. Histology

Lesions bilaterally destroyed A1 and most of the surrounding auditory cortex in every rat (Figure 2). Five rats had their brains stained with Cresyl Violet for comparison. Methodological errors during sectioning prevented staining the sections from the remaining four rats. Visual inspection of the remaining four rats’ brains revealed a similar pattern of destruction. The lesions typically extended bilaterally from roughly 1.6 ± 0.2 mm to 8.2 ± 0.2 mm caudal from bregma (mean \pm standard error of the mean). The dorsal and ventral extent of the lesions was variable amongst the rats and occasionally included parts of the somatosensory cortex, parietal association areas, visual cortices, or temporal association cortex. Damage to the temporal association cortex may contribute to the observed behavioral deficits in complex sound processing, and requires a cautionary note when interpreting the results from this study [19,20].

3.2. Experiment 1

The purpose of Experiment 1 was to evaluate the possibility that auditory cortex lesions would impair consonant discrimination when tested using operant procedures. Rats were rewarded for pressing a lever in response to the word “dad” and not the word “tad”. Testing occurred in quiet and in four levels of background noise. The background noise was added to increase the difficulty of the task to ensure that a negative result was not due to a ceiling effect. Rats were able to discriminate “dad” from “tad” in noise up to 72 dB SPL as accurately or better as they could in quiet (Fig. 3; $t(11) \leq 0.28$, n.s.), but experienced a deficit in noise at 84 dB SPL (quiet = $88 \pm 1\%$, 84 dB SPL = $67 \pm 3\%$, $t(11) = 4.8$, $P < 0.001$). Performance was still above chance when the background noise was 24 dB louder than the speech sounds (chance = 50%; $t(11) = 5.2$, $P < 0.001$). The ability of rats to discriminate these sounds in high levels of noise is consistent with human performance [21–23].

Bilateral auditory cortex lesions failed to generate a long-term impairment in speech sound discrimination. Performance was well above chance on the first day of post-lesion testing, even

when the background noise was 84 dB SPL (performance in 84 dB, SPL = $59 \pm 2\%$, chance = 50%; $t(4) = 17.8$, $P < 0.001$). A small, but statistically significant impairment was observed over the first two days in all but the loudest noise level (In 84 dB SPL, $t(5) = 1.7$, n.s.; In all other levels, $t(5) \geq 2.4$, $P < 0.05$). By the eighth day of post-lesion testing, performance had recovered to pre-lesion levels ($t(5) = 1.19$, n.s.). The observation that discrimination performance recovered completely at every noise level argues against a ceiling effect that would obscure the impact of the lesion. These results support the conclusion of the startle reflex modification study that rats with auditory cortex lesions can accurately discriminate amongst consonant sounds [1].

3.3. Experiment 2

Accurate speech sound discrimination following auditory cortex lesions suggests that the auditory cortex is not necessary for speech discriminations; however, it is possible that lesioned rats quickly adopt a new strategy that relies on the identification of cues in the auditory signal that are not affected by lesions of auditory cortex. Animals with bilateral auditory cortex lesions can discriminate tone frequencies [4,7,19,20,24–29], sound intensity [8–10], sound duration [11], slow amplitude modulations [30], and click trains [31,32] as accurately as unlesioned animals. Since the “dad” and “tad” stimuli used in Experiment 1 varied in cues other than voicing, including their first and second formant frequencies (846 and 2286 Hz vs. 957 and 1984 Hz, respectively), as well as the overall stimulus duration (414 ms vs. 465 ms), it is possible that the lesioned rats used these unrelated cues to perform the task. In Experiment 2, we tested rats on consonant discrimination tasks with speech sounds truncated to 40 ms to eliminate duration differences and make it more difficult to use differences occurring during the vowel component of the sounds.

The sounds were truncated at 40 ms because (1) this interval is known to be sufficient for humans to accurately discriminate consonant sounds [14,33] and (2) rat consonant discrimination using full-length speech sounds is best correlated with neural activity when only the first 40 ms of the A1 response is used [2]. These results suggest that the auditory cortex activity within the 40 ms of speech onset is used to discriminate between consonant sounds. No prior study has reported speech discrimination by animals using such short stimuli. Animals can accurately discriminate consonant [34–36] and vowel sounds [19,24,37], and exhibit similar phonetic boundaries to humans in fricative durations [36] and voice onset time [35]. We predicted that, like humans, un-lesioned rats would also be able to discriminate speech onsets.

