Effects of Adaptation on Neural Coding by Primary Sensory Interneurons in the Cricket Cervel System

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Clague, Heather, Frédéric Theunissen, and John P. Miller. Effects of adaptation on neural coding by primary sensory interneurons in the cricket cercal system. J. Neurophysiol. 77: 207-220, 1997. Methods of stochastic systems analysis were applied to examine the effect of adaptation on frequency encoding by two functionally identical primary interneurons of the cricket cercal system. Stimulus reconstructions were obtained from a linear filtering transformation of spike trains elicited in response to bursts of broadband white noise air current stimuli (5-400 Hz). Each linear reconstruction was compared with the actual stimulus in the frequency domain to obtain a measure of waveform coding accuracy as a function of frequency. The term adaptation in this paper refers to the decrease in firing rate of a cell after the onset or increase in power of a white noise stimulus. The increase in firing rate after stimulus offset or decrease in stimulus power is assumed to be a complementary aspect of the same phenomenon. As the spike rate decreased during the course of adaptation, the total amount of information carried about the velocity waveform of the stimulus also decreased. The quality of coding frequencies between 70 and 400 Hz decreased dramatically. The quality of coding of frequencies between 5 and 70 Hz decreased only slightly or even increased in some cases. The disproportionate loss of information about the higher frequencies could be attributed in part to the more rapid loss of spikes correlated with high-frequency stimulus components than of spikes correlated with low-frequency components. An increase in the responsiveness of a cell to frequencies >70 Hz was correlated with a decrease in the ability of that cell to encode frequencies in the 5-70 Hz range. This nonlinear property could explain the improvement seen in some cases in the coding accuracy of frequencies between 5 and 70 Hz during the course of adaptation. Waveform coding properties also were characterized for fully adapted neurons at several stimulus intensities. The changes in coding observed through the course of adaptation were similar in nature to those found across stimulus powers. These changes could be accounted for largely by a change in neural sensitivity. The effect of adaptation on the coding of stimulus power was examined by measuring the response curves to steps in stimulus power before and after exposure to an adapting stimulus. Adaptation caused a loss of information about the mean stimulus power but did not cause any improvement in the coding of changes in stimulus power. The unadapted response of the cells did not show any saturation even at the highest powers used in these experiments.

INTRODUCTION

The phenomenon of adaptation has been observed in nearly all sensory systems, yet for only a few systems do we have a quantitative understanding of how adaptation affects neural coding. Extensive studies of adaptation have been carried out in several different retinal systems and have led to the hypothesis that adaptation may provide a means to optimize coding under the constraints of limited channel capacity. In retinal systems, adaptation appears to function as a gain control mechanism, allowing cells to maintain responsiveness over a wide range of intensities (Laughlin 1989a,b; Normann and Werblin 1974; Shapley and Enroth-Cugell 1984). As retinal cells change their response characteristics through the course of adaptation, their ability to encode the mean light intensity decreases. This adaptation allows cells to preserve dynamic range for the encoding of modulations around the mean light intensity. These aspects of the stimulus are presumably more relevant from a behavioral standpoint.

The effect of adaptation on neural coding in most other sensory systems is less well understood, in part because of the difficulty in determining precisely what aspects of the stimuli are being encoded. For visual stimuli, stimulus intensity is generally taken as the DC light intensity. Auditory stimuli however are waveform signals for which the DC level is 0. None the less, based on physiological and psychophysical responses, intensity parameters for auditory signals can be defined. For example, for a pure tone, the amplitude of the sinusoid is a stimulus parameter analogous to DC light intensity. For a complex waveform, the average stimulus power can be taken as a measure of the stimulus intensity. By analogy with the visual system, one therefore might be interested in investigating the effect of adaptation on the coding of the power of a signal waveform. Primary auditory afferents have been shown to reduce their spike rates after onset of tone burst stimuli (Kiang 1965; Westerman and Smith 1984; Yates et al. 1985). However, the role of this adaptation in gain control of a cell’s response to the mean power of an auditory stimulus, analogous to gain control of a retinal cell’s response to the mean absolute value of a light stimulus, is unclear.

In addition to information about the mean power of a stimulus, auditory cells can encode information about the precise stimulus waveform. This ability is reflected in behaviors such as phase-locking, exhibited by some auditory neurons. Adaptation has been shown to change the response characteristics of some auditory afferents in complex ways that must reflect changes in the response of the cells to the waveform of the stimulus. (Delgutte and Kiang 1984; Epping 1990; Fay 1986). For example, Epping has shown that the responses of frog neurons to mating calls are qualitatively different when the cells are adapted to randomly structured background noise as compared with responses elicited when the cells are in an unadapted state. In this case, does adaptation allow the cell to optimize coding of the biologically relevant components of the stimulus? To address this ques-
tion, it is necessary to identify those aspects of a stimulus about which a cell discards information and to examine the effect of adaptation on the coding of those aspects about which information is preserved.

We have studied adaptation in two primary sensory interneurons in the cercal sensory system of the cricket to explore in detail the effect of adaptation on coding. We use the term adaptation to describe the decrease in firing rate of these cricket cells after the onset or increase in power of a white noise stimulus. The increase in firing rate after stimulus offset or decrease in stimulus power is assumed to be a complementary aspect of this same phenomenon. We examined the effect of adaptation on coding of both the stimulus waveform and changes in stimulus power with particular emphasis on whether adaptation improved the coding of particular aspects of a stimulus at the expense of other aspects.

The cercal system of the cricket (and many other orthopteran insects) is composed of two appendages covered with mechanoreceptor hairs that are sensitive to air current displacements in the horizontal plane surrounding the animal (Edwards and Palka 1974; Landolfa and Jacobs 1995; Landolfa and Miller 1995; Palka et al. 1977; Tobias and Murphy 1979). The cercal system is used by the cricket to detect approaching predators and generate escape behavior (Gnatzy and Hustert 1989; Gnatzy and Kämper 1990; Hoyle 1958; Sterle et al. 1994). In addition, neurons in this system are sensitive to air currents of the songs produced by conspecifics during mating behavior (Davis and Liske 1988; Kämper and Dambach 1985). Primary mechanosensory afferents connected to individual mechanoreceptor hairs have sensitivities that span the 5- to 1,000-Hz range (Roddey and Jacobs 1996). Twelve pairs of projecting interneurons have been identified in the terminal ganglion (Jacobs and Murphy 1987). These receive direct input from a subset of the primary afferents and can respond to air current stimuli over a wide range of frequencies and intensities (Miller et al. 1991).

