Intracellularly Labeled Fusiform Cells in Dorsal Cochlear Nucleus of the Gerbil. I. Physiological Response Properties

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Hancock, Kenneth E. and Herbert F. Voigt. Intracellularly labeled fusiform cells in dorsal cochlear nucleus of the gerbil. I. Physiological response properties. *J Neurophysiol* 87: 2505–2519, 2002; 10.1152/jn.00342.2001. Fusiform cells in the dorsal cochlear nucleus (DCN) of barbiturate-anesthetized Mongolian gerbils were characterized physiologically and labeled with neurobiotin. This report is based on 17 fusiform cells for which there was reasonable confidence in the association between physiological data and recovered anatomy. The qualitative morphology of these cells was no different from that reported in previous studies. The acoustic response properties were generally consistent with those described in the barbiturate-anesthetized cat. Most responses were of the pauser or buildup type, but a dependence on stimulus frequency and intensity was observed. Stimulus-evoked sustained depolarizations and large, long-lasting afterhyperpolarizations were common membrane potential features. The cells in this study showed a greater tendency to discharge regularly than did those of the cat, likely as a result of the longer interstimulus interval used. Barbiturate anesthesia appears to mask an interspecies difference in DCN physiology that is apparent in unanesthetized, decerebrate preparations. The response of these fusiform cells to a depolarizing current pulse could be altered by the presence of a hyperpolarizing prepulse. Buildup, pauser, and chopper patterns could each be created using appropriate combinations of hyperpolarizing and depolarizing pulse amplitudes. Thus the adult gerbil appears to express the inactivating potassium conductance previously shown to affect fusiform cell firing patterns in vitro. The results further demonstrate that the effects of these potassium currents are readily observed in vivo. Finally, the fusiform cells in this study were quite variable with respect to a number of response properties, including the resting potential, input resistance, spontaneous activity, relative noise index, normalized tone slope, and regularity histogram shape. This diversity likely results from cell-to-cell variations in the balance of activity within the relatively complex network to which the fusiform cells belong, although effects of impalement may contribute to the extent of the diversity.

**INTRODUCTION**

The initial stage of auditory signal processing includes the dorsal cochlear nucleus (DCN), which contains many different cell types organized into a layered structure. There are fundamentally three layers, the superficial layer, the fusiform cell layer, and the deep layer, although in some species, the latter can be further subdivided (Brawer et al. 1974; Lorente de Nó 1981). The descending branch of the auditory nerve (AN) terminates in the deep layer and endows the DCN with a tonotopic organization (Rose et al. 1959; Ryugo and May 1993). The deep layer contains a variety of multipolar cells, including giant cells that project to the contralateral inferior colliculus (Adams and Warr 1976) and contralateral cochlear nucleus (Cant and Gaston 1982). Vertical cells are also located in the deep layer, and are an important source of inhibition within the DCN as well as both subdivisions of the ventral cochlear nucleus (VCN) (Rhode 1999; Saint-Marie et al. 1991; Voigt and Young 1980, 1990; Zhang and Oertel 1993a).

The superficial layer lies just beneath the ependymal surface of the DCN and contains a mixture of cells including cartwheel, stellate, and Golgi cells (Mugnaini et al. 1980a; Wouterlood and Mugnaini 1984; Wouterlood et al. 1984). The somata of granule cells are distributed throughout the DCN, and their axons form a network of parallel fibers, arranged orthogonally to the tonotopic axis, that serves as the principal input to the superficial layer (Mugnaini et al. 1980b). The granule cell domains receive input from a variety of sources, including the somatosensory system (Itoh et al. 1987; Weinberg and Rustioni 1987; Wright and Ryugo 1996), the vestibular system (Burian and Gstoettner 1988; Keveker and Perachio 1989), and the descending auditory system (Benson and Brown 1990; Weedman and Ryugo 1996).

Sandwiched between the superficial and deep layers is the fusiform cell layer, comprising an irregular arrangement of the fusiform cells for which it is named. These neurons have relatively large somata and two dendritic arbors, an apical arbor that extends into the superficial layer and a basal arbor that descends into the deep layer (Brawer et al. 1974; Lorente de Nó 1981). The dendritic arbors are flattened in a plane parallel to the DCN isofrequency laminae (Blackstad et al. 1984). Their axons project out of the DCN to the contralateral inferior colliculus by way of the dorsal acoustic stria (DAS) (Adams and Warr 1976).

The apical arbor is densely branched and covered with spines that are the site of excitatory input from parallel fiber axons (Mugnaini et al. 1980a). The basal dendrites are sparser and free of spines. The distal portions of the basal dendrites receive excitatory synapses from AN fibers (Smith and Rhode 1985). Inhibitory inputs to fusiform cells are located primarily on the soma and proximal dendrites and originate from vertical...
Fusiform cells of particular interest because they represent the majority of the output fibers of the DCN (Adams 1976) and because their bipolar dendritic structure allows them to integrate activity from both of the other DCN layers. It has been suggested, for example, that they are sensitive to the spectral filtering properties of the pinna and that the circuitry of the superficial layer may serve to account for pinna movement (Parsons et al. 2001; Rice et al. 1992; Young et al. 1992, 1995).

Fusiform cells comprise a heterogeneous population in terms of physiological behavior. Response properties in the unanesthetized, decerebrate preparation have been traditionally classified using the response map scheme of Evans and Nelson (1973), as modified and extended by later investigators (Davis et al. 1996; Shofner and Young 1985; Spirou and Young 1991; Young and Voigt 1982). Extracellular recordings in conjunction with antidromic stimulation of the DAS have associated fusiform cells with type III and type IV units in the decerebrate cat (Young 1980). Fusiform cells labeled as part of intracellular recording experiments in the decerebrate gerbil displayed type I/III, type III or type IV-T unit response properties, but never the responses of a classical type IV unit (Ding et al. 1999).