Rats were trained to discriminate the first 40 ms of the target speech sound, “dad”, from similarly truncated versions of distracting speech sounds (“tad”, “bad”, “sad”, “deed”, “dood”, and “dud”). Since discrimination was accurate at every noise level after lesioning in Experiment 1, noise was not used to increase task difficulty in Experiment 2. Instead, six stimuli varying from the target stimulus in either the consonant (“tad”, “bad”, “sad”) or vowel (“deed”, “dood”, and “dud”) were used to provide a range of easy and difficult discriminations. A group of rats tested on full-length versions of the stimuli served as a comparison group to determine if rats could discriminate truncated speech as accurately as full-length speech. Both groups performed the task correctly on $75 \pm 1\%$ (onset trained) and $76 \pm 2\%$ (full-length trained) of the trials over the last two days of testing. These levels of performance are well above chance (50%; $t(23) \geq 16.6$, $P < 0.001$), indicating that both groups accurately discriminated the target sound from the distracting speech sounds. The asymptotic level of performance of the full-length trained group was not significantly different from the performance of the onset trained group ($t(46) = 1.7$, n.s.). This comparable performance between the groups supports predictions

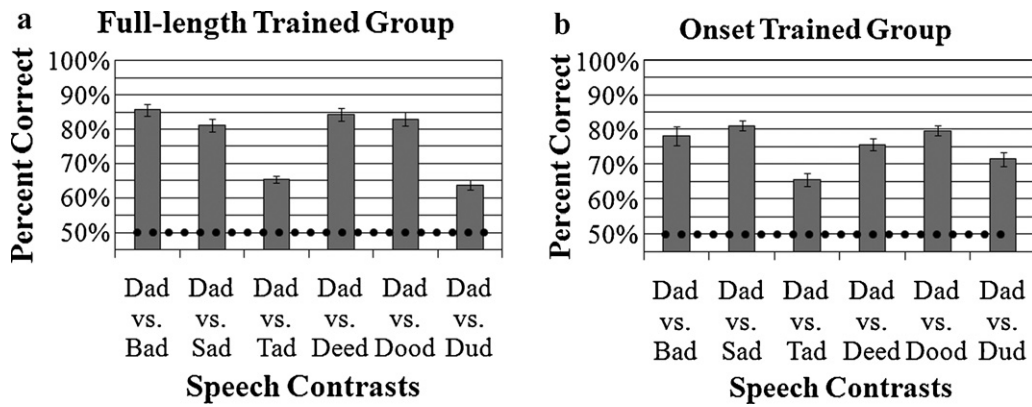


Fig. 4. Comparison of the percent correct discriminations of “dad” with individual distracting stimuli over the last 2 days of testing in Experiment 2. (a) Animals in the full-length trained group, $n = 12$. (b) Animals in the onset-trained group, $n = 12$. The dotted lines represent chance performance at 50%. Error bars show the standard error of the mean. Both groups performed significantly above chance on every task ($t(23) \geq 9.1, P < 0.001$).

made from neurophysiology [2] and human psychophysics [14,33] that rats can discriminate speech onsets as accurately as they can discriminate full-length speech.

Performance on individual contrasts over the last two days of testing were examined to confirm that rats could accurately discriminate between both consonant and vowel sounds using only the speech onsets (Fig. 4a and b). Both onset and full-length trained groups consistently rejected speech sounds that differed from the target sound in the initial consonant or the following vowel. All six speech contrasts tested were well above chance ($t(23) \geq 9.1, P < 0.001$). For both groups, the most difficult consonant to discriminate from the target sound, “dad”, was “tad” (onset trained group = $66 \pm 2\%$, full-length trained group = $65 \pm 1\%$ correct). The most difficult vowel to discriminate from the target sound, “dad”, was “dud” (onset trained group = $71 \pm 2\%$, full-length trained group = $64 \pm 1\%$). Thus, the overall pattern of consonant and vowel discrimination was similar whether the rats used the entire speech sound or only the initial 40 ms of the speech sound. These results indicate that the first 40 ms of the speech sounds tested contain sufficient information to explain the discrimination ability of rats trained on full-length speech sounds.

Rats in the onset trained group learned the task at a notably slower rate compared to the full-length trained group (Fig. 5). The full-length trained group learned the task to a level of 68% correct in 1.8 ± 0.3 days, while the onset trained group took 10.0 ± 1.8 days to reach the same criterion ($t(12) = 4.7, P < 0.001$). The 68% criterion

is the best single day performance of the rat with the worst performance of all the rats tested (i.e. allows all the rats in the study to reach the criteria). Full-length trained rats performed significantly better than the onset trained rats each day during the first ten days of testing ($t(22) \geq 2.6, P < 0.01$). The slow rate of learning in the onset trained group indicates the truncated stimuli were more difficult to discriminate. These results show that the additional acoustic cues included in the full-length stimuli facilitates the learning of the speech discrimination task, but are not required to perform the task accurately.