In the present study, we examined the changes in the coding of a broadband stimulus waveform through the course of adaptation in the right-left pairs of the cricket primary cercal interneurons 10-2 and 10-3. In previous work, we used a stochastic systems analysis to characterize the frequency tuning and quality of coding of these two interneurons in their fully adapted state. In those studies, it was shown that the cells encoded frequencies in the 5- to 70-Hz range and, with the exception of their directional sensitivity, had identical dynamical encoding properties. Here we examine the effect of adaptation on coding of both the stimulus waveform and the stimulus power. Specifically, we addressed the following two questions: What is the effect of adaptation on the accuracy with which these cells encode information about different frequency components of a complex stimulus waveform? Does adaptation perform a gain control function to improve the ability of the cell to encode changes in stimulus power? In addition, we investigated if the observed behavior could be accounted for completely by a shift in neural sensitivity or whether adaptation involves more complex changes in coding characteristics.

To address these questions, we designed three sets of experiments. In the first, we examined changes in frequency coding through the time course of adaptation by measuring the response of these cells during 750-ms intervals of 5–400 Hz white noise stimuli. In the second set of experiments, we determined whether these changes could be accounted for by a change in neural sensitivity by characterizing coding and adaptation behavior over a broad range of stimulus intensities. In the third set of experiments, we investigated whether adaptation improved the ability of the cell to detect changes in stimulus power by measuring response curves to steps in stimulus power before and after exposure to an adapting stimulus.

**METHODS**

All experimental methods were similar to those used in previous studies, reported in detail elsewhere (Theunissen et al. 1996). A brief summary of the techniques is presented here.

**Dissection and preparation of specimens**

All experiments were performed on adult female crickets (Acheta domestica) obtained from a local supplier (Bassett’s Cricket Ranch, Visalia, CA). Specimens were selected that had undergone their final molt within the preceding 4–24 h. The head, legs, and wings were removed from the specimen, and a thin flap of cuticle was removed from the dorsal surface of the abdomen. After removal of the gut and fat tissue, the body cavity was rinsed and subsequently perfused with hypotonic saline (O’Shea and Adams 1981). Hypotonicity facilitated microelectrode penetration of the ganglion sheath. To further facilitate electrode penetration, the sheath was partially digested with a 3% solution of protease (Sigma) in standard hypotonic saline for a 30-s period.

During experiments, the cricket preparation was pinned onto a thin disk of silicone elastomer (Sylgard, Dow Chemical). This disk fit snugly into a hole in the middle of the base of the air current stimulus chamber described below. The abdomen was suspended 2–4 mm above the Sylgard surface on three minutes pins at a height similar to the animal’s normal standing height when legs were intact. This configuration ensured that air could move under and around the abdomen in a natural fashion. The terminal ganglion was stabilized on a miniature Ag/AgCl spoon, which was introduced through a narrow slot in the roof of the chamber. The spoon also served as the ground lead, and was held in place by a micromanipulator.

**Air current stimulus generation**

All experimental recordings were obtained from crickets mounted within a Plexiglas air current stimulus chamber. Laminar air currents were created within the chamber by the movement of two audio loudspeakers mounted on either side of the chamber. Computer-generated voltage waveforms having a variety of precisely controlled shapes and amplitudes were used to drive the speakers. Two types of waveforms were chosen for this study: half-cycles of sine waves, used for determining the optimum excitatory direction of the interneurons (Miller et al. 1991), and band-passed white noise signals. Peak air current velocity was varied by changing the amplitude of the driving waveform. The design and calibration of the chamber and the stimulus generation system have been described in more detail elsewhere (Theunissen et al. 1996).

The entire stimulus apparatus was mounted within a sound-insulated enclosure resting on top of an air-flotation vibration-isolation table. Ambient air current displacements within the enclosure were below the limits detectable with the most sensitive sound pressure and air current-sensitive microphones available (Bennet-Clark 1984).
**Intracellular recording**

Microelectrodes were filled with 3 M KCl solution and had resistances ranging from 20 to 50 MΩ. A small hole in the roof plate of the chamber provided access for the electrode. With the aid of a dissecting microscope, the microelectrode was positioned above the terminal ganglion and then advanced into the connective nerve leaving the ganglion. Electrical activity was recorded with an electrometer in bridge mode (Dagan 8800). Data were acquired with a digital data acquisition system (RC Electronics, Santa Barbara, CA) and synchronized with the wind stimulus waveform generator. Initially, interneurons were identified by visualization under a fluorescent microscope; however, the directional and dynamical properties of the interneurons 10-2 and 10-3 are so specific that with experience, they were identified based on their physiological properties alone. After penetration of either 10-2 or 10-3, the air current stimulus was positioned at the direction that produced the maximum response for that interneuron.

**Experimental protocol**

Three types of experiments were performed. The first type measured changes in coding through the course of adaptation. In each adaptation experiment, 750-ms bursts of 5–400 or 5–70 Hz white noise wind were presented for 125–200 repetitions at a power density between 0.031 and 0.045 (cm/s)^2/Hz, for a total power between 12.5 and 17.5 (cm/s)^2. A different noise waveform was used for each stimulus presentation. A power level that was substantially above the cell’s threshold was selected in every preparation so as to produce a robust adaptation response. At least 7 s of silence preceded each burst. The cells fully recovered after 4 s of silence, as measured by the number of spikes in the first 100 ms after stimulus onset. During some experiments, the bursts of 5–400 and 5–70 Hz white noise wind were interspersed with bursts of other bandwidths of white noise. Responses showed no consistent differences between experiments in which a bandwidth was presented alone and experiments in which stimuli of several bandwidths were interspersed.

The second type of experiment measured the coding characteristics of the cells in the fully adapted state. Five different waveforms of 5–400 Hz bandpass white noise were presented for 5.5 s at total stimulus powers between 9.2 × 10^4 and 9.1 (cm/s)^2. The cells were assumed to be fully adapted after 2 s.

