JOURNAL OF NEUROPHYSIOLOGY
Vol. 76. No. 5. November 1996. *Printed in U.S.A.*

# Light Enhances Hydrodynamic Signaling in the Multimodal Caudal Photoreceptor Interneurons of the Crayfish

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## SUMMARY AND CONCLUSIONS

*1*. The caudal photoreceptor (CPR) interneurons in the sixth abdominal ganglion of the crayfish are complex, multi-modal interneurons. These cells respond directly to light with tonic spike discharges, and they integrate synaptic input from an array of filiform mechanoreceptors on the tailfan. They also provide input to rostral command centers, inducing backward walking at high firing frequencies, and thus directly influence behavior.

*2*. We recorded CPR activity in response to weak hydrodynamic stimulation of the tailfan mechanoreceptors while under varying intensities of light shined on the sixth ganglion. Spike trains were characterized according to the mean discharge rate (MDR) and the power spectrum from which the signal-to-noise ratio (SNR) was calculated.

*3*. Illumination of the CPR enhances the efficiency of transmitting mechanosensory signals. It does so by increasing the SNR of mechanosensory input received from tailfan receptors. A sevenfold, nonlinear increase in the SNRs was observed with increasing light intensity, an effect especially pronounced for weak hydrodynamic stimuli. In comparison with the dark, illumination of the ganglion lowered the hydrodynamic threshold and heightened the response to suprathreshold stimulation. Unlike the SNR, the MDR is little affected by mechanosensory input.

*4*. These results are compared with simulated electronic activity from an analogue threshold model and are discussed with respect to the mechanism of stochastic resonance.

## INTRODUCTION

An important function of the nervous system is the integration of sensory information from different sources and/or modalities. For example, neurons in the deeper collicular layers of the cat respond to visual and acoustic stimuli with interactions that are both facilitory and inhibitory (review by Vidyasagar 1991). In the simpler crayfish nervous system, the caudal photoreceptor (CPR) interneuron provides a unique cellular model for multimodal sensory processing. The CPR is sensitive to light and responds to hydrodynamic stimulation of tailfan mechanoreceptors (Kennedy 1963; Prosser 1934; Wilkens and Larimer 1972). Also, CPR-mediated abdominal photosensitivity elicits behavioral responses (Welsh 1934) which are of considerable interest for sensorymotor integration. Here, we use the CPR to examine interactions between photic and mechanosensory inputs and the resulting effects on signal transmission.

Crayfish are largely nocturnal, e.g., *Procambarus clarkii* (Huner and Barr 1984), although they actively forage on cloudy days, in the evening, and in turbid water as well. As juveniles they are susceptible to predation by fish and aquatic insects and as adults to a variety of larger vertebrate predators. Light and water motion are important sensory components of the shallow aquatic environment, and for crayfish, the integration of both sensory modalities is undoubtedly critical for survival. Accordingly, the crayfish visual system has evolved as a sensory system with great temporal and spatial resolution (Wiersma et al. 1982), and it plays a prominent role in phototactic behaviors. In contrast to the eyes, the extraretinal CPRs mediate photokinetic responses associated with a preference for dark habitats (Welsh 1934). Strong illumination of the tail and/or CPRs (Edwards 1984; Welsh 1934) or direct, high-frequency electrical stimulation of the CPRs (Simon and Edwards 1990) initiates backward walking. Extraretinal photosensory systems are widespread among invertebrate phyla (Yoshida 1979); in some instances, as with the CPR, their functional roles are known, e.g., the caudal tail-spine photoreceptors in the horseshoe crab *Limulus* regulate circadian rhythms (Barlow 1990).

For detecting water motion, crayfish rely largely upon filiform sensory hairs located over the exoskeletal surfaces of the body. These near-field hydrodynamic receptors are sensitive to extremely small water displacements (0.1 $\mu$m) (Wilkens and Douglass 1994) and provide a powerful means for the crayfish to detect weak signals, e.g., currents or low-frequency vibrations (Kalmijn 1988; Tautz 1990a,b). As a mechanosensory interneuron, the CPR is excited monosynaptically by the filiform receptors on the tailfan. These hydrodynamic receptors are directionally sensitive (Douglass and Wilkens 1991; Tautz and Plummer 1994) and contribute to behavioral responses, including lateralized antennal reflexes to water currents near the tailfan (Schmitz 1992), and orientation behaviors to currents produced by swimming fish (Breithaupt et al. 1995). Thus the potential exists for involvement of the CPRs in a variety of behaviors that rely on multimodal processing of light and mechanosensory input.

The CPRs exist as a bilateral pair of identified interneurons arising from the sixth abdominal ganglion in both epigeal (Prosser 1934) and blind cavernicolous crayfish (Larimer 1966). Their use for the study of multimodal sensory integration lies with the fact that they respond directly to light with sustained depolarizations and that spontaneous or light-evoked activity is modulated extensively by mechanosensory input. The response to maintained illumination of the CPR dendrites, the light-sensitive region of the cell in the sixth ganglion (Wilkens and Larimer 1972), is a characteristic transient burst of spikes followed by a tonic discharge with a long time constant (Stark 1968). Early results (unpublished) indicate that in the absence of coherent stimula-

tion of the presynaptic mechanoreceptors, light-induced CPR activity is noisy. Although the mean discharge rate increases substantially in response to steady illumination, the time interval distributions of spikes are characteristically random. We refer to this spontaneous random process as the "internal noise" (Gerstein and Mandelbrot 1964; Moore et al. 1966). More specifically, the interval distributions are gamma-like at low light intensities and become Gaussian-like at higher intensities (Herman and Olsen 1967).

In this paper, we demonstrate that increased levels of illumination heighten CPR sensitivity to hydrodynamic stimuli and simultaneously increase the level of internal noise. Moreover, we provide evidence simulated using neuron models suggesting that the observed increase in sensitivity can be attributed to increases in the internal noise. Both sensory cells and CNS interneurons are inherently noisy. For the sensory nervous system, noise may originate externally in conjunction with environmental signals, as illustrated recently in the crayfish (Douglass et al. 1993) and cricket (Levin and Miller 1966). Moreover, noise may arise internally due to the random activity of ion channels (Bezrukov and Vodyanoy 1995; DeFelice 1981) as expressed in endogenous patterns of neural activity (Barlow et al. 1993). In effect, noise is an inherent component of natural systems and is an important consideration for information processing by the nervous system (Croner et al. 1993; Ferster and Sprusston 1995; Knight 1972a).

Recently it was demonstrated that noise can enhance detection of weak coherent signals and the flow of information through nonlinear systems by means of stochastic resonance (SR), a process observed in a variety of physical systems subject to stochastic forces (Moss 1994; Moss et al. 1994; Wiesenfeld and Moss 1995). Noise also may participate in multisensory processing in neural networks as has been proposed in theory and in numerical simulations with coupled and uncoupled parallel stochastic resonators (Collins et al. 1995; Jung and Meyer-Kress 1995; Jung et al. 1992; Lindner et al. 1995). Currently SR has been demonstrated in nervous systems, e.g., in sensory neurons of the crayfish (Douglass et al. 1993), shark (Braun et al. 1994), and crickets (Levin and Miller 1966) and in neuron models such as electronically simulated simple threshold (Gingl et al. 1995) and Fitzhugh-Nagumo (Moss et al. 1993) models. In the experiments described below, light has been shown to enhance the mechanosensory response of the CPR as measured by large, nonlinear increases in both the signal-to-noise ratio (SNR) of stimulus-evoked activity and correlograms of afferent-interneuron discharge patterns. These results are discussed in terms of potential neural mechanisms based on the premise of light-mediated regulation of internal noise and information transfer, including stochastic resonance. Each of the proposed mechanisms is tested in simulation experiments with the Fitzhugh-Nagumo electronic neuron model, the results of which are compared with the physiological behavior of the crayfish CPRs. A preliminary report of these experiments has appeared elsewhere (Pei et al. 1995b).

