Supplementary Figure 1  SP-SAP specifically ablates NK1R neurons within the preBötC. Schematic of transverse medullary slice denotes extent of preBötC (circle) and surrounding area (rectangle) analyzed for NK1R neurons. All histological analyses were carried out > day 10 post injection, when the rats exhibited an ataxic breathing pattern. *In vivo*, neuronal death following SAP-induced lesions occurs over a period of days with most targeted cells ablated by day 7 post injection\(^1\). Lesions were confined to the preBötC where NK1R immunopositive staining was significantly reduced or completely absent. In some additional cases with identical outcomes (\(n=3\)), a small number of neurons within the nucleus ambiguous (NA) were damaged; thus we can not exclude that the pathological changes in breathing observed here (see also\(^{13}\)) may be partly attributed to a small amount of upper airway motoneuronal damage within the NA, or the destruction of a small number of NK1R bulbospinal neurons that are also located within this area\(^{17}\). However, such changes would be unlikely to account for the disturbances of rhythm. There was no significant neuronal loss in other regions examined (e.g., solitary tract and motor nucleus of vagus), similar to the results reported in our previous study using the same injection protocol\(^{13}\). An ataxic breathing pattern did not develop in rats with destruction of <50% of preBötC NK1R neurons (\(n=3\)). These rats were monitored up to 20 days post-injection, during which time respiratory disorders did not increase significantly in frequency during sleep or wakefulness (\(P>0.05\)). scNA, compact nucleus ambiguous; IO, inferior olive; XII hypoglossal nucleus; 5SP, trigeminal nucleus. Scale bar, 200 µm.