The third type of experiment measured the ability of the cells to detect changes in stimulus power. Each trial began with a period of 2.04 s during which the cells were exposed to either the ambient (very low) background noise level within the chamber or to a 5–400 Hz bandpass white noise stimulus of a specified RMS intensity. After this 2.04-s interval, the power level was stepped to one of seven possible power levels for a duration of 0.89 s. Seven to 13 trials were presented for each stimulus power combination, and the average spike rate was plotted. Stimuli were presented in random order, with a minimum of 5 s of silence between each trial.

**Analytical methods**

We used the stimulus reconstruction method (Bialek et al. 1991; Theunissen 1993; Theunissen et al. 1996; Warland et al. 1991) to quantify how the information encoded in the interneuron spike train about the dynamic aspects of the air current stimulus changed through the course of adaptation. In brief, an estimate of the stimulus waveform is derived from the evoked spike trains by minimizing the mean square difference between the estimate and the actual waveform. The quality of coding, defined as the amount of information carried about the stimulus by the spike trains, can be calculated by comparing the estimate of the stimulus with the actual stimulus. In this work, the estimate was obtained by a linear filter operation on the spike trains. The linear filter which satisfies the least square error constraint is given in the frequency domain by

\[ H(f) = \frac{(R^*(f)S(f))}{(R(f)R^*(f))} \]  

(1)

The numerator of Eq. 1 is the Fourier transform of the cross-correlation of the response \( R \) (the spike trains) with the stimulus waveform \( S \). The denominator is the power spectrum of the response. The asterisk denotes the complex conjugate and the brackets stand for the statistical average over an ensemble of stimuli and response pairs. \( H(f) \) is the linear filter that operates on the spikes trains to produce an estimate of the stimulus. As discussed in a preceding report (Theunissen et al. 1996), this linear filter is believed to decode a large fraction of the information embodied in the response spike patterns of these interneurons.

To calculate the optimal linear filter, a 100-ms segment was taken from each presentation of the 750-ms or 5.5-s stimulus. For each such sample, the cross-correlation in the frequency domain and the power spectrum of the spike train were computed. The linear filter then was obtained from the ratio in the frequency domain of the average across samples of the cross-correlation divided by the average across samples of the spike train power spectrum. The inverse Fourier transform of Eq. 1 was taken to produce the filter in the time domain. The Jackknife procedure (Efron 1982) was used to calculate the standard error of the filter amplitude at each frequency (or at each point in time) and to correct for any bias in the calculation of the mean value. The bias correction was insignificant for the filter calculation in most cases, but did have a small effect in the calculation of the overall accuracy.

A best linear estimate of a stimulus waveform was constructed by convolving the evoked spike train, \( R(t) \), with the optimal linear filter, \( H(t) \), of the neuron being studied

\[ S_\text{est}(t) = H(t)^*R(t) \]  

(2)

The function \( R(t) \) is equal to one if a spike occurred at time \( t \), and is equal to 0 otherwise.

To determine the stimulus encoding accuracies of the receptor neurons, the estimate of the stimulus then can be compared with the actual stimulus. If the encoding is independent across frequencies, the comparison can be done in the frequency domain by comparing independently each frequency component of the estimate to the corresponding frequency component of the actual signal. A measure, called “gain” in this formalism, is defined to quantify the correlation between \( S_\text{est}(f) \) and \( S(f) \) (Theunissen 1993). In general, best estimates of the stimulus waveforms must be constructed before the gain can be calculated. However, for the special case where the stimulus estimation scheme is restricted to a linear transformation of the spike trains, the gain can be calculated directly from the spike trains. This is because in the linear case the gain is equivalent to the coherence function, \( \gamma^2 \), of the stimulus-response pairs

\[ \gamma^2(f) = \frac{(S(f)R^*(f))(R(f)S^*(f))}{(R(f)R^*(f))(S(f)S^*(f))} \]  

(3)

The coherence function is a normalized measure of the correlation between the receptor responses and the stimulus waveforms. If the stimulus at some frequency can be reconstructed with no errors by a linear transformation of the spike train, then the coherence function, or gain, is equal to one at that frequency. If the coherence function is 0 over some frequency range, then a linear transformation of the spike train provides no information about the stimulus in that range. The coherence function is related to the more familiar signal-to-noise ratio, \( SNR \), by the following formula

\[ \gamma^2(f) = \frac{SNR(f)}{1 + SNR(f)} \]  

(4)
The signal in this case is the power of the estimate of the signal derived from the spike trains, and the noise is the power of the difference between the estimate of the signal and the real signal.

The overall coding accuracy over all frequencies can be expressed in information theoretic units in terms of the mutual information (also called the transinformation). For a Gaussian signal and Gaussian noise, the transinformation can be calculated directly from the coherence function

\[ T = \int df \log_2(1 - \gamma^2(f)) \] (5)

This information transmitted is expressed in bits/second. The derivation of the relations presented here are given in a previous report. For Eq. 5 to be correct, not only does the noise need to be Gaussian, but the noise at any frequency also must be independent of the noise calculated at all other frequencies. Evidence that this is the case for these interneurons was presented for the adapted regime in the same previous report (Theunissen et al. 1996). Deviations of the noise density from a Gaussian distribution would result in an under-estimation of the information transmitted using Eq. 5.

**RESULTS**

**Stimulus waveform coding through the course of adaptation to a 5–400 Hz white noise stimulus**

The responses of five cells (4 10-2 cells and 1 10-3 cell) to 750-ms intervals of band-passed 5–400 Hz white noise were recorded from five animals. Figure 1 shows the voltage trace to the speaker and the recorded spikes for one trial. To elicit the maximal adaptation effect, a high stimulus power was used [12.5–17.5 (cm/s)^2]. A plot of spike rate through time for all five preparations is shown in Fig. 2. The rate of decrease in spike rate through time varied from preparation to preparation. Three preparations showed an initial sharp decrease in mean spike rate followed by a slower decrease. One preparation showed a gradual decrease, and one preparation had a slight increase in spike rate followed by a gradual decrease. This variability in the rate of adaptation was not correlated with any aspect of our protocol. In all preparations, the spike rate was depressed to below the spontaneous rate after stimulus offset (data not shown).