To test the importance of auditory cortex in speech discriminations using the minimal acoustic information, six rats from the onset trained group received bilateral auditory cortex lesions and continued to test on speech onset discriminations after one week of recovery (Fig. 6). Lesioned rats were able to reliably discriminate target speech sounds from silent catch trials, indicating that AC lesions did not deafen the rats or impair their ability to perform the operant task (performance = $59 \pm 3\%$, chance = 50%; $t(11) = 3.0, P < 0.01$). Additionally, the unimpaired discrimination performance by the lesioned rats in Experiment 1 argues against non-auditory impairments from collateral damage to other cortical areas from the lesioning process. Rats in Experiments 1 and 2 received the same lesioning procedures and demonstrated similar extents of cortical damage. Despite their ability to hear the stimuli, the lesioned rats in Experiment 2 were impaired to chance

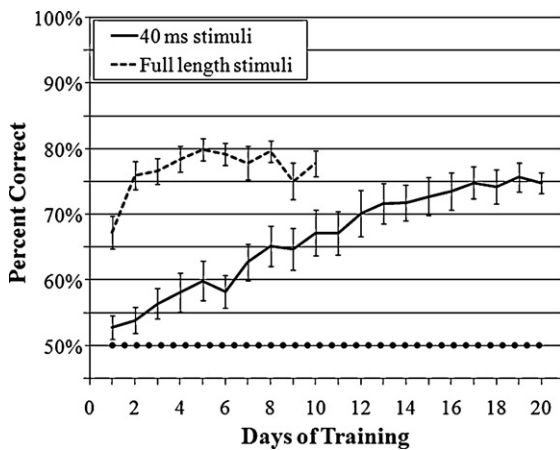


Fig. 5. Timeline of the percent correct discrimination of “dad” from the distracting stimuli for the onset trained group ($n = 12$) and the full-length stimulus trained group ($n = 12$) in Experiment 2. Error bars show the standard error of the mean. The dotted line represents chance performance at 50%.

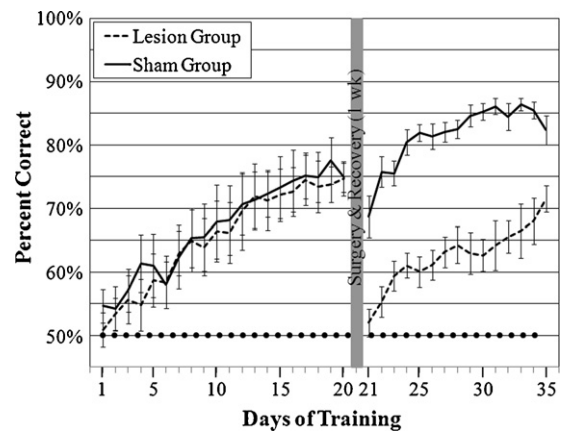


Fig. 6. Timeline of the percent correct discrimination of “dad” from the distracting stimuli for the lesion group ($n = 6$) and the sham group ($n = 6$) from initial testing on the task through retesting after recovery from surgeries in Experiment 2. The grey bar represents the time for the surgery and one week of recovery. Error bars show the standard error of the mean. The dotted line represents chance performance at 50%.

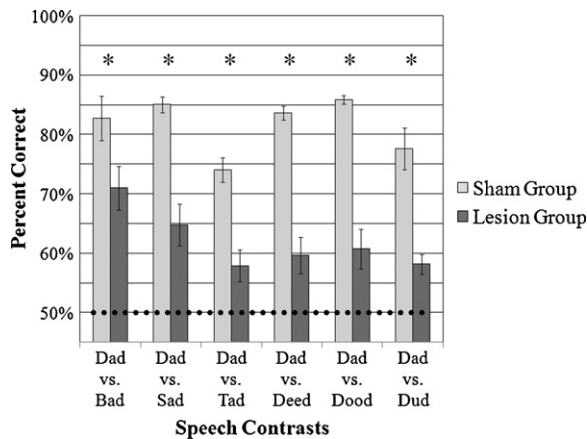


Fig. 7. Percent correct across all post-surgery days for sham and lesion groups on “dad” discrimination from individual distracting stimuli in Experiment 2. Error bars represent the standard error of the mean. $n=6$ in both groups. The dotted line represents chance performance at 50%. Asterisks represent significant differences between the lesion and the sham group, $P<0.05$. Lesion group is significantly worse than the sham group on every speech contrast ($t(10) \leq -2.47$, $P<0.05$).

levels of performance on the first day of continued testing (performance = $52 \pm 2\%$, chance = 50%; $t(5) = 0.92$, n.s.). The six remaining rats from the onset trained group underwent sham surgeries and continued testing with the onset stimuli to serve as an unlesioned comparison group. These sham rats experienced only a slight impairment and their performance was well above chance on the first day of post-surgery testing (performance = $69 \pm 3\%$, chance = 50%; $t(5) = 5.7$, $P<0.001$). Sham rats demonstrate that the weeklong interruption in training following surgery may have caused a slight lapse in performance, but that the damage to the auditory cortex is responsible for the majority of the observed impairment in the lesion group. The lesion group performed the task at above chance levels as testing progressed, but remained significantly worse than the sham group on every day ($t(10) \leq -3.51$, $P<0.01$). Auditory cortex lesions did not affect the whole word discrimination task in Experiment 1, but did impair speech onset discriminations in Experiment 2. These results are consistent with the hypothesis that rats use the first 40 ms of auditory cortex neural activity to discriminate between consonant sounds [2].