## METHODS

### *Preparation*

Crayfish (*P. clarkii*) were obtained from suppliers in Louisiana, bred, and raised to ~1 yr of age in large tanks. For the experiments, the abdomen and tailfan were isolated quickly, fixed ventral side uppermost in a Sylgard-lined chamber, and bathed in crayfish saline (van Harreveld 1936). The exoskeleton and muscles were removed to expose the ventral nerve cord in segments 4–6 and the five to six interganglionic connectives were desheathed to expose nerve bundles for recordings. After the CPR cells were located by removal of overlying fibers (Flood and Wilkens 1978), the nerve cord was cut between the fourth and fifth ganglia and transferred intact including the tailfan to a recording chamber filled with saline. The recording apparatus was supported on a vibration isolation table (Technical Manufacturing).

### *Stimulation*

Hydrodynamic stimulation was applied using techniques previously described (Wilkens and Douglass 1994). Briefly, the tailfan was mounted vertically on a small stage attached to the post of an electromechanical small-motion transducer (Pasco, SF-9324) driven sinusoidally by a function generator. The stimulus was the vertical movement of the stage on which the preparation was mounted and which induced a periodic relative motion between the hair receptors and the saline bath. The tailfan was rotated about an axis perpendicular to the stimulus direction so that the rostrocaudal axis was horizontal. As a result, vertical movements of the tailfan produced transverse hydrodynamic currents in the direction of maximum CPR mechanosensitivity (Wilkens and Douglass 1994). Stimulus amplitude was controlled by step attenuators (Hewlett Packard, 355C and 355D) with function generator output voltage held constant. Stimulus amplitude was attenuated between −80 and −35 dB and corresponded to displacement amplitudes of 0.017–2.4 $\mu$m and relative velocities of 0.905–128 $\mu$m/s at 10 Hz (stimulus calibration by laser-Doppler vibrometry, Polytec, Model OFV 501). Dynamic stimulus characteristics were selected within the optimum range of CPR sensitivity, as determined from frequency response curves measured at constant velocity (see example; Fig. 1*A*, *inset*). Because the CPR is sensitive to low stimulus frequencies (Flood and Wilkens 1978; Plummer et al. 1986; Wine 1984), the intensity response functions were analyzed primarily at 10 Hz, the approximate midpoint of the sensitivity range for low-pass mechanosensory interneurons.

Light was applied from a cold light source (Narashige Illuminator) via an optical fiber directed onto the ventral surface of the sixth ganglion. The unattenuated light intensity was 1.1 $\mu$W/mm$^2$ measured at the location of the ganglion; intensity was adjusted by neutral density filters (Oriel, 0.5–3 log units). Background light inside the recording cage was ~0.1 nW/mm$^2$, well below threshold intensity for the CPR and therefore essentially dark. Stimulus refers to the hydrodynamic stimulus unless otherwise specified.

### *Recording, data acquisition, and analysis*

In this study, we examine multimodal neural interactions based on experimental results from 20 CPR interneurons. Extracellular spike activity was recorded from the CPR axon in the connective between the fifth and sixth ganglia using a suction electrode and AC preamplifier (WPI, DAM 50). CPR spikes were identified from the light response, selected by window discriminator (WPI, Model 121), and stored digitally along with the stimulus trigger for off-line analysis. The power spectrum of the spike train was the primary method for measuring hydrodynamic sensitivity since spectral analysis is especially sensitive to periodic signals; power spectra were constructed by computer (DataWave Technologies, Common Processing Software), as described previously (Douglass et al. 1993). The SNR of spike activity at the stimulus frequency was calculated from power spectra as

$$\text{SNR} = \frac{\text{RESPONSE}}{\langle \text{mean noise} \rangle}$$

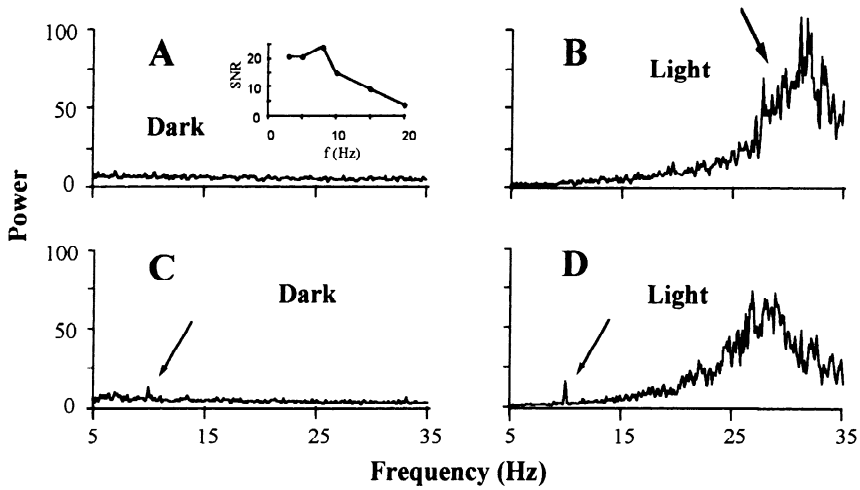


FIG. 1. Power spectra from a caudal photoreceptor (CPR) cell measured in dark (background light = 0.1 nW/mm$^2$, *A* and *C*) and in light (1.1 $\mu$W/mm$^2$, *B* and *D*). In *A* and *B*, power spectra were measured in absence of mechanical stimulation, whereas in *C* and *D* sinusoidal hydrodynamic stimulation of complete tailfan relative to saline at 10 Hz (peak displacement amplitude = 0.147 $\mu$m) was applied. Arrows in *C* and *D* indicate response peak of cell at stimulus frequency. Arrow in *B* points to a broad peak indicating an increase in regularity of discharge at higher frequencies in response to illumination. A similar peak occurs during mechanical stimulation (*D*). *Inset*: typical frequency tuning curve shown at 0.1 $\mu$W/mm$^2$ light intensity.

Here, the response is obtained by integrating a narrow band ($\pm$0.25 Hz) of the power spectrum containing the peak lying above the noise at the fundamental stimulus frequency. The ⟨mean noise⟩ is the average amplitude of broad-band background noise adjacent to the stimulus-induced response peak. At each light intensity, we measured the signal-to-noise ratio during the tonic phase, i.e., after the initial transient response of the CPRs. CPR responses also were characterized on the basis of mean discharge rates (MDR) and spike interval histograms for comparison of response profiles with SNR measurements. In addition, cross correlograms of CPR and presynaptic afferent discharges were obtained and compared at various light intensities.

## RESULTS

The CPR cells have a low endogenous MDR of 5–10 spikes/s in the absence of light and mechanical stimulation of the tailfan. These spontaneous discharges are characteristic of a random process with a flat power spectrum. A typical response is illustrated in Fig. 1*A*. Shining light on the caudal ganglion produces the well-known increase in tonic discharge of the CPRs. The corresponding power spectrum, which was flat in the dark, develops a broad peak between 25 and 35 Hz (Fig. 1*B*, bold arrow). The spike-interval distribution of spontaneous activity, measured independently, is a typical gamma function indicative of an irregular discharge pattern (Fig. 2*A*). Under strong illumination, the distribution pattern is compressed to a narrower, more Gaussian-like distribution (Fig. 2*B*).

In general, the CPR interneurons are highly sensitive to low-frequency hydrodynamic stimuli (Plummer et al. 1986) with threshold sensitivities in the range of 0.02–0.1 $\mu$m for sinusoidal hair-saline relative motions. The response to a weak mechanical stimulus (0.147-$\mu$m peak amplitude) is illustrated in the power spectrum of Fig. 1*C* by a small peak at the stimulus frequency (10 Hz) that corresponds to an SNR of 1.4. Illumination of the CPR substantially increases its sensitivity to hydrodynamic stimulation, as illustrated in Fig. 1*D*, where the peak SNR response at constant stimulus intensity increases to 9.7, nearly seven times greater in light than in the dark (cf. Fig. 1, *C* with *D*, arrows). The time course of the CPR response is illustrated in Fig. 3 (thin trace) showing an initial peak discharge rate of $\leq$40 spikes/s and a subsequent plateau rate that stabilizes after ~2 min at high light intensity.

The time course of the instantaneous firing rate is similar to the SNR (Fig. 3, bold trace) and suggests further that the increase in transmission efficiency is mediated by light. CPR activity, indicated by the MDR plateau as a function of light intensity, is illustrated in Fig. 4 (dashed curves). Over the intensity range used in these experiments, the increments in discharge rate, i.e., the slopes of the curves in Fig. 4, were low in dim light but increased more steeply at higher intensities.