**Calculation of Encoding Accuracy and Frequency Tuning.** Each cell’s frequency tuning was determined by calculating the coherence between the spike trains and the stimulus signal using Eq. 3. In the linear case, the coherence is equal to a normalized quantity called the “gain” which, in general, measures the correlation between an estimate of the stimulus waveform obtained from the spike trains and the actual stimulus waveform (see METHODS).

Figure 3 shows curves of gain versus frequency at two different times through the course of adaptation for one prep-
In all five preparations, transinformation in the high-frequency region decreased. The transinformation in the 5–70 Hz frequency region increased in three of the five preparations and decreased slightly in the other two preparations. Figure 4B shows a plot of transinformation versus time during adaptation for one of these two preparations. The two preparations that showed a decrease in the low-frequency transinformation had the greatest and most rapid decrease in spike rate. In both of these preparations, the transinformation for the high frequencies decreased faster than the transinformation for the low frequencies.

LINEAR STIMULUS RECONSTRUCTION FILTER. Figure 5 shows the optimal linear filters at different points in time for two different preparations. This filter is that which, when convolved with the spike trains, produced the best estimate of the stimulus on average. The filter also can be thought of as the best linear estimate of the air current displacement that preceded a spike, with time 0 indicating the time of spike occurrence. The time interval between the peak of the filter and the time 0 is an indication of the mean latency of the system or the average time lag from when the peak air current displacement occurred to when a spike was generated (Theunissen et al. 1996).

In the first 100 ms after stimulus onset, the filter for each preparation showed a broad peak with a latency of 10–13 ms and a narrow peak with a latency of ~7 ms. The width of the broad peak was ~15 ms, corresponding approximately to the peak of the gain curve in the low-frequency region (33 Hz). The width of the narrow peak was ~3 ms, which corresponds to gain in the high-frequency region (167 Hz). It is noteworthy that the latencies for low- and high-frequency components of the filter were not equal.

Through the course of adaptation, the narrow peak diminished in amplitude, disappearing completely within 750 ms in two preparations. In the other three preparations, the narrow peak was still present (although diminished) after 750 ms. These three cells adapted to a higher spike rate (>60 spikes/s) than did the other two cells (which adapted to ~45 spikes/s at the end of the 750-ms stimulus). It would appear that those cells that achieved a lower adapted spike rate had a more complete loss of sensitivity to the high-frequency components of the stimulus than those that adapted to a higher spike rate.

The broad peak in the linear filter increased in amplitude through the course of adaptation in all five preparations.
These results indicate that as the cells adapted, the average per-spike correlation with high-frequency components of the stimuli deteriorated whereas the correlation with low-frequency components of the stimuli improved.

FUNCTIONAL BASIS FOR THE FREQUENCY DEPENDENCE OF THE ADAPTATION. Two factors could have contributed to the changes in filter shape and the disproportionate decrease in the amount of information about the high-frequency stimulus components during the course of adaptation. First, there could have been a deterioration in the phase-locking of individual spikes to the high-frequency components. Second, there could have been a disproportionate loss of responsiveness (i.e., a decrease in the number of elicited spikes) to the high-frequency components. Although the distinction between these two possible contributing factors is easy to imagine, it was extremely problematic to evaluate the phase-locking of individual spikes. In any given spike train response pattern, it was not possible to identify individual spikes as corresponding to low- or high-frequency stimulus components. However, by measuring the amount of power in the spike trains at the two frequency ranges, it was possible to estimate the number of spikes elicited by particular frequency components of the stimulus. Therefore, we investigated changes in the spike power during the course of adaptation to determine whether there was any differential change in the number of spikes elicited by high- and low-frequency components of the stimulus.

Figure 6A shows the results of one set of calculations for a typical preparation. The solid curve plotted with circles shows the total power of the spike trains. The solid curve plotted with square symbols shows the amount of power in the responses at frequencies between 5 and 70 Hz (i.e., the low-frequency region). The solid curve plotted with triangles shows the amount of power in the responses at frequencies between 70 and 400 Hz (i.e., the high-frequency region). In this and all other preparations, power in the high-frequency region decreased more rapidly during adaptation than did the power in the low-frequency region. This is further demonstrated by the curve with square symbols in Fig. 6B, which plots the ratio of power in the spike trains in the 70–400 Hz frequency interval to the power in the 5–70 Hz frequency interval. These results indicate that more of the high-frequency spikes were lost during adaptation than were the low-frequency spikes. If all of the frequency-dependent adaptation phenomenon had been due to degradation of phase-locking within the high-frequency region, this plot would have been flat. Therefore, at least some of the change in the shapes of the filter and gain curves during adaptation can be explained by a change in the fraction of spikes elicited by low- and high-frequency stimulus frequencies.

It was important to determine that these changes in the fraction of spikes elicited by low- and high-frequency stimulus frequencies were due to a real change in the cells’ frequency sensitivity and were not merely an artifact of a drop in spike rate. To do this, we compared the observed results with a simulated “null case,” in which the simulated response characteristics did not change as the spike rate dropped. We constructed a simulated set of spike trains, in which the decreasing spike rate matched that of an actual experimental data set. However, the timing of the spikes within the simulated responses were determined by an algorithm that was independent of any factors that might have corresponded to a shift in the frequency sensitivity of the cells during adaptation. To create each simulated data record, eight copies of the first 100-ms segment of data from one experimental preparation were concatenated into a single 800-ms spike train. Next, spikes were eliminated randomly from each of the 100-ms segments until the number of spikes remaining in the simulated segment were the same on average as the number in the actual experimental data record at the corresponding interval in time. Because the same set of original data samples were used to create each 100-ms interval of the simulated data, the average response characteristics of the simulated data did not vary in time.

Figure 6B allows a comparison between the experimental and simulated data sets. The curve plotted with circular symbols shows the ratio of power in the spike trains in the 70–400 Hz frequency interval to the power in the 5–70 Hz frequency interval for the simulated data set (The equivalent curve derived from the experimental data, discussed above, is plotted with squares.) When spikes were eliminated randomly, there was no significant change in the ratio of power in the low frequencies to power in the high frequencies. This indicates that the disproportionate loss of power at the high frequencies through the course of adaptation observed in the experiments was significant and that the changes in the encoding characteristics of the cells must have been due to a change in their response properties.

