Impulse Conduction of Olfactory Receptor Neuron Axons

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KEY WORDS olfaction; olfactory bulb; sensory neurons; rat

ABSTRACT Compound action potentials were recorded from rat olfactory receptor neuron axons at measured distances from the stimulation electrode along the lateral surface of the main olfactory bulb. Distances were plotted as a function of the latencies measured from stimulus onset to the prominent negative trough of the triphasic compound action potential. A straight line was fitted to these data to calculate impulse conduction velocity, 0.42 ± 0.01 m/s (n = 25). Two procedures were used to investigate whether those axons that project to caudal regions of the bulb had faster conduction velocities than axons projecting to rostral bulb. First, the stimulating electrode was moved to mid-bulb and the recording electrode was placed on the caudal bulb. Alternatively, axons were stimulated antidromically at the caudal bulb. These two procedures stimulate those axons projecting to caudal bulb and bypass olfactory receptor neuron axons that synapse in the rostral bulb. The mean impulse conduction velocities from these caudal and antidromic recordings were 0.58 ± 0.19 m/s (n = 8) and 0.57 ± 0.19 m/s (n = 9), respectively. Though both of these means are higher than the impulse conduction velocity calculated for stimulation at the rostral bulb, the differences were not statistically significant. *Microsc. Res. Tech. 58:161–167, 2002.*

INTRODUCTION

Olfactory receptor neurons (ORNs) have several properties that make them unusual sensory neurons. First, ORNs express a large number of olfactory receptor proteins, possibly 500 to 1,000 different receptors in rodents (for review see Mombaerts, 1999). During development, ORNs expressing a single receptor project to the main olfactory bulb (MOB) and converge onto one or two glomeruli in each bulb. Secondly, in the adult, ORNs are continuously generated from basal cells, project to the MOB, and reinnervate the appropriate glomerulus. Thirdly, the axons of ORNs are unmyelinated, unbranched, and of small diameter. This latter property was recognized more than 50 years ago following the development of electron microscopy. Since then, scientists have pondered the physiological role of such a large number of very small, slowly conducting axons. In this article, we review measurements of impulse conduction in ORNs and extend this approach to investigate correlations between impulse conduction velocity of ORN axons and the spatial distribution of the glomeruli to which they project in the MOB. We hypothesize that there is a subset of ORN axons with faster conduction velocities that project preferentially to glomeruli in the caudal regions of the MOB.

One of the early investigators interested in ORNs was Herbert Gasser who compared the structure and electrophysiology of unmedullated fibers in skin and olfactory nerves in cat, pig, and pike (Gasser, 1956). In the pig, he determined the ORN axons to be unmyelinated fibers with a diameter between 0.1 and 0.5 μm, and with a mode of less than 0.2 μm. A count of over 14,000 fibers yielded a density of about 10 fibers per μm^2 and a ratio of fibers to bipolar cells in the olfactory epithelium that give rise to these axons of about 1:1.

The pike provided a better animal model for measuring the impulse conduction along ORNs because the olfactory nerve projects from the nasal mucosa to the rostral portion of the MOB in two large bundles of axons over a distance of 10 mm. The pike olfactory nerve preparation was obtained by exposing the lateral aspect of the fish's rostrum, tying off the ends of the nerve bundle, and then severing the nerve distal to the ties. Gasser then placed the free nerve in a nerve chamber filled with Ringer's solution to perform electrophysiological studies; the mean impulse conduction velocity for 6 preparations was 0.2 m/s with a range of 0.18 to 0.22 m/s (Gasser, 1956).

One year later MacLean et al. (1957) published in vivo work on the rabbit, opossum, and monkey. Animals were anesthetized with pentobarbital and the lateral aspects of the olfactory nerve, the MOB, and the pyriform cortex were exposed by removal of the eye and careful removal of the bone of the medial orbit. Insulated stainless steel wires were used for stimulation and recording, with the stimulation electrode placed ~8 mm rostral to the MOB and the recording electrode placed on the MOB itself. The estimated impulse conduction velocity of opossum ORNs was 0.40 m/s (MacLean et al., 1957). Electrophysiological properties of rabbit ORNs were characterized by Iwase et al. (1961) and Nicoll (1972); Nicoll measured the ORN impulse conduction velocity of nerve rootlets at the surface of the MOB to be 0.34 m/s with a range of 0.28 to 0.55 m/s.