A systematic exploration of the effect of light on the SNR and MDR is shown in Figs. 4–6. In general, stimuli of increasing light intensity, with hydrodynamic stimulation held constant, produced increases in both the peak response in the power spectrum and the MDR. These increases are nonlinear, as demonstrated by the SNR and MDR curves. The responses of two CPR interneurons are shown in Fig. 4*A* and they illustrate some variability at saturating light levels. In each experiment, the mechanical stimulus frequency and amplitude was held constant (0.26 $\mu$m for circles and 0.78 $\mu$m for squares). With increasing intensity of illu-

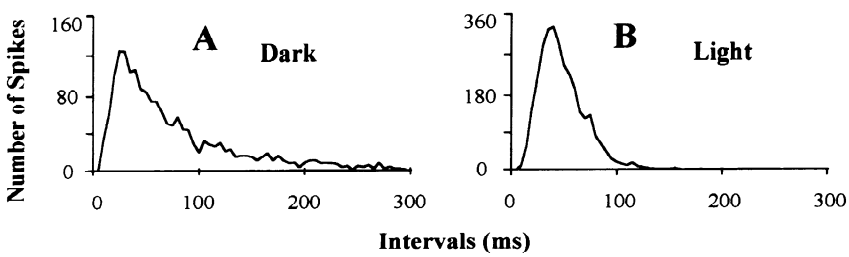


FIG. 2. Spike interval histograms from a CPR cell measured in dark (background light = 0.1 nW/mm$^2$, *A*) and in light (1.1 $\mu$W/mm$^2$, *B*). Data were measured in absence of mechanical stimulation.

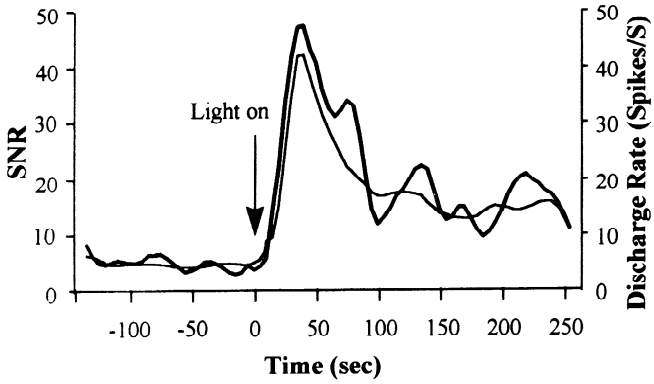


FIG. 3. Time course of CPR response to light (0.1 $\mu$W/mm$^2$), with bold trace representing the SNR (*left* ordinate), narrow trace instantaneous discharge rate (*right* ordinate). Hydrodynamic sinusoidal stimulation was applied continuously at 10 Hz and 0.26 $\mu$m peak displacement amplitude. Light was switched on at arrow ($t$ = 0). Each curve is an average of 3 light responses with an intervening period of dark adaptation (3 min).

mination, the SNR either increased monotonously, with a characteristic sigmoid shape (Fig. 4*A*, solid line, circles), or passed through a maximum (Fig. 4*A*, solid line, squares). The increase in SNR was small at low light levels $\leq$0.0011 $\mu$W/mm$^2$ (3 ND). A large increase in SNR usually occurred at intensities between 0.011 and 0.11$\mu$W/mm$^2$ (2 ND and 1 ND, Fig. 4). At higher intensities, the SNR began to saturate. In 7 of 20 CPRs, the SNRs became fully saturated or had passed through a maximum by the highest light intensity (e.g., Fig. 4*A*, solid line, squares).

In contrast, the MDRs for all CPRs increased monotonously with increasing light intensity (Fig. 4). At low light levels, the increases were small. However, discharge rates climbed rapidly for light levels >0.011 $\mu$W/mm$^2$. In contrast to the SNRs, mean discharge rates (dashed lines) increased throughout the range of light intensities used in these experiments.

The greatest difference between the MDRs and SNRs was evident with respect to mechanosensory stimulation. The stand-alone symbols on the right (Fig. 4*A*) are MDRs obtained at maximum light intensity (0 ND) in the absence of mechanosensory input (amplitude at 0 $\mu$m). Within the limits of statistical precision, these MDRs (open circle and square) are indistinguishable from those at a corresponding level of illumination during mechanical stimulation, an effect discussed further below. The SNRs, on the other hand, drop to 0 without mechanical stimulation.

Figure 4*B* shows the means and standard deviations of the SNRs and MDRs for 14 CPRs. Data for the remaining six CPRs, though consistent with the results shown here, were obtained at different light intensities and therefore were not included in this figure. Individual response curves were normalized for each cell so that each point is the mean relative response at each light intensity. The mean SNRs, like those illustrated in Fig. 4*A*, show large increases at light levels >0.011 $\mu$W/mm$^2$ (2 ND), although they begin to saturate at the highest light intensity. Similarly, MDRs increase rapidly for intensities $\geq$0.011 $\mu$W/mm$^2$, whereas smaller increases occur at lower intensities. The spike rate during the experiments was clearly elevated at 2 ND light levels, indicating a response threshold for light at or near the 0.011 $\mu$W/mm$^2$ intensity level. Unlike SNR values, which have a sigmoid relationship with light intensity, the MDRs for intensities $\leq$1.1 $\mu$W/mm$^2$ are nonsaturating and show a fairly linear rate of increase in mean tonic discharge.

These results demonstrate that light profoundly affects the mechanosensory response of the CPR in addition to its well-established photic behavior. How does light change the CPR stimulus threshold? To investigate this question, we have examined the influence of the light on the stimulus intensity-response functions of the CPRs. Responses from two cells shown in Fig. 5, *A* and *B*, illustrate the light effect. The SNR and MDR curves associated with moderate levels of illumination (circles) both lie entirely above those measured in the dark (squares). Again, the familiar sigmoid form was

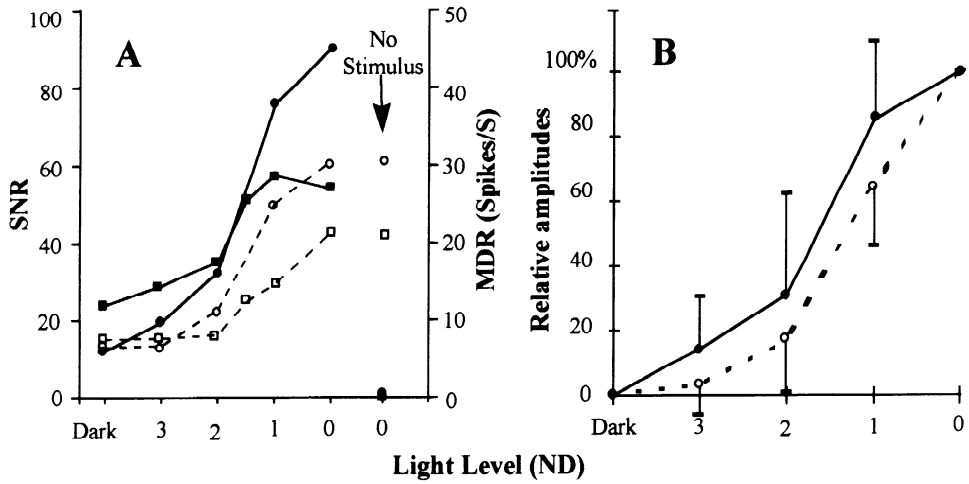


FIG. 4. Efficiency of signal transmission in CPR interneurons at different light levels. *A*: signal-to-noise ratios (SNRs, ——) and mean discharge rates (MDRs, – – –) for 2 CPRs (○, ● and □, ■). Peak stimulus displacement at 10 Hz was 0.26 and 0.78 $\mu$m, respectively, for each cell (●, ○ and ■, □). Light intensity was adjusted by neutral density filters with number of log units of attenuation shown on abscissa. Maximum light intensity (0 ND) was 1.1 $\mu$W/ mm$^2$. SNR is indicated on *left* ordinate and MDR on *right*. Stand-alone symbols at *right* represent MDRs (○ and □) and SNRs (● and ■, horizontal axis) measured at maximum light intensity and 0 stimulus displacement. Thus hydrodynamic stimulus has little or no effect on MDR, but SNR is a sensitive measure of evoked response. *B*: means and standard deviations of SNR (——) and MDR (– – –) for CPR interneurons ($n$ = 14) as a function of light intensity. Mechanical stimulus is same as in Fig. 1*C*.