Additional control calculations demonstrate further that the differences in the coding characteristics observed for the two frequency ranges were significant. The dashed curves in Fig. 4A plot the transinformation versus time during the course of adaptation calculated from simulated data, in which spikes were
discarded randomly as in the previous set of control simulations. The transinformation calculated from the experimental data for this same preparation are plotted with solid curves. In control calculations derived from simulated data for this and all the other preparations, transinformation in the low- and high-frequency regions decreased at equal rates. It is noteworthy that the total transinformation changed at identical rates for both the experimental and simulated data.

NONLINEARITIES IN ENCODING THROUGH THE TIME COURSE OF ADAPTATION. We wished to determine whether some portion of the frequency-dependent adaptation phenomenon described above could be attributed to nonlinearity in encoding during the process of adaptation. That is, we wished to determine whether the quality of encoding within the low-frequency region was dependent on the presence (or relative power) of stimulus components in the high-frequency region. To do this, we compared the responses of the cells elicited by wide band (5–400 Hz) stimuli with their responses elicited by narrow band (5–70 Hz) stimuli. The power spectral density for the two types of stimuli was the same. Therefore they differed only by the presence or absence of stimulus components between 70 and 400 Hz.

We collected data for the 5–70 Hz stimuli in four of the five preparations. The dashed curves in Fig. 6A show the results for one preparation and are presented in the same graph as the wide-band stimulus/response data to allow comparison with the control. Each of the three dashed curves is a plot of the power in the spike trains versus time, elicited in response to the 5–70 Hz stimuli. The top dashed curve represents the decrease in total spike power across all frequency components of the response. The cells fired at significantly lower spike rates in response to 5–70 Hz stimuli than in response to 5–400 Hz stimuli. (The total power of the spikes is directly proportional to the spike rate.) The rate of decrease in spike rate was similar in response to the two stimuli.

The lower two dashed curves represent the proportion of the total power within the two different frequency bands of the response. The center dashed curve represents the power of the spikes occurring at frequencies between 70 and 400 Hz elicited by the 5–70 Hz stimulus, and the lowest dashed curve represents the power of the spikes occurring at frequencies between 5 and 70 Hz elicited by the 5–70 Hz stimulus. The most notable feature of these plots is that the relative and absolute amount of power in the responses due to spikes occurring in the 5–70 Hz region was greater in response to the narrow-band stimulus than in response to the broadband stimulus. This result indicates that the cell fired more spikes correlated with low-frequency stimulus components when only those frequency components were present in the stimulus. As expected from these results, the transinformation for low-frequency stimulus components

FIG. 6. A: power in spike train through course of adaptation in preparation D. Total spike power and power in the 5–70 Hz and 70–400 Hz frequency regions are shown. Solid lines with symbols mark values in response to a 5–400 Hz band-passed white noise stimulus. Dotted lines mark values in response to a 5–70 Hz band-passed white noise stimulus. B: ratio of power in spike train in 5–70 Hz region to power in 70–400 Hz region. Observed values from preparation D and values for a simulated data set in which spikes were eliminated randomly are shown.

2 Note that there are two possible types of nonlinearities in the scheme with which a stimulus is encoded by spike trains. One type of nonlinearity arises if combinations of two or more spikes contain information about the stimulus waveform that cannot be decoded through an analysis of the timing of the individual spikes (that is, if a pair of spikes separated by a particular interval has a different meaning than would be predicted from summing the meaning of each individual spike offset by that observed interval). Such nonlinearities can be expressed as extra terms in the Volterra expansion of the stimulus reconstruction. The second type of nonlinearity reflects any sensitivity of the cell to nonlinear transformations of the stimulus waveform and is best expressed as extra terms in the expansion of the reconstruction of the spike trains (i.e., the “forward” reconstruction). If the reverse reconstruction is performed in a case where this second type of nonlinearity is present, then the spike trains will reconstruct a nonlinear transformation of the stimulus (such as the half-wave rectified waveform) better than it would reconstruct the stimulus itself. It is evident from the relatively large gain curves derived for these cells in this work that the first (i.e., linear) term in the stimulus reconstruction expansion represents a significant amount of information about the stimulus waveforms. Therefore, we assume that a significant proportion of the coding can be considered as being linear in the first sense. That is, we use only the first term of the Volterra expansion of the stimulus reconstruction. The nonlinearities discussed in the text are of the second type and provide clues as to which aspects of the stimulus are encoded by the spike trains (see DISCUSSION).
was higher when only the low-frequency band was present in the stimulus. Thus responsiveness of the cells to high-frequency stimulus components resulted in a decrease in the number of spikes phase locked to low-frequency stimulus components, thereby deteriorating low-frequency coding. This was found to be the case for all four preparations and indicates the presence of some nonlinearities in encoding.

Figure 6A shows data from one of the three preparations in which the transinformation for the low frequency stimulus band increased during the course of adaptation to 5–400 Hz stimuli. In those three preparations, the low-frequency transinformation decreased during the course of adaptation to the 5–70 Hz stimulus. This is shown in Fig. 7 for the same preparation used in Fig. 6A.

**Adaptation as a change in neural sensitivity**

The adaptation phenomenon we observed caused the cells 10-2 and 10-3 to decrease their responses after the onset of (or increase in power of) white noise stimuli. In the simplest case, this decrease in responsiveness could have resulted from a shift in the cells’ sensitivities to the stimuli (as measured by the stimulus intensity required to elicit a given response by a cell). In this case, any change in a cell’s encoding characteristics during adaptation would be entirely the result of this shift in its sensitivity, and its encoding characteristics at any time point during adaptation would be equivalent to the characteristics it would display after full adaptation to some different stimulus power. Alternatively, adaptation might change a cell’s response characteristics in more complex ways that could not be predicted from its adapted responses to different stimulus intensities, such as has been observed in some auditory neurons (Epping 1990).