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In a study of olfactory bulb potentials induced by electrical stimulation in the frog, Ottoson (1961) reported an average conduction velocity of the action potential of 0.14 m/s with a range from 0.11 to 0.22 m/s with conduction distances from 2 to 6 mm. The absolute refractory period was about 30 ms followed by relative refractory period of about 200 ms. In Ottoson's study, the olfactory bulb was either pinched or removed to avoid interference from bulb potentials.

In 1965, Easton performed in vitro studies on the ORN axon of the garfish (Easton, 1965). The garfish is a superior experimental model for studying the olfactory nerve since its rostrum is very long and segmented, with exposure of the distinct nerve bundles easily accomplished by simply pulling off these rostral segments. Easton was able to extract up to 15 mm of olfactory nerve rostral to the MOB, which he then placed into a nerve perfusion system. Action potentials were recorded at 2 mm average electrode distance intervals and the impulse conduction velocity of the garfish ORN axon was calculated to be 0.10 to 0.20 m/s (Easton, 1965).

In vivo work was performed by Walter Freeman in the early 1970s on the MOB of the cat. Cats were anesthetized with pentobarbital, the lateral MOB was exposed through the medial aspect of the orbit of the eye, and compound action potentials were recorded on the surface of the bulb. Insulated steel wires were used to stimulate and record the action potentials at a constant electrode distance of 2.5 mm. Freeman (1972) determined the impulse conduction velocity of the cat ORN axon to be 0.42 $\pm$ .05 m/s.

Eng and Kocsis (1987) performed experiments on the turtle MOB utilizing an in vitro whole cranium preparation. The bone overlying the forebrain and the meninges were removed, exposing 5 to 7 mm of MOB. Teflon-coated stainless steel wires were used to stimulate the ORN axons and a micropipette electrode was used to record the action potentials. The distances between the stimulation and recording electrodes ranged from 1 to 5 mm and the impulse conduction velocity of the turtle ORN axon was calculated to be 0.16 to 0.18 m/s.

Noticeably absent from this review of conduction velocity measurements are data from rats and mice. The conduction velocity of olfactory nerve fibers in hamster were estimated at 0.26 m/s (Costanzo and O'Connell, 1978). Griff et al. (2000) measured the impulse conduction velocity of ORN axons in the mouse to characterize the recently developed OMP-null mutant (Buiakova et al., 1996). Insulated stainless steel wires were used to stimulate and record at electrode distances between 0.5 and 1.0 mm; the mean impulse conduction velocity of the wildtype mouse ORN was calculated to be 0.40 $\pm$ .09 m/s. At about the same time, the current experiments were undertaken to analyze impulse conduction in the rat.

The recent discovery of odorant receptor proteins located on olfactory vesicle kinocilia membranes of ORNs (Buck and Axel, 1991) has renewed interest in olfactory coding and the possible role(s) ORNs, their projections, and impulse conduction velocity may play in this coding. ORNs expressing a single presumptive receptor protein have been traced from the olfactory epithelium to the MOB where they project to just a few glomeruli (Mombaerts et al., 1996). Foci in the MOB responding to specific odorants have also been visualized with the 2-deoxyglucose technique (e.g. Jourdan et al., 1980; Stewart et al., 1979). However, removal of even large areas of the MOB does not prevent animals from detecting and discriminating specific odorants (Slotnick et al., 1987). Our understanding of the coding of olfactory information is thus still incomplete. The present study assesses the ORN axon conduction velocity in the rat and investigates the possibility of a subset of faster conducting ORNs projecting to caudal MOB. Identification of such a subset of ORNs could provide clues to the temporal/spatial pattern of ORN projections and to mechanisms of olfactory coding.

METHODS

Animal Preparation

Conduction velocity was measured on 15 male and 11 female Sprague-Dawley rats, ages 7 to 14 weeks. All procedures adhered to guidelines established by the American Association for the Accreditation of Laboratory Animal Care and the NIH Guide for the Care and Use of Laboratory Animals, and were approved by the University of Cincinnati Institutional Animal Care and Use Committee. Rats were anesthetized with 4% chloral hydrate (400 mg/ kg, IP) with supplemental injections (8.0 mg/ kg) given every half hour as needed. Rats were placed in a stereotaxic instrument and the lateral MOB surface was exposed through the orbit. The surface of the MOB was kept moist with saline, and body temperature was maintained between 36 and 37°C. Chloral hydrate, kynurenic acid, and cobalt chloride were obtained from Sigma (St. Louis, MO).