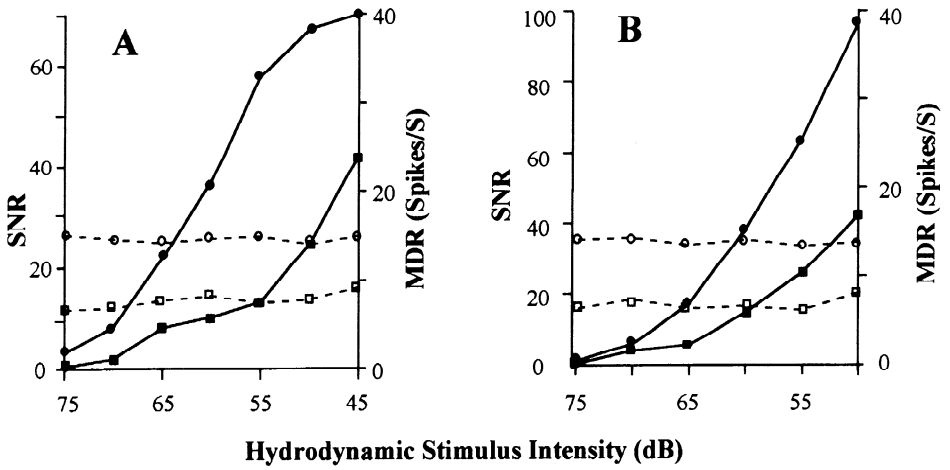


FIG. 5. Stimulus intensity response functions for 2 CPR cells (*A* and *B*) measured in dark and at moderate light intensity (0.1 $\mu$W/mm$^2$, □, ■ and ○, ●). Hydrodynamic stimulus intensity (abscissa) at 10 Hz was adjusted by step attenuators and varied from 0.78 $\mu$m (45 dB) to 0.03 $\mu$m (75 dB). Solid lines indicate SNR (*left* ordinate), as determined from power spectra, whereas broken lines represent MDRs (*right* ordinate).

observed in the light-enhanced SNR response. Threshold mechanosensitivity based on the SNR measurements can be defined as any response clearly >0, i.e., below which no peak can be discerned in the power spectrum. Based on this threshold criterion, it can be seen in Fig. 5*A* that the CPR responds to the weakest stimulus (−75 dB), albeit weakly (circles), only when illuminated. For weak stimuli, as illustrated in this cell, threshold is lowered ~5 dB; for slightly suprathreshold stimuli, sensitivity increases from −65 dB in the dark to −70 dB in the light (e.g., at SNR 9). Similarly, threshold sensitivity for the cell illustrated in Fig. 5*B* increased from −65 to −70 dB at an SNR of ~5. Illumination of the CPR increased sensitivity not only at or near threshold, but resulted in steeper SNR curves and >10 dB increases in sensitivity at higher stimulus intensities (cf. curves where SNR ≥ 15, Fig. 5, *A* and *B*), indicating an increase in transmission efficiency with light.

In neurons, a reduction in threshold implies an increase in sensitivity to both signal and noise components, assuming that both converge at the level of the membrane potential. As shown consistently above, our experimental results show an increase in the SNR with increasing light intensity. It is clear that this enhancement is due to a nonlinear process, because linear increases in sensitivity would amplify both noise and signal equally with the result that no change in the SNR would be indicated. Thus the effects of light are more complex. On the one hand, light generates a monotonous increase in the MDR (Fig. 4*B*) while at the same time generating an even larger light-induced amplification of the coherent signal. Although both the SNR and the MDR increase strongly with increasing light intensity (Fig. 4), their behavior was quite different with regard to hydrodynamic stimulus intensity. For low-amplitude hydrodynamic stimuli (near threshold), the MDRs vary only little over a 30-dB range of stimulus intensity as shown by representative results in Fig. 5, dashed lines. We conclude that the MDR by itself is not an accurate measure of signal transmission at or near threshold stimulus intensities and that the SNR more accurately characterizes the mechanosensory transmission properties of the CPR interneurons.

Previously it has been shown that strong mechanical stimuli can suppress rates of spike discharge in the CPR (Galeano and Beliveau 1973). However, we have never observed suppression at the low intensities of hydrodynamic stimulation used here. Instead, weak stimuli cause discharge patterns to become more regular and synchronous with periodic stimulation. At somewhat greater stimulus amplitudes, slight increases in MDR were observed, but these increases were not significantly greater than fluctuations due to the cell's random discharge (Fig. 6*A*, dashed lines). These results are consistent with earlier CPR studies (Kennedy 1963) where it was shown that low-intensity mechanosensory stimulation was excitatory whereas strong stimulation was predominantly inhibitory and mediated polysynaptically. The growth of synchronization with increasing stimulus intensity is evident in spike interval histograms and is consistent with earlier notions of noise-mediated signal transmission efficiency in excitable systems (Longtin 1993; Longtin et al. 1991, 1994).

In addition, Fig. 6 illustrates the interaction of the intensity (*A*) and frequency (*B*) parameters of the stimulus. For mechanosensory stimuli of moderate intensity (e.g., equivalent to points midway along the sigmoid curve in Fig. 5*A*), there was a parallel increase in MDR at both stimulus intensities over the full range of light stimulation (dashed lines). Similarly, changes in stimulus frequency (at constant velocity) had only minor effects on the MDRs (Fig. 6*B*, dashed lines). In contrast, the SNRs increased dramatically at a lower stimulus frequency (3 Hz) consistent with the known low-frequency tuning characteristics of the CPR. Again, the SNR curves were approximately parallel to each other over the range of light intensities tested. Below saturation levels, the light effect on the SNRs and the MDRs was similar to the results shown in Figs. 1 and 4. The increase of the MDR was monotonous (although there was a tendency toward eventual saturation) and was independent of the frequency or the amplitude of the mechanical stimulus while the SNRs saturated and eventually decreased at high light intensities.

Enhancement of hydrodynamic signaling by the CPR cells was further demonstrated by cross-correlation analysis of CPR output relative to activity in presynaptic sensory fibers. Data representative of results from four experiments is illustrated in Fig. 7. In the absence of a mechanical stimulus, there was a correlation peak with an 8- to 20-ms offset, indicating that inputs from spontaneous presynaptic fibers yielded postsynaptic potentials that were transferred to the output at a certain probability value. Illuminating the cell increased the probability of signal transmission by 30% for

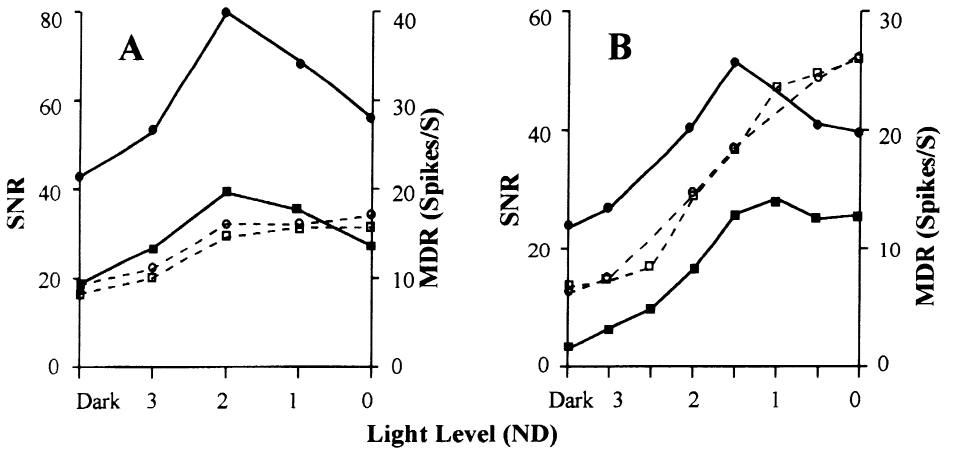


FIG. 6. Influence of light on signal transmission in CPR interneurons at different intensities and frequencies of hydrodynamic stimulation. Light intensity was adjusted with neutral density filters as indicated on the abscissas. *A*: CPR responses at stimulus amplitudes of 0.26 $\mu$m (■, □) and 0.42 $\mu$m (●, ○) at 10 Hz. Solid lines are SNRs (*left* ordinate), broken lines are MDRs (*right* ordinate). *B*: CPR responses at stimulus frequencies of 10 Hz (■, □) and 3 Hz (●, ○), with corresponding displacement amplitudes of 0.08 and 0.26 $\mu$m (constant velocity), respectively.

this particular input (cf. thin and bold traces in Fig. 7*A*). With mechanical stimulation, the correlogram shows a periodic modulation resulting from synchronization with the stimulus (Fig. 7*B*). The signal transfer efficiency was again higher in the light than in the dark (thin vs. bold traces). Also, a slight phase advance was observed.