In barbiturate-anesthetized animals, it has been more common to use poststimulus time histograms (PSTHs) for classifying DCN response types (Pfeiffer 1966). Extracellular recordings from the fusiform cell layer were predominantly pauser or buildup units in the anesthetized cat, suggesting that these may be the responses of fusiform cells (Godfrey et al. 1975). This conclusion was confirmed using intracellular recording and labeling techniques (Rhode et al. 1983). Furthermore, Rhode et al. demonstrated that a single fusiform cell can show pauser, chopper, or onset patterns, depending on the frequency and level of the stimulus and on other experimental conditions. Similarly, fusiform cells in vitro respond to depolarizing current pulses with buildup, pauser, or chopper patterns depending on the level of a hyperpolarizing prepulse (Kanold and Manis 1999; Manis 1990).

This report describes a study in which intracellular recording and labeling techniques were used to investigate the response properties of fusiform cells in barbiturate-anesthetized Mongolian gerbils. One aim was to compare the results of this study to the physiology and anatomy of fusiform cells previously reported in the barbiturate-anesthetized cat (Rhode et al. 1983; Rhode and Smith 1986; Smith and Rhode 1985). Such comparison is of particular importance since there are significant differences between decerebrate gerbils and decerebrate cats (Davis et al. 1996). In particular, whereas type IV units comprise ±32% of the units encountered in the decerebrate cat (Shofner and Young 1985), they represent only 11% of the units in decerebrate gerbil (Davis et al. 1996). Furthermore, fusiform cells in the gerbil may not have type IV unit properties at all (Ding et al. 1999), while some subset of those in cat almost certainly do (Young 1980). The results qualitatively confirm the earlier studies showing that fusiform cells exhibit a variety of response properties, including pauser, buildup, and chopper, depending on acoustic stimulus and current-clamp parameters (Manis 1990; Rhode et al. 1983).

This work represents part of the doctoral dissertation of K. E. Hancock.

METHODS

Surgery

The experimental protocols described below were approved by the Boston University Institutional Animal Care and Use Committee. Female Mongolian gerbils (Meriones unguiculatus), 3–6 mo in age, were anesthetized with intraperitoneal injections of pentobarbital sodium (Nembutal, 65 mg/kg). Supplemental anesthetic doses (13 mg/kg) were administered approximately every half hour to maintain areflexia to paw pinches. A thermostatically controlled heating blanket (Harvard Apparatus) maintained the core body temperature at 38°C. Following a tracheotomy, small subcutaneous periauricular injections of lidocaine (Xylocaine) were given and the pinnae removed. The animal was mounted on a stereotaxic apparatus (Kopf 1730) and the head immobilized with earbars and a mouth bar.

Access to the brain stem was attained using a transbulla approach (Frisina et al. 1982). Blunt dissection was used to reflect the tissue overlying the bulla dorsal and posterior to the ear canal, and the soft bone was gently removed using no. 5 forceps. The temporal bone inside the ring formed by the posterior semicircular canal was repeatedly perforated with a sharp probe until it broke free. The bone was removed with forceps, exposing the lateral surface of the cerebellum. The meningeal covering of the cerebellum was carefully peeled away, and mineral oil was applied liberally to the surface to keep it moist.

Electrodes

Sharp micropipette electrodes were pulled from 1 mm OD borosilicate glass tubing with filaments (WPI 1B100F-4) using a Flaming-Brown horizontal puller (Sutter P80-PC). The electrodes were filled with a 1–2% solution of neurobiotin (Vector Labs) in 2 M KAc. The impedance at 1 kHz was measured (Winston Electronics BL-1000) and reduced from a typical initial value of 150 MΩ to 60–80 MΩ by beveling the electrodes in a rotating slurry of silicon carbide powder in physiological saline.

The lead wire and reference electrode were both Ag/AgCl. The reference was coiled and placed in the musculature near the shoulder. The electrode was advanced into the brain stem in a coronal plane at an angle of 21° above the horizontal using a microproposition driven by a stepper motor (Kopf Model 660). Current pulses 10 ms in duration and ~1 nA in amplitude were used to monitor electrode impedance and to verify optimal capacitance compensation.

Recording system

Stimulus generation (see following text) and data acquisition were controlled by a personal computer (Gateway 2000, 486/33) interfaced to System II hardware from Tucker-Davis Technologies (TDT). The electrode signal was amplified (Axon Instruments, Axoclamp 2A) and sent through two parallel data acquisition channels, one for intracellular measurements and one for extracellular measurements. Signals were filtered and amplified (intracellular channel: DC–10 kHz, typically ×50, Tektronix AM-502; extracellular channel: 0.3–10 kHz, typically ×2000, Tektronix AM-502). Signals in the intracellular channel were digitized at 20 kHz (TDT AD2) and stored on hard disk. Action potentials in the extracellular signal were recorded using a Schmitt trigger to signal an event timer (TDT ET1).

Acoustic system

The stereotaxic apparatus was housed in a sound-attenuating chamber (IAC 1202A). A headphone speaker (BeyerDynamics DT48A) delivered sound to the left ear canal through an annulus in the earbar.
Tonal search stimuli were generated from a manual oscillator (Wavetek 188), while all other stimuli were digitally created on a PC-controlled array processor (TDT AP1) and realized with a signal processing D/A converter (TDT PD1) using a sampling rate of 50 kHz. Stimuli were routed through a graphic equalizer (BSR EQ-3000) to help flatten the spectral characteristics of the acoustic system and attenuated (TDT PA4) to achieve desired sound pressure levels. A custom-built current amplifier was used to drive the headphone speaker.