Electrodes and Recording

Responses were recorded to both orthodromic and antidromic electrical stimulation. Bipolar stimulation electrodes were made from a pair of 125 μm stainless steel wires insulated except for the cut tips. The recording electrode was a 75 μm stainless steel wire, insulated except for the cut tip. For orthodromic stimulation, the stimulation electrode was positioned rostral to the recording electrode, either at rostral MOB, or midway on the MOB for orthodromic stimulation of only those axons that project to the caudal MOB. For antidromic stimulation, the stimulating electrode was placed at caudal MOB, and the recording electrode positioned rostral to it (see Fig. 1).

Stimuli were constant currents between 10 and 100 μA intensity and 0.03- and 0.10-ms duration (Grass Medical Instruments Model S44). Single stimuli were delivered at 0.5 Hz; paired-pulse stimuli were delivered with interpulse intervals between 2 and 20 ms. Responses were amplified (Dagan Corp., Model 2400), filtered to pass frequencies between 100 and 1,000 Hz, displayed on an oscilloscope (Tektronix Inc., Model 5111), digitized (Cambridge Electronic Design Ltd. [CED], Model 1401), and stored on computer. The typical compound action potential from which conduction velocities were calculated was triphasic with a prominent negative component (Nicoll, 1972). *Spike2* software (CED) was used to calculate the time between the stimulus onset and the trough of this negative component of the compound action potential.

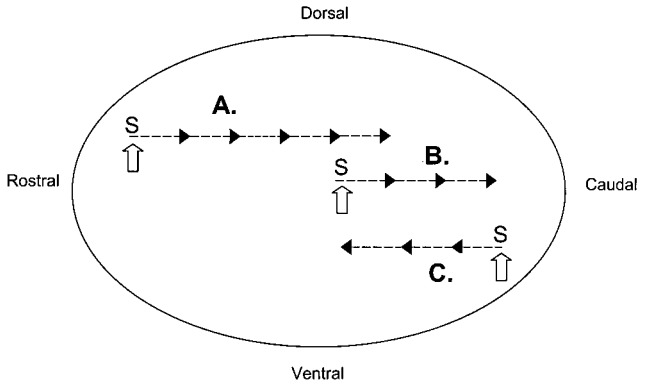

Fig. 1. Schematic of electrode positions on the lateral surface of the MOB. Open arrows show the position of stimulation electrodes (S); filled arrowheads indicate the positions of recording electrodes; the direction of the arrowheads indicates the direction of propagation of the compound action potential. **A,B:** Orthodromic stimulation. **C:** Antidromic stimulation. In A, the stimulation electrode was at the rostral bulb and recordings were made at three to five positions along the horizontal axis across the lateral surface of the MOB. In B, the stimulation electrode was positioned at mid bulb and three recordings were made in the caudal half of the MOB. In C, the stimulation electrode was positioned in the caudal bulb to stimulate ORN axons antidromically; recordings were made at three positions more rostral.

Protocol and Data Analysis

The conduction velocity was calculated from the distance between the stimulation and recording electrodes and the latency between the stimulus onset and the trough of the compound action potential. The recording electrode was first aligned with the anodal pole of the stimulation electrode and then moved to the recording position on the MOB with a translator; the distance moved was the difference between the two positions measured with a vernier (resolution of 0.001 in) converted to millimeters.

For each stimulation/recording configuration (see above), measurements were made for at least 3 distances and at each distance 10 stimuli were presented. The latency for each response was measured, and a mean latency for each distance was calculated. Distance was plotted as a function of mean latency (rather than latency as a function of distance) so that the slope was equal to the conduction velocity. The slope was determined from a linear regression calculated by a weighted method of determinants (Bevington and Robinson, 1992). Differences between conduction velocities obtained using the three stimulation/recording configurations were tested for significance by an analysis of covariance and P values are reported; P values less than 0.01 were considered significant.

RESULTS

Identifying the Compound Action Potential

Electrical stimulation of the ORNs evoked a series of responses recorded from the surface of the MOB. A fast component of the response following the stimulus artifact was triphasic, consisting of a positive, then a negative, and finally another positive deflection. The negative deflection was the most prominent of this triphasic component (see Fig. 2A,B). A slower, negative component followed the triphasic response. To demonstrate that the faster, triphasic component had a presynaptic origin, blockers of synaptic transmission were applied to the surface of the MOB.