To examine this issue for the cells under study, we determined whether the specific changes in coding, observed while the mean spike rate decreased through the course of adaptation, were similar to the differences in coding behavior between the adapted responses to stimuli of decreasing intensity. We measured the responses of four cells (1 10-2 and 3 10-3) to stimuli of varying intensity. The stimuli were 5.5-s segments of 5–400 Hz band-passed white noise at several different power levels. Figure 8 plots the change in spike rate through time after the onset of stimuli at four different power levels for one typical preparation. The cell responded to the onset of a stimulus with a high level of spiking activity and adapted to a lower spike rate over the course of 2 s. This effect was most pronounced at high stimulus intensities. At all intensities, the spike rate was depressed after stimulus offset, recovering to the spontaneous level within a few seconds. Higher powers produced more marked depression and slower recovery to the spontaneous level.

Figure 9 shows a plot of adapted spike rate versus the stimulus power for the four cells. Each cell had its own characteristic set of spike rates. Because of this interanimal variability, observations were limited to trends within individual preparations. There was no saturation of adapted spike rate at the stimulus powers reached during these experiments ($\leq 9.09 \text{ (cm/s)}^2$).

Figure 10, A and B, shows plots of the transinformation rates versus the average adapted spike rate for two preparations. For each cell, the upper curve is the total transinformation rate across all frequency components of the stimulus. The lower two curves in each panel show the transinformation rates for stimulus frequency components within the low-frequency range (5–70 Hz, square symbols) and the high-frequency range (70–400 Hz, triangular symbols). Note that for the elicited responses with low mean spike rates, very little transinformation was carried by the spikes about stimulus frequencies between 70 and 400 Hz. However, the transinformation carried by spikes in this high-frequency band increased at higher spike rates obtained at the highest stimulus power levels. As transinformation in the high-frequency region rose, transinformation in the 5–70 Hz region decreased in two preparations (for example, A of Fig. 10) and leveled off in two preparations (for example, B of Fig. 10).

Figure 11 shows the stimulus reconstruction filters obtained from a single cell at three stimulus powers. Each curve was derived from data recorded after the cell had fully adapted to the stimulus. These filters have been scaled to the stimulus amplitude. For these and all other preparations, the relative amplitude of the narrow peak with a 7-ms latency in the filter was correlated with stimulus power: the greater the RMS stimulus amplitude, the greater the amplitude of the filter peak. Conversely, the broad peak at $-13 \text{ ms}$ decreased in amplitude with increasing RMS amplitude of the stimuli. These results indicate that as the power level decreased, each spike became less correlated with high-frequency deflections on average, and more correlated with low-frequency deflections, as was seen through the course of adaptation.

For each of the above measures, the trends observed in the adapted response to stimuli of decreasing power levels were similar to the trends observed through the course of adaptation. There is no evidence to suggest that more complex changes in the cell’s response characteristics occurred during adaptation. Therefore, the coding changes that occurred during adaptation appear to be accounted for by a change in the cells’ sensitivity to the stimulus.

**Effect of adaptation on coding of stimulus intensity**

In the retina and other systems, adaptation has been described as a mechanism for gain control, through which a
a 100-ms time window starting at the time of the step in stimulus power.

A typical data set is shown in Fig. 12. As expected, adaptation to decreasing or moderately increasing step changes in the stimulus power resulted in the restoration of the cell’s mean activity level to very near its prestimulus level. Adapted levels differed from the prestimulus levels only in situations where the step power changes were extremely large. These results supported the notion that adaptation resulted in a decrease in information about the absolute power of the stimulus. This is further supported by the graphs shown in Fig. 13. The bold solid curve plots the spike rate during the first 100 ms after a jump in stimulus power (following a 2.04-s period of exposure to ambient background noise) to each of seven new power levels. The dashed curve shows the adapted spike rate recorded 0.89 s after the change in stimulus power. The adapted spike rates varied considerably less with stimulus power than did the initial response rates, as shown by the much shallower slope of the dashed curve compared to the solid curve. For a given integration time, the cells predicted the stimulus power less accurately from the adapted spike rate than from the initial unadapted spike rate.

If adaptation improved the relative sensitivity of the cell, the cell should have been better able to discriminate between the steps of stimulus power after it had adapted to a stimulus whose power was in the middle of that range of intensities. The thin solid curve in Fig. 13 shows the spike rates during the first 100 ms after a step in stimulus power that followed a 2.04-s period in which the cell had been allowed to adapt to a stimulus with a power of 252 (cm/s)². Exposure to the adapting stimulus shifted the response curve to lower spike rates but did not increase its slope, indicating that there was no significant change in the relative sensitivity of the cell to changes in stimulus power after adaptation.

**DISCUSSION**

The primary goal of this study was to determine the effect of adaptation on two aspects of the coding of a broadband stimulus: coding of the stimulus waveform and coding of changes in stimulus intensity (measured as the average stimulus power). We addressed the following two questions: How does adaptation affect the accuracy with which these
not appear that adaptation functions as a gain control mechanism to adjust the dynamic range to the intensity of the stimulus.

It should be noted, however, that the cells we studied did not show any saturation of their unadapted responsiveness, even to stimuli at the highest powers that were achievable with our wind generating apparatus [up to a stimulus power spectral density of 40.5 (cm/s)^2/Hz]. In cases in which adaptation has been shown to improve the ability of a cell to encode changes of stimulus intensity, the effect was largest in the range of stimulus intensities to which the cell’s response saturated (for example, Maddess and Laughlin 1985). Therefore an improvement in the ability of the cell to encode changes in stimulus power might become apparent if much higher stimulus intensities are used.

It is interesting that the response of these cells did not saturate at the stimulus powers used in the present experiments. In previous work on 10-2 and 10-3, the response of these cells to unidirectional half-sine 250-ms air puffs saturated at peak air current velocities ~1 cm/s (Miller et al. 1991). Such a 250-ms pulse has a bandwidth of ~4 Hz. At the maximum stimulus intensities used in the present experiments, the power spectral density was 40.5 (cm/s)^2/Hz so that a 4-Hz bandwidth stimulus of this same power spectral density would have peak velocities on the order of 18 cm/s. It is possible that these cells saturated at lower powers in the previous experiments because the stimuli were at a lower frequency than those used in the present experiments (2 Hz for the air puffs vs. the minimum frequency of 5 Hz in the white noise stimuli). Alternatively, the presence of high-frequency stimulus components may have shifted the cell’s saturation curve to higher stimulus velocities.