The acoustic system was calibrated at the beginning of each experiment. A ½-in. microphone (BK 4134), couple to the earbar through a small probe tube, was used to record acoustic signals near the tympanic membrane. The acoustic system impulse response was obtained by averaging 100 digitized responses (2,048 samples at 50 kHz) to 20-μs, 4-V clicks. The system frequency response was computed by taking the fast Fourier transform (FFT) of the impulse response and compensating for the probe tube contribution. Desired sound pressure levels (SPLs) for tones were achieved by attenuating 1-Vrms sinusoids according to the acoustic system frequency response.

For presentation of broadband stimuli, an inverse filter was designed to compensate for the nonflat spectral characteristics of the acoustic system. The inverse filter was created in the frequency domain by inverting the magnitude spectrum of the acoustic system and assigning a linear phase characteristic. The inverse FFT of this signal was computed and truncated with a Hanning window to arrive at a finite impulse response (FIR) representation of the inverse filter. The FIR coefficients were loaded into a digital signal processing (DSP) module on the D/A converter, providing real-time precompensation of broadband stimuli. Note that this precompensation was valid only for random phase signals, such as noise, because the acoustic system phase characteristic was ignored when creating the inverse filter.

The maximum broadband noise SPL, attainable by the system was determined by frequency averaging 100 responses (2,048 points at 50 kHz) to a 1-Vrms noise signal and computing the power of the resulting average. Desired noise SPLs were achieved by generating 1-Vrms noise and attenuating appropriately. The SPLs of click stimuli were determined by frequency averaging 100 responses (2,048 points at 50 kHz) to 20-μs, 4-V clicks. The system frequency response was computed by taking the fast Fourier transform (FFT) of the impulse response and compensating for the probe tube contribution. Desired sound pressure levels (SPLs) for tones were achieved by attenuating 1-Vrms sinusoids according to the acoustic system frequency response.

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**Brainstem auditory-evoked response**

After calibrating, the brainstem auditory-evoked response was measured to assure the integrity of the auditory system. Platinum needle electrodes were inserted subdermally near the nose, the left shoulder, and the middle of the back. Click responses were amplified (×100) by a series combination of two amplifiers (Grass P15d, Tektronix AM-502) and then digitized (600 samples at 20 kHz; TDT AD2). Averages of 350 presentations were obtained at each of several SPLs to determine a threshold. The experiment continued if the threshold was within the normal range for gerbils (Burkard and Voigt 1989).

**Data collection**

Broadband noise was used as a search stimulus while the electrode was advanced through the cerebellum into the cochlear nucleus. When background driving was detected, the best frequency (BF) of the activity was determined approximately. BF tones were subsequently used as search stimuli.

Intracellular penetrations were marked by sudden negative shifts in the DC potential recorded by the electrode. A penetration was considered acceptable when the resting potential was less than −50 mV, the action potential amplitude greater than 40 mV, and the neuron discharged in response to small depolarizing currents. Exceptions to these criteria were occasionally made based on a subjective assessment of the impedance quality. Once an acceptable penetration was made, the BF of the neuron was estimated audiovisually. Data were then collected according to the following protocol.

1) The membrane potential was digitized in response to tones at three frequencies (BF, BF ± 0.7 octaves) systematically varied in intensity (0–80 dB SPL in 5-dB steps). Each stimulus condition was presented three times. Three frequencies were used to provide a record of behavior in the sidebands as well as at BF. Stimuli were 100 ms in duration with 5-ms rise/fall times and a 500-ms interstimulus interval. Responses to broadband noise were then collected varying intensity in the same manner and using the same timing paradigm.

2) Current-clamp responses were obtained using 50-ms current pulses presented once every 250 ms. The pulse amplitude was varied between −1 and +1 nA in steps of 0.125 nA. The paradigm was repeated to assure response consistency.

3) The effect of prehyperpolarization on discharge pattern was determined using a paradigm similar to that of Manis (1990). Depolarizing current pulses 100 ms in duration were alternated with 100-ms hyperpolarizing pulses, stepped in amplitude from −1 to 0 nA in 0.2-nA increments. For each hyperpolarizing current level, the depolarizing current pulse amplitude was varied from 0 to +1 nA in 0.2-nA increments, with responses to three pulses obtained at each increment. The procedure was performed twice to assure consistency.

4) A PSTH classification was made on the basis of event times recorded for 100 repetitions of BF tone bursts 20 dB above threshold. The tone bursts were 200 ms in duration with 5-ms rise/fall times and a 1,000-ms interstimulus interval.

5) Neurobiotin was iontophoresed using +4-nA, 150-ms current pulses delivered at a rate of 4 Hz for 2 min. The membrane potential was monitored and recorded during the current-off time. Iontophoresis continued as long as the potential remained within 20 mV of its initial value. Stability of the membrane potential provided evidence that the electrode stayed inside the neuron during injection. Such evidence is justification for associating the recorded physiology with subsequently recovered morphology. If the neuron continued to fire action potentials after neurobiotin injection, at least part of the preceding protocol was repeated. Otherwise, the electrode was withdrawn or another injection was initiated.

**Data analysis**

**Rate-level curves.** Rate-versus-level curves were constructed by averaging the firing rate over the last 80% of the stimulus-on period.