These experiments demonstrate that the crayfish CPRs, cells embedded in the sixth abdominal ganglion, exhibit increased efficiency in signal transmission to hydrodynamic stimulation of the tailfan mechanoreceptors when stimulated by light. This was accompanied by a lowering of the threshold to hydrodynamic stimuli by ~5 dB. Moreover, we observed that elevated light intensities increased both the SNR and the MDR; however, only the SNR increased with stimulus intensity. In the next section, we propose three phenomenological mechanisms to account, qualitatively, for these results along with demonstrations of their behavior using the electronic Fitzhugh-Nagumo (FN) neuron model.

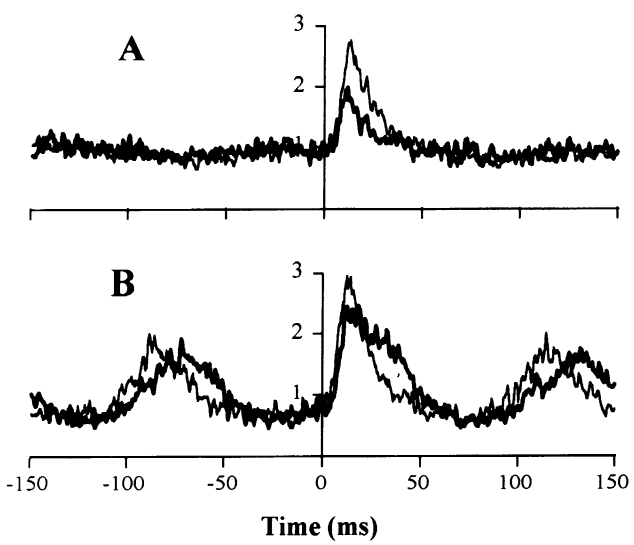


FIG. 7. Cross-correlation analysis of activity from CPR and a presynaptic mechanoreceptor afferent from ipsilateral root 2. *A*: correlogram based on spontaneous activity with light (narrow trace, 0.1 $\mu$W/mm$^2$) and without light (bold trace, 0.1 nW/mm$^2$). *B*: correlogram measured during hydrodynamic stimulation (10-Hz, 0.42-$\mu$m peak displacement amplitude), with (narrow trace) and without light (bold trace). Curves are normalized to mean level of correlogram.

## DISCUSSION

Sensitivity to an external coherent signal is a critical feature for the sensory nervous system. Light enhancement of signal transmission in sensory integration by an increase in SNR illustrates an integrative mechanism which can account for the regulation or improvement in threshold sensitivity. By another mechanism, Manra et al. (1993) report that an electrical coupling between mechanoreceptor afferents facilitates signal transmission and suggest that this would enhance motoneuron input to the proximal leg muscles controlling locomotion in the crayfish. Furthermore, it has been shown that internal (neuronal) noise plays an important role in signal transmission, as demonstrated recently by the electrosensitivity in sharks (Braun et al. 1994) and mechanosensitivity in crickets (J. Levin and J. P. Miller, personal communication). Much earlier it was suggested that random spike activity (system noise) enhanced signal transmission in single neurons (Knight 1972b; Moore et al. 1966), in coupled networks (Buhman and Schulten 1987), and in populations of neurons (Knight 1972a). We show in this report that signal transmission is influenced by light in a unique, bimodal crayfish CPR sensory neuron. Apart from its role in spatial vision, light is an environmental signal that changes in intensity with time and location of the animal and is therefore of fundamental importance for nervous function. We have shown in the crayfish extraretinal CPRs that light plays a role in the integration of mechanosensory information by the central nervous system by regulating the efficiency of signal transmission. We now discuss possible mechanisms by which these results may be understood.

### *Mechanisms underlying light-mediated increases in mechanosensitivity*

There are several mechanisms for regulating of signal transmission by light. First, we can rule out a direct effect of light on mechanoreceptor transduction. We have made simultaneous SNR measurements (not reported here) from both the sensory afferents and the CPR that show no measurable effects of light on sensory transduction in the periphery. Therefore the light regulation of signal transmission occurs at either pre- or postsynaptic sites within the sixth ganglion. We examine three distinct phenomenological mechanisms

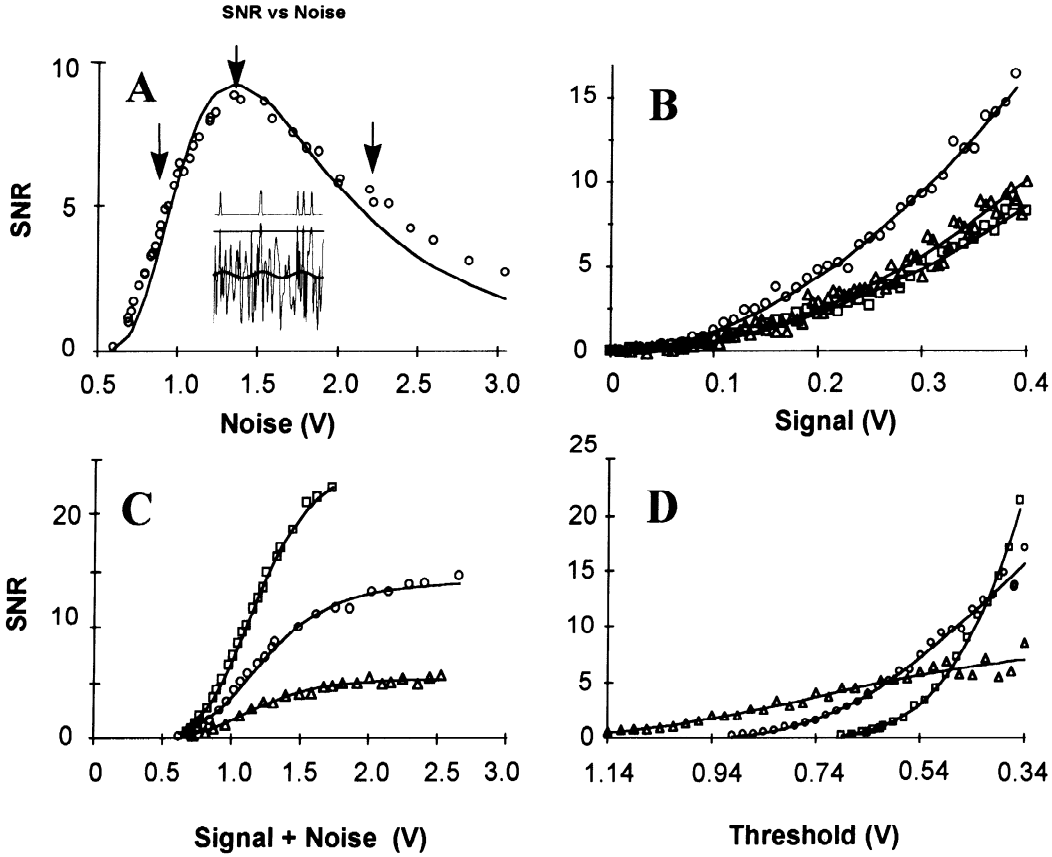


FIG. 8. Results of simulations with an analogue electronic Fitzhugh-Nagumo model neuron. Threshold for firing was set to 0.5 V in all simulations except that shown in *D*. Signal intensity was set at 0.145 V peak (subthreshold) for simulations in *A* and *D*. Signal frequency was 20 Hz for all simulations. Noise was a band limited (3 kHz) white noise. *A*: SNR vs. noise intensity in root mean square V (abscissa). Arrows identify an optimal noise intensity (1.35 V), corresponding to maximum SNR, a smaller than optimal intensity (0.9 V) and a larger than optimal intensity (2.2 V), which values are used in subsequent simulations. *B*: SNR vs. signal intensity for 3 noise intensities: optimal (○); smaller (□); and larger (△); showing a lowering of detection threshold for optimal noise. *C*: SNR vs. sum of signal plus noise multiplied by a gain factor. Abscissa represents root mean squares of sum voltages. No response is measurable until sum exceeds threshold at 0.5 V. *D*: SNR vs. threshold for a fixed signal amplitude (0.1 V) at 3 noise intensities: optimal (○); smaller (□); and larger (△). In all simulations, solid curves represent results of a simple, threshold theory of stochastic resonance details of which are reported elsewhere (see text).