Further work must be done to determine the stimulus powers that cause the responses of these cells to saturate for both narrow band and wide band stimuli and whether, at power levels that induce saturation of the cell’s response, adaptation would in fact be useful as a gain control mechanism. Notably, however, for two types of natural stimuli for the cercal system that have been analyzed with respect to peak air current velocities, the maximum air current velocities generated were in the range of 1–2 cm/s (Gnatzy and Kämper 1990; Kämper and Dambach 1985). Although these
Experiments do not exclude the possibility of more intense biologically relevant stimuli, they make even more interesting the fact that these cells show such marked adaptation (and hence loss of information about the absolute average stimulus power) in this range of velocities in which there is no risk of saturation.

Effect of adaptation on frequency coding

Although adaptation did not appear to function as a gain control mechanism in this stimulus regime, it did have a frequency dependent effect on the ability of the cells to encode information about the stimulus waveform. As shown in Fig. 4, as the spike rate decreased through the course of adaptation, the total information about the stimulus waveform carried by the spike trains also decreased. However, in all of the preparations, the cells lost information about the high-frequency components of the stimulus faster they lost information about low-frequency components. The ratio of spike power in the low-frequency region to the power in the high-frequency region increased in all five preparations, implying that adaptation caused the cell to lose high-frequency spikes faster than low-frequency spikes.

Our results agree qualitatively with those of Fay (1986), who studied the effect of adaptation on frequency tuning in goldfish auditory nerve fibers. He found decreased spike rates in response to high-frequency tone bursts (600–1,000 Hz) during the second 25-ms interval after stimulus onset as compared with during the first 25 ms. The cells were equally responsive to low-frequency tone bursts (100–600 Hz) in the two time intervals. Although the high- and low-frequency ranges were defined differently than in our system, his results do agree with our observation that adaptation causes cells to lose sensitivity differentially to different frequency bands within their sensitivity range and, specifically, to adapt to high frequencies more than to low frequencies.

Transinformation in the low frequencies increased in some preparations. One of the most striking results of our analysis was that in three of five preparations, transinformation in the low frequencies actually increased in the first few hundred milliseconds. During this same period, the power of the spikes in the 5–70 Hz frequency region decreased, indicating that the improved transinformation was not due to an increase in the number of spikes dedicated to low-frequency stimulus deflections. We propose that spikes elicited by high-frequency components of the stimulus degraded the coding of low-frequency components. Thus, “high-frequency spikes” were eliminated, the transinformation in the low-frequency region increased.

Our experiments with 5–70 Hz stimuli provide support for the hypothesis that the increase in low-frequency transinformation seen in three preparations was due to a decrease in the number of high-frequency spikes (which contribute to a degradation of the low-frequency encoding). If this idea is correct, there should have been no increase in low-frequency transinformation through the course of adaptation to stimuli that did not contain high-frequency stimulus components. This was seen to be the case. None of those cells which showed an increase in low-frequency transinformation through the course of adaptation to a 5–400 Hz stimulus showed such an increase during adaptation to a 5–70 Hz stimulus (for example, Fig. 7). Because there were no high-frequency spikes to eliminate, the change in information rate reflected only the drop in the number of spikes available to encode the low-frequency components of the stimulus.

The absolute number of spikes elicited by low-frequency stimulus components dropped through the course of adaptation, which tended to lower the amount of information carried by the spike trains about the low frequencies. In the two preparations with the most rapid decrease in spike rate, there were net decreases in the low frequency transinformation. In the three other preparations, there were net increases in low-frequency transinformation. The fact that the low-frequency transinformation was not seen to change in a consistent manner over the five preparations indicates that the opposing effects upon coding due to the decrease in the number of high-frequency spikes and the decrease in the number of low-frequency spikes were similar in magnitude.

To demonstrate that the effect of the decrease in the degradation is of the right order of magnitude to have caused the observed increase in low-frequency transinformation seen in three preparations, we performed a simulation in which “high-frequency spikes” were added to spikes from the second 100-ms time interval in the response to a 5–400 Hz stimulus, such that the total spike rate was equal to that of the first 100-ms interval. In effect, we added back the high-frequency spikes that were presumably lost from the first to the second 100-ms time interval. For the source of these high-frequency spikes, we used spikes from the response of the neuron to a stimulus that contained only frequencies from 70–400 Hz. We simulated the case in which all of the drop in spike rate from the first to the second 100-ms was due to loss of high-frequency spikes. In the actual experimental data, both low- and high-frequency spikes were lost. Therefore the decrease in low-frequency transinformation upon adding these spikes should have been more than the difference between the second and first 100-ms time point. This was shown to be the case in Fig. 14.
A MODEL OF ENCODING BY 10-2 AND 10-3. The fact that spikes correlated with one frequency range degraded the coding of another frequency range is an indication that the coding of the waveform by the interneurons 10-2 and 10-3 is not entirely linear in frequency.\(^3\) Part of this nonlinearity must stem from the directional tuning of these cells. These neurons give a maximal response to air current stimuli presented from a particular direction. When a white noise stimulus was presented along the axis of this preferred direction, the cell only responded to positive deflections of the stimulus waveform. These cells reconstructed a half-wave rectification of the white noise stimulus better than the entire waveform. Half-wave rectification is a nonlinear transformation of the original stimulus waveform; such a transformation of a 70–400 Hz stimulus will contain power \(<\)70 Hz. Spikes correlated with half-rectified stimulus components between 70 and 400 Hz contributed power to the spike trains below 70 Hz. This spike power was not correlated with the stimulus \(<\)70 Hz and therefore deteriorated coding at low frequencies.

The half-wave rectification property of these cells does not completely account for the nonlinearity in frequency coding. If no other factors contributed to the nonlinearity of frequency coding, the number of spikes elicited in response to any particular frequency band of the stimulus would have been independent of the power of the stimulus in any other frequency band. In fact, the number of spikes elicited in response to the 5–70 Hz components of the stimulus (as approximated by the power of the spike trains between 5 and 70 Hz) was reduced when the stimulus also contained components between 70 and 400 Hz (Fig. 6A). It appears that the presence of stimulus components between 70 and 400 Hz decreased the number of spikes phase-locked to the 5–70 Hz stimulus components. This effect was largest in the preadapted state and at high stimulus powers, in which cases the sensitivity of the cells to high frequencies were the greatest.