**TABLE 1.** Basis for associating recovered neurons with intracellular recordings

<table>
<thead>
<tr>
<th>Cell</th>
<th>Deposits Recovered</th>
<th>Clamped</th>
<th>Basis for Identification</th>
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<tbody>
<tr>
<td>95026</td>
<td>1 1 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>95055</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>95082</td>
<td>1 3 3</td>
<td></td>
<td>Intensity</td>
</tr>
<tr>
<td>95135</td>
<td>1 2 1</td>
<td></td>
<td>See text</td>
</tr>
<tr>
<td>97002</td>
<td>1 1 1</td>
<td></td>
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</tr>
<tr>
<td>97005a</td>
<td>1 3 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>97008b</td>
<td>2 2 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9702a</td>
<td>1 3 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>97054a</td>
<td>1 4 2</td>
<td></td>
<td>RC sep, depth</td>
</tr>
<tr>
<td>97054b</td>
<td>1 4 2</td>
<td></td>
<td>Intensity</td>
</tr>
<tr>
<td>97055a</td>
<td>2 3 2</td>
<td></td>
<td>Depth</td>
</tr>
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<td>97072a</td>
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<td></td>
<td></td>
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<td>1 1 2</td>
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</tr>
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</tr>
<tr>
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</tr>
<tr>
<td>98024a</td>
<td>2 3 3</td>
<td></td>
<td>Depth</td>
</tr>
</tbody>
</table>

D, number of deposits made; R, number of deposits recovered; RC sep, rostral-caudal separation.
Spontaneous rates were averaged over a period of equal length at the end of the stimulus-off period. For each neuron, the average spontaneous activity over the entire BF rate-versus-level series was used as a representative spontaneous rate in tabulating the data. The normalized tone slope was computed from the BF data by measuring the slope of the curve from its maximum to the following minimum and dividing by the maximum firing rate (Young and Voigt 1982). The relative noise index, $p$, divides the maximum broadband noise driven rate by the maximum BF tone driven rate, where the driven rate in each case is the difference between the maximum total firing rate and the spontaneous rate (Young and Voigt 1982).

**POSTSTIMULUS TIME HISTOGRAMS.** The PSTH and regularity histogram data were plotted using 1-ms bins and classified using the decision tree of Gdowski (1995) as a guide. Briefly, the four major PSTH classes, namely pausers, buildups, choppers, and onsets (Pfeiffer 1966), were subdivided based on the shape of the regularity histogram, which plots the mean and SD of the interspike interval (ISI) distribution as a function of time (Bourk 1976). Important subcategories include chopper-transient-plus (CT+) and chopper-transient-minus (CT−), for which the ISI transiently increases and decreases, respectively, and pauchoppers (PaC), which exhibit brief pauses followed by a regular sustained discharge pattern.

**Histological processing**

After a survival time of at least 2 h, the animal was deeply anesthetized and perfused transcardially with a mixture of buffered aldehydes (3% glutaraldehyde, 0.5% paraformaldehyde in 0.1 M phosphate buffer). The brain was removed and immersed overnight in fixative at 4°C. The tissue was shelled in gelatin-albumin and cut into 50-μm coronal sections (Vibratome Series 1000). The sections were washed thoroughly then agitated overnight in

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**FIG. 1.** Fusiform cell 97073. A: reconstruction of neuron in a coronal viewplane. Arrowhead points to the initial axon segment. Coronal (B) and sagittal (C) renderings show position with respect to the layers of the dorsal cochlear nucleus (DCN). In each panel, the DCN is drawn in the plane containing the soma. Gray stipple, fusiform cell layer; D, dorsal; M, medial; R, rostral. Scalebar = 100 μm. D: discharge rate vs. sound pressure level in response to broadband noise, best frequency (BF) tones, and sideband tones at BF ± 0.7 oct. Rates plotted are the average of 3 trials at each level. Sound bursts were 100 ms in duration, and the stimulus interval was 500 ms. E: poststimulus time histogram (PSTH) derived from 100 responses to BF tone bursts. Stimulus duration and interval were 200 and 1,000 ms, respectively. F: regularity histogram computed from the same data. Bar indicates the stimulus-on period. ●, mean interspike interval; ○, SD; CV, coefficient of variation. G–J: selected membrane potential records from the data represented in D. Resting potentials are indicated at the bottom left of each panel. Stimulus bar is shown at the bottom of J.
Vector ABC reagent. The reaction product was visualized using standard diaminobenzidine histochemistry intensified with nickel ammonium sulfate and cobalt chloride (Adams 1981). The sections were mounted and air dried before they were counterstained with cresyl violet and coverslipped with permount.

Anatomical reconstructions

Neurons were reconstructed working from camera lucida drawings. Separate drawings were made of each section (×100, oil immersion), and the depths at various points were noted from the fine focus knob of the microscope. The drawings were then scanned into the computer and custom-designed software used to extract neural structures and depths. Points along each dendritic branch were specified by the user who then entered the corresponding depths. The software automatically measured the diameter at each point and stored the resulting data as a set of \([x, y, z, d]\) quadruples. The software was then used to align and connect the reconstructions of adjacent sections. Perspective drawings were created by storing the data in a form compatible with the ray-tracing package POV-Ray.

Reconstructions of the DCN were made in a similar manner. For each tissue section containing a portion of the DCN, outlines of the DCN and fusiform cell layer were drawn on paper using the camera lucida tube. A series of MATLAB scripts was used to extract and align the scanned drawings and to superimpose neural reconstructions on the appropriate section.

RESULTS

In the course of this study, intracellular recordings were made from 78 neurons of which 53 were labeled with neurobiotin. Of these recovered neurons, 17 were identified as fusiform cells based on the bipolar arrangement of the dendrites and, in most cases, on the trajectory of the axon.

Confidence in the association between physiology and anatomy

In all experiments, the stability of the resting membrane potential was monitored during iontophoresis of neurobiotin. Injections were halted if the resting membrane potential became greater than 20 mV above the starting value. In the cases for which the impalement was stable after iontophoresis, some portion of the data collection protocol was repeated. Despite precautions, there were ambiguities in some cases.

