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Benzyl Alcohol Blocks Voltage-Gated Sodium Channels and Firings of Cortical Neurons of Rat Brains

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Background: Benzyl alcohol (BZA) is an organic compound widely used as a bacteriostatic preservative. In addition, BZA has local anesthetic properties. This is supported by previous studies which have demonstrated that BZA inhibits muscle and neuronal sodium channels expressed in human embryonic kidney cells (Haeseler et al., 2000; Buchholz et al., 2009). However, the effects of BZA, on sodium currents and action potentials of native neurons, have not been reported. In this study, we have examined the properties of BZA on voltage-gated sodium channel currents, and the spontaneous firing, of cortical neurons from rat brains.

Method: Patch-clamp techniques, including the whole-cell and cell-attached modes, were used to record electrophysiological signals from neurons freshly-isolated from the cortex of rat brains (age 10-20 postnatal days).

Results: BZA reversibly suppressed depolarization-induced whole-cell sodium inward currents. This blockade depended on BZA concentrations, with an IC(50) of 7.5 mM for the sodium currents evoked from a holding potential of -70 mV. BZA also blocked the sodium currents evoked by all test potentials. In addition, BZA (8.3 mM) significantly shifted the ½ activation voltage from -49.5 ± 3.6 mV to -39.6 ± 5.8 mV (n = 8) and the slope factors from 3.7 ± 4.2 to 7.2 ± 5.1. Conversely, BZA (8.3 mM) shifted the ½ inactivation voltage from -66.0 ± 0.5 mV to -72.4 ± 0.4 mV (n=7) without significantly changing the slope factors. Also, BZA (8.3 mM) did not significantly change the recovery times. These were noted to be 27.0 ms for the control group and 28.3 ms for the BZA treated group. This blockade was noted to start immediately as BZA was applied to each cell body. The blockade then reached a plateau within 3 s. Following this, the blockade recovered to its control level within 2 s after washout of the BZA-containing solution. Furthermore, increasing the stimulating frequency, from 1 to 5 Hz, shortened the onset times but prolonged the offset times. This indicates that BZA blockade is use-dependent. Finally, BZA suppressed the spontaneous firing of cortical neurons.

Conclusion: BZA blocks voltage-gated sodium currents, and spontaneous firings, of freshly-isolated neurons from the cortex of rat brains. These effects may contribute to its local anesthetic properties.

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