for the action of light in regulating the integration of hydrodynamic signals by the CPR. Common to these are the assumptions that the activity of the CPR can be characterized by a simple firing threshold and that sub- and suprathreshold stimulus-induced coherent signals are additive with noise, as shown schematically in Fig. 8*A* (*inset*). A spike is generated when the sum of signal and noise exceeds the threshold.

The first mechanism assumes that light affects noise within the CPR itself, e.g., CPR ion channel activity, and that the noise adds to the coherent external signal, which in these experiments remains constant. As a result of the increase in light intensity, the integration of mechanosensory synaptic input is modulated, consistent with the emerging concept of stochastic resonance (Douglass et al. 1993; Moss et al. 1994; Wiesenfeld and Moss 1995), i.e., with a resultant increase in threshold crossing rates and enhancement of the SNR. That the addition of noise to the subthreshold signal can improve the efficiency of signal transmission is based on the assumption that for a fixed firing threshold, the probability for subthreshold signals to trigger a spike is 0 in the absence of noise (no threshold crossings). However, the addition of noise increases the probability of threshold crossing events and hence, the generation of spikes. What is not as obvious, but which has been demonstrated frequently in physical systems, is an enhanced degree of coherence between a noise-evoked spike train and a subthreshold signal as the noise intensity increases. This occurs because the most probable position for a threshold event coincides with the peak of the signal, in this case peak depolarization of excitatory postsynaptic potentials evoked by the mechanoreceptor afferents. Thus for periodic signals, a peak will appear in the power spectrum at the signal frequency (e.g., see Fig. 1*C*).

The second mechanism proposes that increasing light intensity increases the gain of a putative amplifier within the CPR and/or its affiliated neuronal circuits. Furthermore, it is proposed that noise originates at the level of the mechanoreceptors in the periphery so that an increase in light intensity enlarges the signal and noise proportionately. For weak stimuli, input signals are embedded in ongoing postsynaptic events, and mostly subthreshold, and do not evoke a postsynaptic (CPR) response. Changing the gain of the system amplifies the potentials together with noise, bringing them closer to or further away from the firing threshold. We as-

sume, as before, that each threshold event results in a detectable neural response. The spike train thus would be a combination of random and coherent events. The power spectrum, again, would be a convenient measure of the coherence relative to noise.

The third mechanism proposes that light merely depolarizes the cell, with the signal and underlying noise remaining constant. Membrane depolarization, or an equivalent decrease in the firing threshold, clearly will increase the probability that a postsynaptic potential exceeds the spike threshold. Indeed, light triggers a long-lasting membrane depolarization (Wilkens and Larimer 1972).

In tests of the three proposed mechanisms, the neural response was simulated with an electronic Fitzhugh-Nagumo model, which could be driven by a periodic signal in combination with noise. The model was operated in a subthreshold mode for the periodic signal; its dynamic operation is illustrated by Fig. 8*A, inset.* The device and its operation have been described in detail elsewhere (Moss et al. 1993). Four experiments were performed, the results of which are illustrated in Fig. 8. In the first simulation (to test the 1st mechanism), a subthreshold periodic signal was applied with a variable noise signal. For each noise intensity, we measured the SNR in the standard way, as previously described. The results, measured on the Fitzhugh-Nagumo circuit, are shown in Fig. 8*A* (open circles), where it is clear that the SNR passes through a well-defined maximum at an optimal noise intensity. This demonstrates the phenomenon of stochastic resonance. The solid curve is taken from a simple threshold theory of stochastic resonance (Gingl et al. 1995; Moss et al. 1994). Although the theory and the simulation do not correspond precisely, the main features of the behavior of the SNR versus noise intensity are captured by the theory. The details of the theoretical description of this simulation will be published elsewhere (Pei et al. 1995a). The arrows in Fig. 8*A* identify three noise intensities: small (0.9 V), optimal (1.35 V), and large (2.2 V), respectively, which will be used in the subsequent simulations.

The second simulation (to test the 1st mechanism) is designed to measure the signal-detection threshold of the device. In this case, we kept the noise intensity constant at each of the three values delineated by the arrows in Fig. 8*A*. The SNR was measured relative to the signal amplitude varied from 0 to an arbitrary value. For each noise intensity, the SNR increased with signal intensity approximately as a square law, as illustrated in Fig. 8*B*. The square law behavior is consistent with the stochastic resonance theory as shown by the solid curves. It is notable that the optimum noise intensity, determined empirically in the first stimulation, also generates the highest SNRs (Fig. 8*B*, ○). Thus the FN model exhibits lowest threshold sensitivity at the optimal noise intensity predicted by SR theory. In contrast, both lower (□) and higher (△) noise levels result in markedly higher threshold values.

The third simulation is to test the second mechanism whereby both signal and noise would be amplified by the same factor. In this case, light would increase the gain of an amplifier whose input was the sum of the signal and noise. This simulation was repeated for the same three noise intensities, but in this experiment noise was added to the standard signal level (0.1 V peak). The sum of the signal and noise then was amplified by a factor that varied between 0 and 2.5, with the resulting voltages represented on the abscissa (Fig. 8*C*). SNR again increased with input intensity; the square, circle, and triangle symbols correspond to the small, optimal, and large noise intensities previously identified. In each of these simulations, lower noise levels result in larger SNRs and visa versa, as results expected for a conventional linear system. Thus increasing noise serves only to elevate the detection threshold for this application of the model.

The final simulation is to test the effect of varying the firing threshold (the 3rd mechanism) while maintaining the signal and noise intensities constant. The results shown in Fig. 8*D* depend much on which intensity of noise was added to the signal. Threshold sensitivity, as interpreted from the SNR versus firing-threshold results, is a complex relationship as indicated by the intersecting curves. For large firing thresholds (>0.64 V), the largest noise level (△) results in the lowest detection threshold, but the SNR for this level of noise is also small over much of the firing-threshold range; the smallest noise level (□) results in the greatest detection threshold, although this behavior is reversed for low firing thresholds (<0.44 V).

The simulated results from the Fitzhugh-Nagumo model can be compared to the physiological data in the following ways. Recall that the central question concerns the mechanistic action of light. For the first and second simulations, we assume that an increase in light intensity increases only the noise. The effect of noise on signal transmission, as indicated by SNR values, is therefore comparable for both the simulated and physiological results (cf., Figs. 8*A* and 4*A*), with light substituting for noise. In contrast, neither an increase in gain (system amplification) nor a change in firing threshold yields results consistent with the physiological data. In particular, an increase in signal plus noise results in SNR curves which seem to saturate without passing through a maximum (Fig. 8*C*), a result not seen in the data presented in Fig. 4*A* or Fig. 6. Moreover, there is no evidence for an optimum noise level; all noise intensities degrade the signal-detection efficiency for this mechanism. Likewise, changing the threshold does not yield results comparable with the physiological data, e.g., the curves for the selected noise levels intersect inexplicably (Fig. 8*D*), in contrast to the essentially parallel behavior of the CPR at different light levels (Fig. 6). We conclude that the first mechanism is consistent with both the physiological data and the Fitzhugh-Nagumo model simulation. This mechanism is consistent with the observation that, in the absence of stimulation, the light-evoked firing intervals of the CPR are characterized by a random distribution and that the mean discharge rate increases with light intensity (dashed lines, Fig. 5). Given that light triggers a long-lasting membrane depolarization and perturbation in CPR activity, both noise and threshold changes could be involved in the light enhancement of signal transmission.