FIG. 2. Fusiform cell 97022a. Same organization as Fig. 1.
Table 1 details the types of ambiguities encountered and the basis for identification for each of the 17 fusiform cells in this study. The left side of Table 1 lists the number of neurobiotin deposits attempted and the number of neurons recovered for each experiment. Six cases were unambiguous in that only one injection of neurobiotin was attempted and only one neuron was recovered.

For the cases in which multiple deposits were made, recovered neurons were identified on the basis of the separation of electrode tracks in the rostral-caudal dimension. When this was not clear, identifications were made by comparing the relative depths of the labeled neurons to the micropositioner readings recorded during the experiment.

A second class of ambiguity occurred when more neurons were recovered than deposits were made. A hierarchy of criteria was used to make identifications in these cases. 1) Faintly or incompletely labeled neurons were assumed to result from neurobiotin leakage during brief intracellular contacts and were eliminated from consideration. 2) Small current pulses used to study responses were often sufficient to label neurons. These were identified on the basis of position as described above. 3) Recovered cartwheel cells could usually be matched up with experiment notes indicating brief impalments of complex-spiking neurons. 4) In one case (97005), two fusiform cells were recovered in different portions of the tonotopic axis and an identification was made on the basis of best frequency. In this instance, the association of physiology with a specific neuron is perhaps a bit tenuous, but the classification of fusiform cell is certain. Finally, responses of two fusiform cells were not identifiable and were rejected from consideration in this study.

Anatomical properties

These fusiform cells are anatomically similar to those described by earlier studies in a variety of species, as illustrated in Figs. 1–6. Spinous, thickly branched apical dendrites extend into the molecular layer, typically reaching all the way to the ependyma. The basal dendrites are less densely branched and spine-free and descend into the deep layer of the DCN. Pianarity of the dendritic fields is particularly conspicuous in Figs. 2 and 6. Axons were identified in 15 cases, arising from either the soma (Figs. 1, 3, and 6) or a proximal basal dendrite (Figs. 2 and 5) and joining the DAS to leave the nucleus at its dorsomedial border. The labeling of the axon was typically intermittent, consistent with the presence of a myelin sheath, which hinders the access of histochemical reagents (Zhang and Oertel 1993b). No axon collaterals were ever observed, a consistent observation in rodents (Ding et al. 1999; Manis 1990; Zhang and Oertel 1994) that stands in contrast to observations in the cat (Lorente de Nó 1981; Rhode et al. 1983; Smith and Rhode 1985).

Physiological properties

The physiological characteristics of the 17 fusiform cells are summarized in Table 2. The responses are quantified using a variety of common measures, including spontaneous firing rate (SR), PSTH shape, normalized slope of the BF rate-level curve, relative noise response, input resistance, and resting potential (RP). By any of these metrics, the fusiform cell population demonstrates a wide range of physiological properties. Specific examples are illustrated in Figs. 1–6.

The responses of cell 97073 (RP = −66 mV, SR = 0.2 spikes/s, R_in = 8 MΩ) were weak and of high threshold (Fig. 1D). In response to sideband tones, it did not fire action potentials, although at high levels did respond with subthreshold depolarizations of approximately 5 mV in amplitude (Fig. 1, G and I). The cell discharged in a buildup pattern (Fig. 1E). The PSTH data were obtained shortly before the impalement was lost at a point when the threshold had decreased. Thus the PSTH in Fig. 1E exhibits a larger rate and shorter latency than the membrane potential record of Fig. 1H obtained earlier in the impalement.

Cell 97022a (RP = −62 mV, SR = 0.2 spikes/s, R_in = 29 MΩ) was a pauser unit with regular interspike intervals (Fig. 2, E and F). The rate-level curves for BF tones, below-BF tones, and

<table>
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<tr>
<th>Cell</th>
<th>BF, kHz</th>
<th>Spont, sp/s</th>
<th>PSTH</th>
<th>m₀, μs/ms</th>
<th>m₁, μs/ms</th>
<th>m₂, μs/ms</th>
<th>CV</th>
<th>T_slope, 10⁵ Db</th>
<th>RNI</th>
<th>R_in, MΩ</th>
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BF: best frequency; Spont: spontaneous rate; PSTH: poststimulus time histogram; BU: buildup; PaC: pauchopper; Pa: pauser; CS: chopper sustained; m₀, m₁, m₂: regularity histogram slopes (see text); CV: coefficient of variation; T_slope: normalized tone slope; RNI: relative noise index; R_in: input resistance; RP: resting potential; AP: action potential amplitude.
and broadband noise were highly nonmonotonic (Fig. 2D). The action potentials had deep undershoots and were accompanied by large sustained depolarizations, approximately 10 mV in size. Depolarizations during the stimulus were present even as the number of spikes decreased at high levels (Fig. 2J). The presence of sideband inhibition is suggested by hyperpolarization of the membrane during the stimulus in response to high-frequency tones (Fig. 2G).

Cell 97055a (RP = −76 mV, SR = 15.2 spikes/s, R\text{in} = 32 M\Omega) was classified as a pauchopper, which is characterized by a long first interval followed by regular spiking in the steady state (Fig. 3, E and F). The firing rate was a monotonic function of level when the cell was driven with BF tones, below-BF tones, or broadband noise (Fig. 3D). The broadband noise series was obtained shortly after impalement before the membrane potential stabilized, and thus the noise responses have a larger spontaneous rate (Fig. 3D) and a larger resting potential (Fig. 3J) than the remainder of the data. Stimulation with above-BF tones evoked inhibition of the spike rate (Fig. 3D) and hyperpolarization of the membrane (Fig. 3G).