Cobalt inhibits post-synaptic responses by competitively blocking calcium influx into the pre-synaptic terminal and preventing vesicular release of neurotransmitters (Weakly, 1973). Figure 2A shows that 10 mM cobalt chloride, applied to the surface of the MOB, greatly reduced the slower, negative component of the response without affecting the faster, triphasic component. When the cobalt chloride was removed by flushing the surface of the MOB with saline, the slower component recovered. This result suggests that the triphasic component has a presynaptic origin, while the slower negative response has a postsynaptic origin.

Higher concentrations of cobalt and/or longer exposure times did decrease the triphasic component; the mechanism is not understood and was not further investigated here. To circumvent this complication, a synaptic transmission inhibitor that acts postsynaptically was used. In an in vitro rat preparation, ORNs were shown to make excitatory, glutamatergic synapses with mitral cells in the glomerular layer of the MOB (Ennis et al., 1996). Figure 2B shows that the glutamate receptor antagonist kynurenic acid (KYN) greatly reduced the slower negative component of the responses, but did not affect the faster, triphasic component. When the KYN was flushed from the surface of the MOB with saline, the slower component recovered. Therefore, the faster, triphasic response was identified as the compound action potential from the ORN axons.

Figure 2C shows responses to paired-pulse stimulation. As the interpulse interval was decreased from 20 to 3 ms, the amplitude of the triphasic component of the response initiated by the second stimulus decreased. A similar decrease in amplitude was observed in the turtle olfactory nerve and was attributed to an increase in extracellular potassium (Eng and Kocsis, 1987). At an interpulse interval less than 3 ms, a second triphasic component could not be detected, indicating that the absolute refractory period of this triphasic potential is less than 3 ms. The ability of the triphasic component to follow paired-pulse stimulation above 333 Hz is consistent with the absolute refractory period of action potentials (Erlanger and Gasser, 1936; Nicoll, 1972).

Olfactory Receptor Neuron Conduction Velocity

Triphasic compound action potentials were recorded at several distances from a rostral stimulating electrode and the latency to the trough of the large negative deflection was measured. Figure 3 shows a plot of mean latency as a function of distance for these orthodromic stimulations at rostral MOB. The inset in Figure 3 shows a typical response evoked by rostral bulb stimulation. The line through the data is a linear regression calculated by a weighted method of determinants. This line has a slope of 0.42 ± 0.01 m/s ($n = 25$); this slope is equal to the conduction velocity of the rat ORN axons.

To record responses from ORN axons that project to glomeruli in the caudal MOB, the stimulation electrode was positioned about midway across the MOB with the recording electrode at more caudal positions (see Fig. 1). With this configuration, only axons that project to