### *SNR versus MDR*

In our analyses, the signal-to-noise ratio is considered to be a more sensitive measure of signal transmission than either mean discharge rate or time-interval histograms of spike activity. The instantaneous MDR is used widely to describe physiological responses, especially to constant or

slowly changing stimuli to which the response is indicated by a change in the instantaneous firing rate. This measure is not well suited, however, for detecting responses to near-threshold stimulus intensities in a system with relatively high levels of spontaneous activity (high MDRs). Under these circumstances, a response may be too small to be detected within the background fluctuations, as is the case for the CPR interneurons. In contrast the SNR, as calculated from the power spectrum, is an extremely sensitive measure of coherence for evaluating stimulus-response characteristics. The response is described by the power at the stimulus frequency and, by comparing the signal with the broad-band noise background, the signal transmission efficiency can be measured accurately.

### *Excitation and inhibition*

It is known that the crayfish CPRs receive both excitatory and inhibitory synaptic inputs in response to mechanosensory stimuli (Kennedy 1963; Wilkens and Larimer 1972). Galeano and Beliveau (1973) report weak, tonic inhibition from ipsilateral and contralateral mechanoreceptors, whereas Flood and Wilkens (1978) have shown that ipsilateral excitatory inputs are strong for nerve roots 2–5. In addition, the CPRs receive substantial inhibitory input from neighboring CNS interneurons (Marzelli and Wilkens 1979). However, in the data presented here, no inhibitory effects, such as a reduction in MDR, have been observed. In fact, an increase in intensity of the hydrodynamic stimulus typically had little effect on the MDR; changes, if any, were within the range of random fluctuations. Furthermore, in the cross-correlogram (Fig. 7), no inhibitory valley was evident after the excitatory peak. One reason might be that our cross-correlogram was not sensitive enough to detect low-level inhibition. However, a more likely explanation is that the intensity of the hydrodynamic stimuli used in these experiments was below threshold for activation of inhibitory pathways. The amplitude of the sinusoidal displacements was only in the range 0.05–1.4 $\mu$m peak amplitude, a very small vibration. Thus our results are consistent with the observation by Kennedy (1963) that for a small stimulus, the CPR received exclusively excitatory input directly from the mechanoreceptor efferents, whereas a stronger stimulus produces inhibition with longer latencies. In many of these early studies, the hairs were stimulated by direct mechanical contact. This technique necessarily resulted in much stronger stimuli than used in our experiment, with the result that large synchronized discharges and/or inhibition of the CPR was the result.

### *Functional significance of abdominal photosensitivity*

Crayfish live in dark habitats. Emergence from burrows or other protective habitat during the day increases their exposure to predators. Our results, showing enhanced mechanosensitivity of the CPR as a function of light, are consistent with the hypothesis that abdominal photosensitivity is an adaptive mechanism important for the survival of the animal in a predator rich environment (reviewed by Wilkens 1988). Evidence for this hypothesis includes a demonstrated link between photonegative behavior and a functionally intact sixth ganglion (containing the CPR) (Welsh 1934), the initiation of backward walking in response to strong illumination of the tail (Edwards 1984) and/or direct high-frequency stimulation of the CPRs (Simon and Edwards 1990), and CPR sensitivity to mechanosensory inputs from the tailfan receptors. Therefore it is inviting to suggest that CPR-mediated abdominal photosensitivity exerts a controlling influence on threshold mechanosensitivity of the animal by regulating signal transmission efficiency. It is essential for crayfish to be able to detect the weak vibrations produced by fish (Breithaupt et al. 1995; Tautz 1990a,b) or other predators. Therefore it would seem to be important for crayfish to increase their alertness, including the sensitivity of the predator-detection apparatus, when they venture into an illuminated part of the environment. Also, it is interesting to speculate that light may indirectly influence threshold sensitivity for the tailflip escape reflex. For crayfish hidden in a burrow or other protective habitat, a reduced mechanosensitivity in the absence of light would decrease the likelihood for inappropriate locomotory or escape behaviors.

In summary, we have observed that the sensitivity of the crayfish to hydrodynamic vibrations is regulated by illumination of the caudal ganglion and CPR interneurons. We have proposed a phenomenological mechanism for the action of light which, when tested with the Fitzhugh-Nagumo neuron model, was shown to be consistent with the physiological data. Finally, we have offered a plausible survival function associated with the ability of the CPR to regulate hydrodynamic sensitivity.

We thank K. Bachmann for assistance on performing the Fitzhugh-Nagumo simulations and Dr. B. Schmitz for insights and assistance during early experiments.

This work was supported by U.S. Office of Naval Research Grant N00014-90-J-1327.

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Received 28 September 1995; accepted in final form 28 June 1996.

## REFERENCES

BARLOW, R. B. What the brain tells the eye. *Sci. Am.* 262: 90–95, 1990.

BARLOW, R. B., BIRGE, R. R., KAPLAN, E., AND TALLENT, J. R. On the molecular origin of photoreceptor noise. *Nature Lond.* 366: 64–66, 1993.

BEZRUKOV, S. M. AND VODYANOY, I. Noise-induced enhancement of signal transduction across voltage-dependent channels. *Nature Lond.* 378: 362–364, 1995.

BRAUN, H. A., WISSING, H., SCHÄFER, K., AND HIRSCH, M. C. Oscillation and noise determine signal transduction in shark multimodal sensory cells. *Nature Lond.* 367: 270–273, 1994.

BREITHAUPT, T., SCHMITZ, B., AND TAUTZ, J. Hydrodynamic orientation of crayfish (*Procambarus clarkii*) to swimming fish prey. *J. Comp. Physiol. A Sens. Neural Behav. Physiol.* 177: 481–491, 1995.

BUHMAN, J. AND SCHULTEN, K. Influence of noise on the function of a "physiological" neural network. *Biol. Cybern.* 56: 313–327, 1987.

COLLINS, J. J., CHOW, C. C., AND IMHOFF, T. T. Stochastic resonance without tuning. *Nature Lond.* 376: 236–238, 1995.

CRONER, L. J., PURPURA, K., AND KAPLAN, E. Response variability in retinal ganglion cells of primates. *Proc. Natl. Acad. Sci. USA* 90: 8128–8130, 1993.

DEFELICE, L. J. *Introduction to Membrane Noise.* New York: Plenum, 1981, p. 500.

DOUGLASS, J. K. AND WILKENS, L. A. Directional sensitivities of long feathered hair mechanoreceptors in the crayfish tailfan (Abstract). *Am. Zool.* 31: 33A, 1991.

DOUGLASS, J. K., WILKENS, L., PANTAZELOU, E., AND MOSS, F. Noise enhancement of information transfer in crayfish mechanoreceptors by stochastic resonance. *Nature Lond.* 365: 337–340, 1993.

EDWARDS, D. H. Crayfish extraretinal photoreception. I. Behavioral and

motoneuronal responses to abdominal illumination. *J. Exp. Biol.* 109: 291–306, 1984.

FERSTER, D. AND SPRUSTON, N. Cracking the neural code. *Science Wash. DC* 270: 756–757, 1995.

FLOOD, P. M. AND WILKENS, L. A. Directional sensitivity in a crayfish mechanoreceptive interneuron: analysis by root ablation. *J. Exp. Biol.* 77: 89–106, 1978.

GALEANO, C. AND BELIVEAU, S. Mechanoreceptor and photoreceptor tonic integration in the Crayfish. *Can. J. Physiol. Pharmacol.* 51: 949–958, 1973.

GERSTEIN, G. AND MANDELBROT, B. Random walk models for the spike activity of a single neuron. *Biophys. J.* 4: 41–68, 1964.

GINGL, Z., KISS, L. B., AND MOSS, F. Non-dynamical stochastic resonance theory and experiments with white and arbitrarily colored noise. *Europhys. Lett.* 29: 191–196, 1995.

HERMANN, H. T. AND OLSEN, R. E. Dynamic statistics of crayfish caudal photoreceptors. *Biophys. J.* 7: 279–296, 1967.