Cell 98001 (RP = −57 mV, SR = 11.9 spikes/s, R\text{in} = 26 M\Omega) was excited by BF-tones, sideband tones, and broadband noise. In contrast to the previous examples, this cell showed no appreciable sustained depolarization during the stimulus, but did exhibit prolonged afterhyperpolarizations. The PSTH data clearly show a pause following the first spike (Fig. 4E) and regular discharges in the steady state (Fig. 4F).

Cell 97002 (RP = −66 mV, SR = 0 spikes/s, R\text{in} = 14 M\Omega) is classified as a pauser unit, since the mean interspike interval was initially long and then declined to a constant steady-state value (Fig. 5F). The BF rate-level curve is characterized by a low threshold and firing rates that decreased steadily for levels above 40 dB SPL (Fig. 5D). The cell was excited by broadband noise and sideband tones without a rollover in the rate at high levels. During excitation, action potentials with deep undershoots were superimposed on a large sustained depolarization followed by little or no afterhyperpolarization (Fig. 5, H–J). Figure 5G shows that in response to a tone more than an octave above BF, the cell still experienced sustained, albeit subthreshold, depolarizations. Thus in contrast to some of the previous examples, there was no indication of sideband inhibition.

Cell 97054b (RP = −54 mV, SR = 20.2 spikes/s, R\text{in} = 16 M\Omega) was excited by BF tones and more weakly excited by below-BF tones and broadband noise (Fig. 6D). Excitation was

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**FIG. 3.** Fusiform cell 97055a. Same organization asFig. 1, except arrowhead in A points to the site on the soma from where the axon arises (axon not drawn).
accompanied by minimal sustained depolarization, but substantial afterhyperpolarizations (Fig. 6, H–J). The above-BF rate-level curve in Fig. 6D shows inhibitory responses at high levels. Under such stimulus conditions, the membrane depolarized weakly and then underwent a large hyperpolarization that outlasted the stimulus by several milliseconds (Fig. 6G). This cell is a pauchopper unit, as evidenced by the long first interspike interval and subsequent chopping pattern (Fig. 6E).

Sustained stimulus-evoked changes in membrane potential

Fusiform cells often exhibited sustained depolarizations and afterhyperpolarizations in response to acoustic stimulation, as illustrated in Fig. 7. For five cells, responses to BF tone bursts of increasing level are shown. Three responses were averaged at each sound level and the plots scaled to emphasize the slow potential changes; consequently, the action potentials have been attenuated and in some cases, clipped. Also, note that the data were collected in 5-dB increments, but for purposes of clarity, only every other level is shown.

In general, both sustained depolarizations and afterhyperpolarizations grow monotonically in amplitude with increasing sound level, as is the case for fusiform cells in the anesthetized cat (Rhode et al. 1983). Figure 7A shows an example of a cell that showed sustained depolarizations during the stimulus, but no afterhyperpolarizations. Stimulus-evoked depolarization was apparent at 20 dB SPL, but did not reach spike threshold. The depolarization grew larger with increasing sound level, reaching spike threshold at 50 dB SPL, after which it remained relatively constant in amplitude. This cell was one of the few in our survey that showed conspicuous postsynaptic potentials.

Figure 7, B and C, shows two cells that did not have spontaneous activity and for which the sustained depolarizations were accompanied by pronounced afterhyperpolarizations. The latter grew in both amplitude and duration with increasing sound pressure level. Afterhyperpolarizations were often followed by rebounds, indicated by the arrowheads in Fig. 7, B and C. In most cases, the rebound did not reach the resting potential, but a nonmonotonic membrane potential trajectory was nevertheless apparent.

Figure 7 also demonstrates that afterhyperpolarization had a profound effect on spontaneous firing. For the cell in Fig. 7D, the accumulated effect of afterhyperpolarization resulted in a
5-mV decrease in the resting potential and eliminated spontaneous firing altogether.

**Current-clamp results using the Manis paradigm**

A current-clamp paradigm was previously used in vitro to demonstrate the ability of a fast, inactivating K⁺ current to alter fusiform cell firing patterns (Kanold and Manis 1999; Manis 1990). This current was elicited by first deinactivating it with a hyperpolarizing pulse of injected current, then activating it with a depolarizing pulse. The resulting outward K⁺ current inactivated with a time constant of about 11 ms and opposed membrane depolarization, turning a chopper discharge pattern into a buildup or pauser pattern.

We used this current-clamp paradigm on nine fusiform cells to demonstrate the existence of the inactivating K⁺ current in vivo. Four of the cells showed results consistent with those described in the slice. Membrane potential waveforms for three such cells are shown in Fig. 8. For the lowest holding currents and smallest pulse amplitudes (bottom left of each panel), each cell showed a buildup pattern. The long-latency portion of the buildup is characterized by a gradual increase in the membrane potential toward spike threshold. The latency shifted to shorter values as the pulse amplitude increased (toward the top of each column). The latency also shortened at a fixed pulse amplitude when the holding current became less negative. Large pulse amplitudes sometimes evoked an onset spike before the buildup, creating a pauser discharge pattern (Fig. 8, B and C). Finally, at the largest pulse amplitudes and smallest holding currents, the cells responded with a chopping pattern for the duration of the pulse (top righthand plot in each panel).

Only four of the nine fusiform cells tested showed the results represented in Fig. 8. These had larger input resistances ($R_{in}$) than the remainder, which showed chopper responses unaffected by prior hyperpolarization. It is likely that, for the holding currents used ($\approx -1 \text{ nA}$), the latter group could not be sufficiently hyperpolarized to deinactivate the underlying K⁺ currents.

