Neural Cell Adhesion Molecule expression in the human carotid body

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SUMMARY

We studied by immunocytochemistry the expression of NCAM in human carotid bodies, sampled at autopsy from 16 adult subjects (mean age ± SD: 44.3 ± 3.4 years). No NCAM immunoreactivity was visible in type II cells. An high percentage (78.3 ± 7.2%) of type I cells showed positive anti-NCAM immunoreaction. Statistically significant differences were not found in anti-NCAM immunostaining of light and dark cells (80.2 ± 6.2% vs 74.7 ± 13.4%, P>0.05). The high expression level of NCAM in the carotid body indicates a role in regulating adhesion between type I cells. The ascertained role of NCAM in neural mechanisms of differentiation, survival and cell plasticity suggests a possible involvement in the development/differentiation process of the carotid body and in determining cellular/molecular changes due to chronic hypoxia.

INTRODUCTION

The carotid body is a small tissue mass, which is located at the carotid bifurcation and plays a role as arterial chemoreceptor, inducing increases in ventilatory volume and frequency in response to hypoxia, hypercapnia, or reduction of blood pH. The carotid body is organized in lobules, separated by thin connective septa arising from the capsule. The cells of the lobules belong to two different populations: type I (or chief) cells, with much cytoplasm and a few dendritic processes extending into extracellular spaces, and type II (or sustentacular) cells, with fusiform shape and located at the edges of the clusters (Verna, 1979; Smith et al., 1982; Pallot et al., 1986; Pallot, 1987). Type I cells, separated into light, dark and pyknotic, release various neurotransmitters or neuromodulators in response to carotid body stimulation and represent the real chemoreceptor elements of the carotid body. Type II cells show astrocytic markers and play a supportive role (Pallot et al., 1986; Pallot, 1987), although it has also been reported that these cells exposed to prolonged hypoxia may behave as stem cells precursors for type I cells (Pardal et al., 2007). Carotid body cells are known to express many different growth factors and