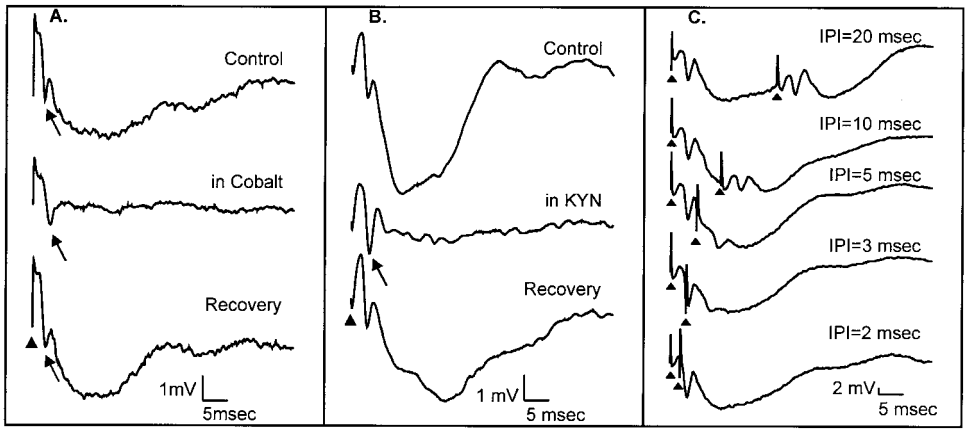

Fig. 2. Identification of the compound action potential in the rat. **A:** Effects of Cobalt. Each response was evoked by a 30 μs, 720 μA stimulus (filled arrowhead) and recorded by a surface electrode 0.5 mm from the stimulating electrode. The control response (**top**) consisted of a stimulus artifact, a fast, triphasic component with a prominent negative trough (arrow) at a latency of 2.3 ms, and a slower negative component that reaches its minimum with a latency of about 14 ms. The **middle** response was recorded after a 10-mM solution of cobalt chloride was applied to the surface of the bulb for 4.5 minutes; cobalt diminished the slower negative component, but spared the triphasic component of the response (arrow). The **bottom** response was recorded after the bulb was flushed with saline for 10 minutes to remove the cobalt; the slower negative component that follows the triphasic component (arrow) recovered (Exp 092397E). **B:** Effects of kynurenic acid (KYN). Each response was evoked by a 20 μs, 930 μA stimulus (filled arrowhead) and recorded by a surface electrode 0.83 mm from the stimulating electrode. The control response (**top**) is similar to the control response in A. The middle response was recorded after a 10-mM solution of KYN was applied for 3.5 minutes; KYN diminished the slower negativity, but spared the triphasic component (arrow). The **bottom** response was recorded after the bulb was flushed with saline for 10 minutes; the control response recovered (Exp 050798D). **C:** Paired-pulse stimulation. Each response was evoked by a 50 μs, 930 μA stimulus (filled arrowhead) and recorded by a surface electrode 1.05 mm from the stimulating electrode. Stimuli with different interpulse intervals (IPI), indicated above each trace, were presented. A second triphasic component from the second stimulus of a pair was detected at an IPI of 3 ms but could not be detected when the IPI was reduced to 2 ms (Exp 030697C).

glomeruli in the caudal half of the MOB should be stimulated. The inset in Figure 4 shows that the responses from these more caudally projecting ORN axons were similar to the responses evoked by rostral bulb stimulation. Such "caudal responses" were recorded at several positions (distances) caudal to the stimulating electrode, and the latency to the negative component of the compound action potential was measured. Figure 4 shows a plot of mean latency as a function of distance. The slope of the calculated regression line, equal to the conduction velocity of ORN axons that project to the caudal MOB, is 0.58 ± 0.19 m/s (n = 8). An analysis of covariance for significance between this value and the one determined from data recorded using orthodromic stimulation at rostral MOB gave a P value of 0.02, not quite statistically different.

A second approach to selectively stimulate those ORN axons that project to caudal aspects of the MOB is antidromic stimulation, with the stimulation electrode at the caudal MOB. The inset in Figure 5 shows a typical antidromically driven response. The waveform of the compound action potential is similar to responses evoked by orthodromic stimulation. However, rather than being followed by a slow negative deflection, the slower component of the antidromic response has a positive deflection. This slow positivity has been attributed to inhibitory currents that were being generated by deep bulbar neurons located under the recording electrode (Shepherd and Haberly, 1970). Antidromic responses were recorded at several distances from a caudal stimulation electrode. Figure 5 shows a plot of mean latency as a function of distance. The slope of the calculated regression line, representing the conduction velocity of ORN axons projecting to the caudal bulb, is 0.57 ± 0.19 m/s (n = 9). The P value of a comparison between this velocity value and the value determined using orthodromic stimulation at rostral MOB is 0.22, indicating that the conduction velocities of caudally projecting ORNs are not significantly different from ORNs projecting to more rostral targets.

DISCUSSION

The pike and the garfish are good experimental models for studying impulse conduction along olfactory receptor neurons because 10 to 15 mm of olfactory nerve can be extracted. However, many of the recent studies of the physiology of ORNs has been done with rats and mice. In this review, impulse conduction of rat ORNs was studied by recording the compound action potential along the surface of the olfactory bulb. Despite relatively short distances separating the stimulation and recording electrodes, the impulse conduction velocity and changes in conduction velocity with position on the MOB could be analyzed.

The triphasic compound action potential was identified in field potential recordings from the lateral surface of the MOB of the rat based on its waveform, relatively short latency, and its ability to follow paired-pulse stimulation at <333 Hz. These characteristics have been used in previous studies in rabbit (Nicoll, 1972), cat (Freeman, 1972), and mouse (Griff et al., 2000) to identify the compound action potential. A search through the literature suggests that the present study is the first analysis of the compound action potential from olfactory receptor neurons in rat.