**Temporal response patterns**

For PSTH data from the DCN of anesthetized gerbil, Gdowski (1995) quantified the shape of the regularity histo-
gram by simultaneously fitting two lines to the mean ISI and noting the transient \( (m_T) \) and steady-state \( (m_{SS}) \) slopes. In the present study, however, there were several instances that seemed to call for a three-line fit (e.g., Fig. 9M). These cases typically had a sharp initial decrease in the mean ISI, followed by longer periods of transient and steady-state firing. Since the initial decrease was a common feature of these units, it was not a useful means of characterizing their behavior, and so a three-line fit was not attempted. Rather, the data were recomputed using 5-ms bins and the two-line fit repeated after eliminating the first point. The slopes of these two lines were called \( m_1 \) and \( m_2 \) to distinguish them from \( m_T \) and \( m_{SS} \).

For the data of Fig. 9M, the slope value \( m_1 \) captures the gradual increase in ISI over the first 50–75 ms, whereas the transient slope \( m_T \) as computed in earlier studies would be dominated by the very brief initial decrease. The positive value of \( m_1 \) is interpreted as weak inhibition, in that the trend of increasing ISIs is reminiscent of the behavior of auditory nerve fibers and so does not reflect significant modification by inhibitory processes. This is in contrast to Fig. 9C, for example, for which the slope value \( m_1 \) is steeply negative. The negative value indicates that the ISI trend is opposite that of the underlying excitatory drive from auditory nerve fibers. It is thus interpreted as reflecting an inhibitory input that is initially strong and gradually weakens over the first half of the stimulus.

The regularity histograms in Fig. 9, A–P, are plotted in order of increasing \( m_1 \) values. Across this sample of the fusiform cell population, the interval varies from steeply negative \( (A) \) to steeply positive \( (P) \). This variation is also apparent in the distribution of \( m_1 \) values plotted in Fig. 10A.

Another metric for quantifying the extent to which inhibition contributes to DCN physiology is the normalized tone slope (Davis et al. 1996; Young and Voigt 1982). This quantity is measured by first finding the level at which the BF rate-level function either saturates or becomes nonmonotonic. From this level up, a line is fit to the rate data and the slope divided by the maximum firing rate to give a normalized tone slope in units of dB\(^{-1}\). More negative tone slopes indicate more highly nonmonotonic rate-level functions and therefore responses characterized by greater inhibitory influence at higher levels.
Figure 10B shows the distribution of fusiform cell normalized tone slopes. In general, the values are not distributed as evenly as those of the regularity slope. A majority of the values (9/17) are clustered between $-4 \cdot 10^{-3}$ and $-8 \cdot 10^{-3}$ dB.

**DISCUSSION**

This report describes 17 fusiform cells obtained as part of an in vivo intracellular recording and labeling survey of the DCN in barbiturate-anesthetized Mongolian gerbils. Previous reports of the acoustic response properties of identified fusiform cells are limited to one set of studies in anesthetized cats (Rhode et al. 1983; Rhode and Smith 1986; Smith and Rhode 1985) and one study in decerebrate gerbils (Ding et al. 1999). The present study forms a bridge between the previous two in that it combines the anesthetic state of the former and the animal model of the latter. Understanding this relationship is important since DCN physiology differs significantly between decerebrate cats and gerbils (Davis et al. 1996), and between the barbiturate-anesthetized and unanesthetized decerebrate preparations (Evans and Nelson 1973; Fan 2000; Gdowski and Voigt 1997).
Afterhyperpolarizations are often of sufficient magnitude and long-lasting afterhyperpolarizations of similar magnitudes. Depolarizations about 10 mV in amplitude during the stimulus and afterhyperpolarizations of the membrane in response to cat and the gerbil is the presence of sustained depolarizations study. Similar results were obtained in this type, although responses were shown to depend on stimulus frequency and intensity. Similar results were obtained in this study.

Another feature of fusiform cell physiology common to the cat and the gerbil is the presence of sustained depolarizations and afterhyperpolarizations of the membrane in response to acoustic stimulation. Fusiform cells in both species exhibit depolarizations about 10 mV in amplitude during the stimulus and long-lasting afterhyperpolarizations of similar magnitudes. Afterhyperpolarizations are often of sufficient magnitude and duration to eliminate spontaneous activity.

Some differences were apparent, but it was not clear whether these were interspecies differences or whether they resulted from methodological differences. Pauser and buildup responses predominated in the cat, but a quarter of the cells in our study (4/16) were classified as chopper units. Furthermore, most of the pausers were subclassified as pauchoppers (6/9), indicating a relatively brief pause followed by a sustained period of regular firing. The responses of DCN neurons are known to be sensitive to interstimulus interval, likely as an effect of the long-lasting afterhyperpolarization (Rhode and Smith 1986). The longer interstimulus interval in the present study (1 s vs. 100 ms) probably allowed greater recovery from the hyperpolarization, resulting in response patterns with more chopper-like character.

The similarity in DCN physiology between the cat and gerbil in the barbiturate-anesthetized preparation contrasts with results from the decerebrate preparation. Specifically, Davis et al. (1996) found that the incidence of type IV units, which show strong on-BF inhibition, was much lower in the gerbil than in the cat (11% vs. 32–45%). Antidromic stimulation studies indicate that at least some fusiform cells in the decerebrate cat are type IV units (Young 1980). In contrast, an intracellular recording and labeling study in the decerebrate gerbil found that 12 of 13 identified fusiform cells were type III units (Ding et al. 1999), which have V-shaped excitatory receptive fields flanked by sideband inhibition. The one fusiform cell in that study with type IV unit properties was actually a type IV-T unit, which exhibits relatively weak on-BF inhibition. Thus an interspecies difference in the balance of excitation and inhibition appears to be masked through the suppression of inhibitory effects by barbiturate anesthesia (Evans and Nelson 1973; Fan 2000; Young and Brownell 1976).