A linear coding regime over a wide frequency band would require high enough firing rates that low-frequency air current transients could be distinguished from several consecutive high frequency transients. In reality, the firing rates of the interneurons 10–2 and 10–3 were much too low to achieve such linearity of coding. The firing characteristics observed in these neurons were such that at low amplitudes, each cell fired one or two spikes to low-frequency deflections and none to high-frequency deflections and at high stimulus powers, each cell fired only slightly more spikes to low-frequency deflections and one spike to large amplitude high-frequency deflections. Such behavior is characteristic of a threshold-crossing detector with a higher threshold for high-frequency deflections than for low-frequency deflections.

To quantify the effects of these nonlinearities, the coding accuracy for a nonlinear transformation of the stimulus can be calculated and compared with the coding accuracy for the untransformed stimulus. For example, the occurrence of frequency-dependent threshold crossings could be considered as the relevant aspect of the stimulus being encoded.\(^4\) In terms of the effect of adaptation, however, similar qualitative conclusions would be reached. That is, the accuracy of the coding of high-frequency threshold crossings decreased during adaptation at a much faster rate than for the low-frequency crossings.

**Mechanisms underlying adaptation**

ADAPTATION AS A CHANGE IN NEURAL SENSITIVITY. If the model of encoding behavior described above is correct, then the seemingly complex adaptive properties seen in these interneurons could be obtained by a uniform decrease in neuronal sensitivity across all frequencies. There would be no need to invoke more complex changes in encoding properties. Our experiments with stimuli of different intensities were consistent with this idea. The changes in coding and spike statistics that we observed through the course of adaptation could be accounted for largely by a change in neural sensitivity, as they were mimicked by a decrease in stimulus power.

It should be noted that we assumed that the cells were completely adapted after 2 s. At higher stimulus intensities, the spike rate was observed actually to drop slightly from 2 to 5.5 s after stimulus onset. However, this drop was small relative to that in the first second. Given that the majority of the change in both spike rate and coding properties of these cells occurred within the first 400 ms after stimulus onset, our assumption that the cells were adapted fully after 2 s was acceptable for the questions addressed here. We did not observe any differences in responsiveness through the course of an hour long experiment.

EVIDENCE FOR TWO POPULATIONS OF MECHANORECEPTOR INPUTS. The shape of the filter of 10-2 and 10-3 in the preadapted state and at high stimulus power is particularly interesting in light of the filter shapes of afferent neurons. Previous studies of these interneurons indicate that they receive...
synaptic input from afferents associated with long mechanoreceptor hairs (>1,100 μm), which have the lowest threshold of all the hairs and encode stimulus components in the lowest frequency range. The stimulus reconstruction filters for afferents associated with these long hairs are relatively broad and have latencies of ~10 ms (Roddey and Jacobs 1996). Afferents associated with short mechanoreceptor hairs (200–750 μm in length), which have higher thresholds and preferentially encode higher stimulus frequencies, have narrower filters with shorter latencies (~7 ms). The reconstruction filters for 10-2 and 10-3 cells at high stimulus intensities and in the preadapted state are bimodal, with broad and narrow peaks having latencies similar to the filters for these two different length classes of afferents. This similarity suggests that the neural input to 10-2 and 10-3 cells might include both short and long hair afferents. If this were the case, short hair afferent input to the interneurons would be activated at higher stimulus intensities and would adapt more quickly than long hair afferent input.

The distinct peaks in the interneuron filters, and the discontinuity of the gain curves between 75 and 100 Hz, may be indications that 10-2 and 10-3 cells sample from discontinuous populations of the mechanosensory hairs, drawing more input from short and long hairs than from the class of medium length hairs (between 750 and 1,100 μm). These medium hairs have a median gain ~100 Hz. Direct experimental measurements will be required to test this possibility.

**Biological relevance of adaptation**

The interneuron types 10-2 and 10-3 are 2 of the 12 bilaterally symmetric pairs of projecting interneurons that have been identified in the cricket terminal abdominal ganglion. They are responsive to the lowest range of air current velocities known to be detectable by the cercal system and have directional sensitivities that span the 360 deg of the horizontal plane around the animal. Behaviorally relevant signals in a cricket’s environment have power spectra that correspond to the velocity range of these cells (Gnatz and Kämper 1990; Kämper and Dambach 1985). Although other interneurons are sensitive to air current stimuli in this velocity range, none of these have been found to display significant directional tuning. Therefore the set of four 10-2 and 10-3 cells can be considered as a functional unit with the task of representing information about the direction of air currents in the lowest velocity range to which the cercal system is sensitive.

In our previous examination of the encoding properties of the neurons 10-2 and 10-3 in the adapted state, we described them as encoders of frequencies between 5 and 70 Hz. The ability of these cells to encode frequencies >70 Hz is only apparent at high stimulus intensities and in the preadapted state. Such changes in frequency tuning with stimulus amplitude are not unprecedented. For example, Möller (1983) has shown in rat auditory nerve fibers that frequency selectivity deteriorates at high stimulus intensities, as measured by the broadening of the frequency transfer function in response to pseudorandom noise. This phenomenon is believed to arise from changes in the tuning characteristics of the basilar membrane at high stimulus intensity (Rhode and Robles 1974).

Is the responsiveness of the neurons 10-2 and 10-3 to frequencies >70 Hz at high stimulus powers an integral part of their function in the cercal system or does it represent a breakdown of mechanisms that otherwise preserve frequency selectivity? It is impossible to know the answer to this question without understanding more about the operation of the cercal system and without better knowledge of the natural stimuli to which the cercal system responds. If the set of four 10-2 and 10-3 neurons can be best thought of as a low-frequency encoding system, then the increase in frequency specificity that occurs through the course of adaptation can be understood as a mechanism through which each cell can maintain accuracy for the encoding of those aspects of the stimulus that are of the most behavioral relevance.

Other projecting interneurons in the cricket terminal ganglion show encoding characteristics and adaptation behavior that are different from what we have described for 10-2 and 10-3. For example, we have observed several interneurons that are silenced completely after a few hundred milliseconds of exposure to continuous broadband white noise but sustain a response to short bursts of such a stimulus. It will be important to examine the relationship between adaptation and encoding for such cells having other functional roles in the cricket cercal system.

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