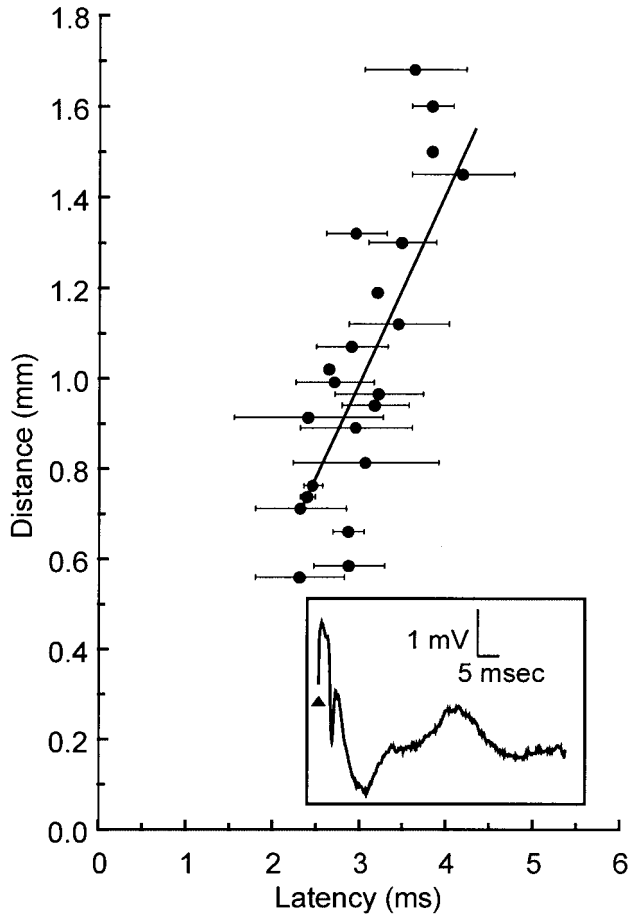

Fig. 3. Orthodromic stimulation at rostral olfactory bulb. Graph: Mean latencies and standard deviations of compound action potentials were plotted as a function of the distance between the stimulating and recording electrodes. A linear regression line was calculated using the weighted method of determinants; the slope of this line, 0.42 ± 0.01 m/s, is equal to the conduction velocity. **Inset:** This representative response was evoked using orthodromic stimulation (arrowhead) at the rostral MOB and recorded with an electrode 1.28 mm away; the latency to the trough of the compound action potential was 3.18 ms (Exp 070897G).

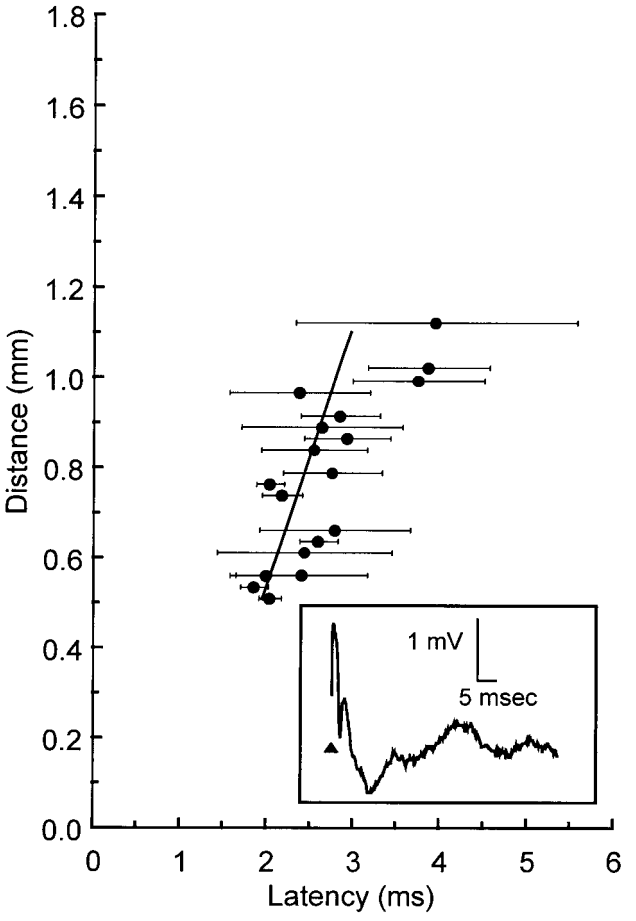

Fig. 4. Orthodromic stimulation at caudal olfactory bulb. Graph: Mean latencies and standard deviations of action potentials were plotted as a function of the distance between the stimulating and recording electrodes. A linear regression line was calculated using the weighted method of determinants; the slope of this line, 0.58 ± 0.19 m/s, is equal to the conduction velocity. **Inset:** This representative response was evoked using orthodromic stimulation (arrowhead) at the middle of the MOB and recorded at the caudal MOB with an electrode 0.9 mm away; the latency to the trough of the compound action potential was 2.28 ms (Exp 070897G).