Quantification of discharge patterns

Discharge patterns were quantified using a two-line fit to the ISI plot (Fig. 9). The first bin, representing 5 ms of data, was excluded so that, as described in RESULTS, the value of \( m_1 \) did a better job of quantifying the rate trend over the first 50–75 ms of the response than did the transient slope \( m_T \) used in the classification scheme of Gdowski (1995). Also, for the data in this study, the value of \( m_1 \) appears to be a more useful measure of inhibition than the normalized tone slope, because its values are more evenly distributed over a wider interval (Fig. 10). Normalized tone slope was devised as a useful metric for distinguishing type II units from type III units in the decerebrate cat (Young and Voigt 1982) and appears not to be as well suited for identifying trends within the cell population in this study.

The slope \( m_1 \), as described here has not been used in the past to analyze PSTH data. Previous studies have used the transient slope as one means of dividing data into discrete categories (Blackburn and Sachs 1989; Gdowski 1995; Parham and Kim 1992; Young et al. 1988). In this study it is known that all of the PSTHs, regardless of shape, arise from the same neuron type. The focus of the present analysis was to quantify regularity histogram shape using a continuous measure within a small, homogeneous neuron population, rather than to identify discrete categories within a large, heterogeneous population. The interpretation of \( m_1 \) as a measure of inhibition is reason-

![Image](http://jn.physiology.org/doi/fig/10.22033.1/0937-419X-87-5-2516-2516)
able in this study because the cells are anatomically similar and presumably receive similar sets of synaptic inputs.

Fusiform cells likely receive input from vertical cells (Saint-Marie et al. 1991; Voigt and Young 1980, 1990; Zhang and Oertel 1994), which discharge in a pattern characterized by transiently decreasing interspike intervals (Rhode 1999 and our own unpublished observations). They are also thought to receive input from a source of wideband inhibition (Nelken and Young 1994; Spirou and Young 1991), possibly arising from stellate cells in the PVCN that discharge with an onset-chopper pattern (Oertel et al. 1990; Smith and Rhode 1989). Since the fusiform cell PSTHs presented here were derived from responses to BF tones, it is more likely that vertical cells are responsible for the observed inhibitory effects, since the onset-choppers of the PVCN typically respond better to broadband noise than to tones (Winter and Palmer 1995). Whatever the source, the results suggest that the strength of the inhibitory input varies significantly across the fusiform cell population.

**Responses to pulses of injected current**

Individual cells were capable of discharging in pauser, buildup, and chopper patterns, depending on current pulse amplitude and prior hyperpolarization (Fig. 8). These results suggest that the fusiform cells of the adult gerbil express an inactivating $K^+$ conductance similar to that presumed to influence discharge patterns of fusiform cells in vitro (Kanold and Manis 1999; Manis 1990). Furthermore, the effect of these currents can be observed in vivo despite spontaneous synaptic bombardment arising from the parallel fiber network and the peripheral auditory system.
About one-half of the fusiform cells tested in this study showed the voltage-dependent effect as opposed to 90–95% of the fusiform cells in the guinea pig slice (P. B. Manis, personal communication). Input resistance was a limiting factor in producing appreciable latency shifts, and the input resistances in this study were smaller than those in the guinea pig slice (17.9 ± 9.7 MΩ vs. 27.0 ± 16.6 MΩ) (Manis 1990). The presence of spontaneously active input in our preparation may account for this difference, since such input reduces cell input resistance (Destexhe and Paré 1999; Rapp et al. 1992).

**Fusiform cells exhibit a variety of response properties**

The data in Table 2 show that the fusiform cells in this study exhibited a wide variety of response properties. The resting potentials varied from −76 to −50 mV. The input resistances spanned more than an order of magnitude, from 3 to 37 MΩ. Spontaneous activity ranged from 0 to more than 50 spikes/s, relative noise index from 0 (no noise response) to greater than unity (responds better to noise than to tones), and the normalized slopes of the BF rate-level curves vary from essentially flat to steeply nonmonotonic.

Furthermore, there is no obvious correlation of the different response measures. For example, among the cells with low spontaneous activity, there are those with small (e.g., **cell 97002**, Fig. 5 and 97073, Fig. 1) and those with large (e.g., **cell 97022**, Fig. 2) input resistances. Similarly, there are pauser units showing little evidence of inhibition (e.g., **cell 98001**, Fig. 4) and pausers with pronounced inhibitory responses (e.g., **cell 97022**, Fig. 2).

What underlies this diversity? A large body of evidence indicates that DCN principal cell responses are shaped by a relatively complex network of acoustically driven inputs. These inputs include excitatory drive from the auditory nerve (Smith and Rhode 1985), narrowband inhibition from vertical cells (Saint-Marie et al. 1991; Voigt and Young 1980, 1990), and wideband inhibition (Nelken and Young 1994; Spirou et al. 1993), possibly originating in the PVCN (Oertel et al. 1990; Smith and Rhode 1989; Zhang and Oertel 1994). In addition, the apical dendrites of fusiform cells are the targets of a second neural network driven by nonauditory inputs (Golding and Oertel 1997; Itoh et al. 1987; Kevetter and Perachio 1989; Muguinaini et al. 1980a; Weedman and Ryugo 1996; Weinberg and Rustioni 1987; Wright and Ryugo 1996). The observed diversity of physiological behavior likely results from cell-to-cell variations in the balance of activity within and across these networks. The techniques used in this study do not allow us to gauge directly this balance of activity, but to the extent that the interaction of a particular cell with these networks is determined by the details of its morphology, the anatomical data may provide some insight into the physiological diversity. A detailed quantitative comparison of fusiform cell physiology and morphology is the subject of the companion paper.

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