HUNER, J. V. AND BARR, J. E. Red swamp crawfish: biology and exploitation. Louisiana Sea Grant College Program, Louisiana State University, Baton Rouge. 1984.

JUNG, P., BEHN, U., PANTAZELOU, E., AND MOSS, F. Collective response in globally coupled bistable systems. *Physiol. Rev. A* 46: R1709–R1712, 1992.

JUNG, P. AND MEYER-KRESS, G. Spatiotemporal stochastic resonance in excitable media. *Physiol. Rev. Lett.* 74: 2130–2133, 1995.

KALMIJN, A. J. Hydrodynamic and acoustic field detection. In: *Sensory Biology of Aquatic Animals,* edited by J. Atema, R. R. Fay, A. N. Popper, and W. N. Tavolga. New York: Springer-Verlag, 1988, p. 82–130.

KENNEDY, D. Physiology of photoreceptor neurons in the abdominal nerve cord of the crayfish. *J. Gen. Physiol.* 46: 551–572, 1963.

KNIGHT, B. W. Dynamics of encoding in a population of neurons. *J. Gen. Physiol.* 59: 734–766, 1972a.

KNIGHT, B. W. The relationship between the firing rate of a single neuron and the level of activity in a population of neurons. *J. Gen. Physiol.* 59: 767–778, 1972b.

LARIMER, J. L. The presence of a functional caudal photoreceptor in blind cavernicolous crayfish. *Nature Lond.* 210: 204–205, 1966.

LEVIN, J. E. AND MILLER, J. P. Broadband neural encoding in the cricket cercal sensory system enhanced by stochastic resonance. *Nature Lond.* 380: 165–168, 1996.

LINDNER, J. F., MEADOWS, B. K., DITTO, W. L., INCHIOSA, M. E., AND BULSARA, A. R. Array enhanced stochastic resonance and spatiotemporal synchronization. *Physiol. Rev. Lett.* 75: 3–6, 1995.

LONGTIN, A. Stochastic resonance in neuron models. *J. Stat. Phys.* 70: 309–328, 1993.

LONGTIN, A., BULSARA, A., PIERSON, D., AND MOSS, F. Bistability and the dynamics of periodically forced sensory neurons. *Biol. Cybern.* 70: 569–578, 1994.

LONGTIN, A., BULSARA, B., AND MOSS, F. Time-interval sequences in bistable systems and the noise-induced transmission of information by sensory neurons. *Physiol. Rev. Lett.* 67: 656–659, 1991.

MANRA, A. E., CATTAERT, D., WALLEN, P., DICAPRIO, R. A., AND CLARAC, F. Electrical coupling of mechanoreceptor afferents in the crayfish: a possible mechanism of enhancement of sensory signal transmission. *J. Neurophysiol.* 69: 2248–2251, 1993.

MARZELLI, G. A. AND WILKENS, L. A. Centrally-mediated synaptic input: effects on an identified crayfish mechanosensory interneuron. *J. Comp. Physiol.* 134: 1–10, 1979.

MOORE, G. P., PERKEL, D. H., AND SEGUNDO, J. P. Statistical analysis and functional interpretation of neuronal spike data. *Annu. Rev. Physiol.* 28: 493–522, 1966.

MOSS, F. Stochastic resonance: from the ice ages to the monkey's ear. In: *Contemporary Problems in Statistical Physics,* edited by G. H. Weiss. Philadelphia, PA: SIAM, 1994, p. 205–253.

MOSS, F., DOUGLASS, J. K., WILKENS, L., PIERSON, D., AND PANTAZELOU, E. Stochastic resonance in an electronic FitzHugh-Nagumo model. *Proc. NY Acad. Sci.* 706: 26–41, 1993.

MOSS, F., PIERSON, D., AND O'GORMAN, D. Stochastic resonance: tutorial and update. *Int. J. Bifurc. Chaos* 4: 1383–1398, 1994.

PEI, X., BACHMANN, K., AND MOSS, F. The detection threshold, noise and stochastic resonance in the Fitzhugh-Nagumo neuron model. *Physiol. Lett. A* 206: 61–65, 1995a.

PEI, X., WILKENS, L., AND MOSS, F. Light illumination enhances the efficiency of signal transduction to hydrodynamic stimulation in the caudal photoreceptor cells of the crayfish sixth ganglia. *Biophys. J.* 68: 132, 1995b.

PLUMMER, M. R., TAUTZ, J., AND WINE, J. J. Frequency coding of waterborne vibrations by abdominal mechanosensory interneurons in the crayfish, *Procambarus clarkii. J. Comp. Physiol. A Sens. Neural Behav. Physiol.* 158: 751–764, 1986.

PROSSER, C. L. Action potentials in the nervous system of the crayfish. II. Responses to illumination of the eye and caudal ganglion. *J. Cell. Comp. Physiol.* 4: 363–377, 1934.

SCHMITZ, B. Directionality of antennal sweeps elicited by water jet stimulation of the tailfan in the crayfish *Procambarus clarkii. J. Comp. Physiol. A Sens. Neural Behav. Physiol.* 171: 617–627, 1992.

SIMON, T. W. AND EDWARDS, D. H. Light-evoked walking in crayfish: Behavioral and neuronal responses triggered by the caudal photoreceptor. *J. Comp. Physiol. A Sens. Neural Behav. Physiol.* 166: 745–755, 1990.

STARK, L. Transfer function of a photoreceptor ganglion. In: *Neurological Control Systems: Studies in Bioengineering.* New York: Plenum, 1968, p. 4–71.

TAUTZ, J. Coding of mechanical stimuli in crustaceans—what and why? In: *Frontiers in Crustacean Neurobiology,* edited by K. Wiese, W.-D. Krenz, J. Tautz, H. Reichert, and B. Mulloney. Basel: Birkhäuser Verlag, 1990a, p. 200–206.

TAUTZ, J. Can the directional sensitivity of single mechanosensory neurons in arthropods tell the animal anything about stimulus direction. In: *Sensory Systems and Communication in Arthropods,* edited by F. G. Gribakin, K. Wiese, and A. V. Popov. Basel: Birkhäuser Verlag, 1990b, p. 359–363.

TAUTZ, J. AND PLUMMER, M. Comparison of directional selectivity in identified spiking and nonspiking mechanosensory neurons in the crayfish Orconectes limosus. *Proc. Natl. Acad. Sci. USA* 91: 5835–5837, 1994.

VAN HARREVELD, A. A physiological solution for fresh-water crustacea. *Proc. Soc. Exp. Biol.* 34: 428–432, 1936.

VIDYASAGAR, T. R. Interactions between visual and other sensory modalities and their development. *Vision Visual Dysfunct.* 11: 369–385, 1991.

WELSH, J. H. The caudal photoreceptor and responses of crayfish to light. *J. Cell. Comp. Physiol.* 4: 379–388, 1934.

WIERSMA, C. A. G., ROACH, J. L. M., AND GLANTZ, R. M. Neural integration in the optic system. In: *The Biology of Crustacea: Neural Integration and Behavior,* edited by D. C. Sandeman and H. L. Atwood. New York: Academic, 1982, p. 1–31.

WIESENFELD, K. AND MOSS, F. Stochastic resonance and the benefits of noise: from ice ages to crayfish and squids. *Nature Lond.* 373: 33–36, 1995.

WILKENS, L. A. The crayfish caudal photoreceptor: advances and questions after the first half century. *Comp. Biochem. Physiol.* 91: 61–68, 1988.

WILKENS, L. A. AND DOUGLASS, J. K. A stimulus paradigm for analysis of near-field hydrodynamic sensitivity in crustaceans. *J. Exp. Biol.* 189: 263–272, 1994.

WILKENS, L. A. AND LARIMER, J. L. The CNS photoreceptor of crayfish: morphology and synaptic activity. *J. Comp. Physiol.* 80: 389–407, 1972.

WINE, J. J. The structural basis of an innate behavioral pattern. *J. Exp. Biol.* 112: 283–319, 1984.

YOSHIDA, M. Extraocular photoreception. In: *Handbook of Sensory Physiology,* edited by H. Autrum. New York: Springer-Verlag, 1979, p. 581–640.