The compound action potential was further differentiated from other components of the bulb field potential by evaluating the effects of drugs known to block postsynaptic responses. Cobalt blocks vesicular release of neurotransmitters (Weakly, 1973), and at 10 mM abolished the slower components while sparing the triphasic component; higher concentrations or prolonged exposure eliminated the entire response indicating a nonspecific action of cobalt at higher levels. Kynurenic acid, a non-selective antagonist of glutamate receptors (Watkins and Olverman, 1987) also abolished the slower component while sparing the triphasic component. These results provide evidence that the triphasic component is generated by neurons presynaptic to the bulbar neurons, most likely the ORNs, and support its identification as the compound action potential.

The results with kynurenic acid also support a hypothesis based in part on in vitro studies that the neurotransmitter released by rat ORNs onto mitral cells is glutamate (Ennis et al., 1996; Aroniadouo-Anderjaska et al., 1997). In those studies, a dual-component glutamatergic synaptic response was observed in the field potential and in single-unit recordings from mitral cells; a triphasic compound action potential was not evident. In the above experiments (Fig. 2B), no attempt was made to identify two components in the postsynaptic field potential.

The latency measured to the trough of the compound action potential was used to calculate the conduction velocity of ORNs in rat from a plot of latency vs. distance (Fig. 3). The slope of a straight line fitted to the data is an estimate of the conduction velocity; it was 0.42 ± 0.01 m/s for orthodromic stimulation at the rostral MOB. This value is similar to the conduction velocities of ORNs in other mammals (Freeman, 1972; Griff et al., 2000; MacLean et al., 1957; Nicoll, 1972).

Published data for impulse conduction velocities in various experimental animals are summarized in Ta-

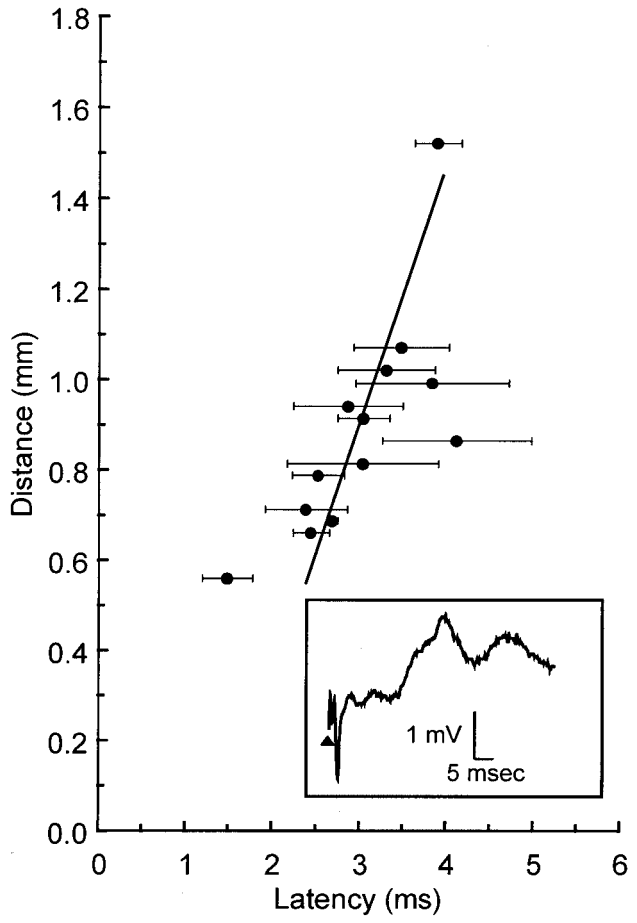

Fig. 5. Antidromic stimulation of the olfactory bulb. Graph: Mean latencies and standard deviations of action potentials were plotted as a function of the distance between the stimulating and recording electrodes. A linear regression line was calculated using the weighted method of determinants; the slope of this line, 0.57 ± 0.19 m/s, is equal to the conduction velocity. **Inset:** This representative response was evoked using antidromic stimulation (arrowhead) at the caudal MOB and recorded with an electrode 0.7 mm away (more rostral); the latency to the trough of the compound action potential was 2.48 ms (Exp 070897B).