Performance of both the lesion and sham groups improved significantly from the 21st to the 35th day of testing ($t(5) \leq -3.3$, $P<0.01$). Performance by the lesion group was approximately 19% lower than the sham group on every day of post lesion testing. If the rats were recovering a previous ability, we would expect to see an increased rate of improvement in the performance compared to the sham rats. Since the difference between the groups’ scores remained consistent over the additional testing ($R^2 = 0.07$, n.s.), we conclude that the deficit is stable for at least a month and that the improvement in both groups is due to ongoing learning.

Six distracting stimuli created a range of easy and difficult discriminations from the target speech sound to test if discriminating the subtle differences in speech onsets was impaired following bilateral auditory cortex lesions (Fig. 7). Lesion rats were significantly worse than the sham group on the average discriminations of the target sound from each of the distracting sounds over all post-surgery testing (Fig. 6; Main effect of group, $F(1, 60) = 180.0$, $P<0.001$; $t(10) \leq -2.47$, $P<0.05$). Additionally, the degree of impairment from pre-lesion to post-lesion performance is similar for each of the contrasts tested (average pre-lesion minus post-lesion performance = -12% ; No main effect of task, $F(5, 30) = 0.89$, n.s.). With the exception of the “sad” contrast, the ranking of the highest to lowest scored contrast was the same between the lesion and the sham group (Fig. 7; Interaction of group and

individual contrast, $F(5, 60) = 2.0$, n.s.). This consistent pattern of impairment after bilateral auditory lesions suggests that the average impairment is not due to any one contrast, but is a general deficit across the consonant and vowel discriminations tested.

4. Discussion

Operant speech discrimination ability was preserved after bilateral auditory cortex lesions unless the speech stimuli were limited to the first 40 ms. This result is in agreement with an earlier study finding spared discrimination of speech stimuli with an average duration of 150 ms (110–260 ms) tested using an acoustic reflex modification paradigm [1]. The longer stimuli contain several potential auditory cues, including stimulus duration, which could allow discrimination by rats with auditory cortex lesions. The sounds from Experiment 1 were shortened by 90% to reduce or eliminate these cues. Normal rats still accurately discriminated the truncated speech sounds. Auditory cortex lesions impaired the rats’ discrimination ability of the truncated speech sounds. These results suggest that the auditory cortex plays an important role in distinguishing the rapidly occurring acoustic transitions at the beginning of speech sounds.

Similarities in the final performance of the onset and the full-length trained rats confirm earlier predictions that the entire speech sound is not necessary for accurate speech discrimination amongst consonants and vowels. Although human psychophysics supported this finding [14,33], onset discrimination has not been previously reported in animals. Human performance was more accurate than rat performance, which might be expected since humans have thousands of hours experience with speech sounds in their native language. By comparison, the onset trained rats in our study heard the speech for less than 80 h total. Humans exhibit significantly worse discrimination of contrasts not used in their native language [38–41]. These psychophysical results suggest that humans and rats discriminate consonant and vowel sounds in a similar manner.

Our earlier study found that the differences in the first 40 ms of A1 neural activity better predict whole word discrimination in rats than the A1 responses to the whole sound [2]. Predictions that these brief, spatiotemporal activity patterns in A1 are critical for onset discriminations were confirmed in Experiment 2 by the loss of discrimination ability following auditory cortex lesions. A recent fMRI study of native and non-native English speakers reported that (as in rats) the distinctiveness of the A1 activity patterns evoked by consonants was correlated with discrimination ability [2,42]. Collectively, the results from previous studies and the current study suggest that humans and animals likely share similar neural processing mechanisms for encoding speech sounds up to the level of A1. Animal models have proven useful in elucidating the biological cause of some forms of communication disorders. For example, cortical microgyric lesions in the parietal cortex affect the temporal processing of speech sounds in rats in a manner that closely resembles the deficits seen in children with specific language impairment [43–46]. These results have contributed to the development of treatment models for specific language impairment in humans. Additionally, a genetic model in rats has contributed to explaining some of the underlying causes of dyslexia [47–49]. Understanding the contributions of the auditory system in discriminating speech sounds in rodent models may lead to the refinement of techniques for treating different sources of language impairments in humans.

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