TABLE 1. Conduction velocities of ORNs in vertebrates

Type	Animal	Velocity (m/s)	Source
Poikilotherms	Frog	0.14	Ottoson (1961)
	Garfish	0.1–0.2	Easton (1965)
	Pike	0.2	Gasser (1956)
	Turtle	0.16–0.18	Eng and Koscis (1987)
Homeotherms	Cat	0.42 ± 0.05	Freeman (1972)
	Hamster	0.26 ± 0.08	Costanzo and O'Connell (1978)
	Mouse	0.40 ± 0.09	Griff et al. (2000)
	Opposum	0.4	MacLean et al. (1957)
	Rabbit	0.44	Nicoll (1972)

ble 1. Impulse conduction measurements for poikilotherms (0.14–0.2 m/s) are two to three times slower than the impulse velocity measurements for the homeotherms (0.4–0.44 m/s), though the diameter of the axons in both poikilotherms and homeotherms is about 0.2 μm (Gasser, 1956). The difference in impulse conduction can be explained by the differences in temperature; the typical range of Q_{10}'s for most biological systems is in the range of 2 to 3.

In Figure 3, it is noteworthy that the fitted line does not pass through the origin, indicating that it takes time to generate an action potential at the stimulating electrode. This time has been termed the foot (Aidley, 1971). The foot time affects the calculation of conduction velocity since the measured latency includes the conduction time plus the foot time. When the recording electrode is close to the stimulating electrode, the foot time will be a larger percentage of the measured latency. Thus, at short distances, the latency overestimates the conduction time so that a conduction velocity calculated from this latency will be an underestimate. When the recording electrode is moved farther from the stimulating electrode, the foot time is a smaller percentage of the latency, and the conduction velocity appeared to increase. This provoked examination of the conduction velocity of ORN axons projecting to caudal glomeruli.

Measurements of the conduction velocity of ORN axons that project to the caudal bulb were made using orthodromic stimulation at more caudal positions and using antidromic stimulation. Linear regressions through these data yielded conduction velocities of 0.58 and 0.57 m/s, respectively. Even though these velocities are both greater than the velocity measured from orthodromic stimulation at rostral MOB, statistically they are not different. One complication is that rostral orthodromic stimulation activates axons that project to both rostral and caudal areas of the MOB; axons projecting to only rostral targets are not isolated. Thus, the analysis for rostral orthodromic stimulation may overestimate the conduction velocity of axons that project only to rostral targets. Also noteworthy are the relatively large standard errors for both caudal and antidromic stimulation. One explanation is the smaller number of measurements. In addition, relatively few axons project to the caudal bulb and can be stimulated. Some of these caudally-projecting axons may have slower conduction velocities producing the larger variability in the measurement. Nonetheless, the current study provides no clear evidence for a subset of faster (or slower) conducting axons projecting to the caudal MOB.

Faster conduction to the caudal bulb would allow action potentials to arrive at glomeruli in the caudal bulb at approximately the same time as impulses to rostral glomuli. Given the geometry of projections from the nasal compartment to the MOB, a conduction velocity of ORN axons to caudal glomeruli up to two times the conduction velocity of axons targeting rostral glomeruli would allow action potentials in the rostral and caudal bulb to arrive simultaneously. The data, however, did not show a significant increase. Faster conduction may not be necessary if the integration time of most mitral and/or tufted cells is long enough to effectively sum signals with delays of many milliseconds. However, some mitral cells exhibit very short excitatory responses (Jiang et al., 1996). Thus, there may need to be a subpopulation of mitral cells that have a short effective integration time and require faster con-

ducting ORN axons to ensure simultaneous excitations of these mitral cells in different glomeruli. Results from a recent in vivo study suggest that synchronized spike discharges of mitral/tufted cells from different glomeruli may be important for integrating signals from different ORN receptors (Kashiwadani et al., 1999). Thus, continued progress in understanding how olfactory information is coded may provoke further consideration of ORN conduction velocity and vice versa.

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