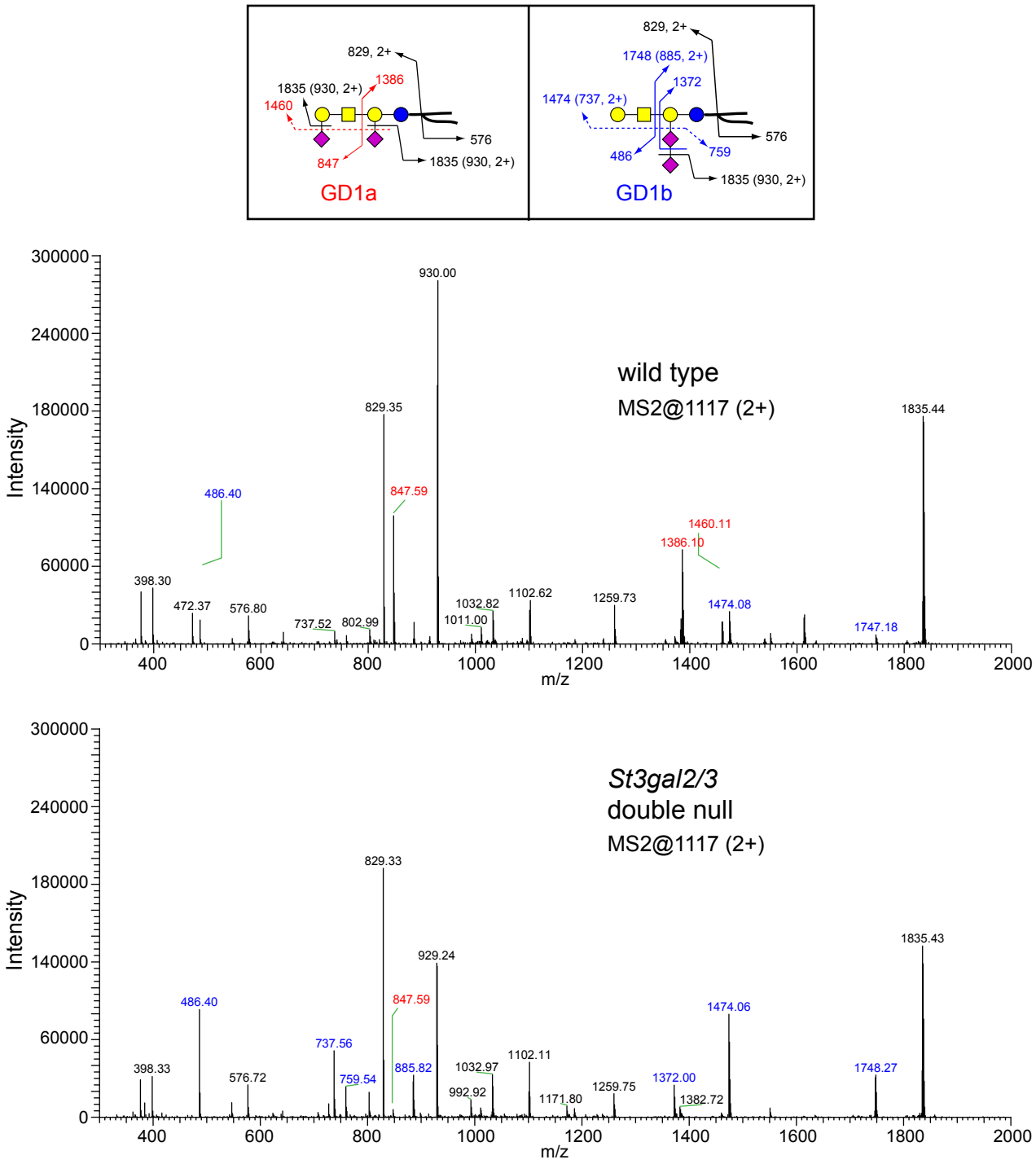

Fig. S1. GD1a is depleted in St3gal2/3-double null mice. Wild type and mutant mice express comparable intensities of GD1 ions common to GD1a and GD1b. Whereas the wild type is relatively enriched in GD1a ions (red), the mutant is greatly enriched in GD1b ions (blue).

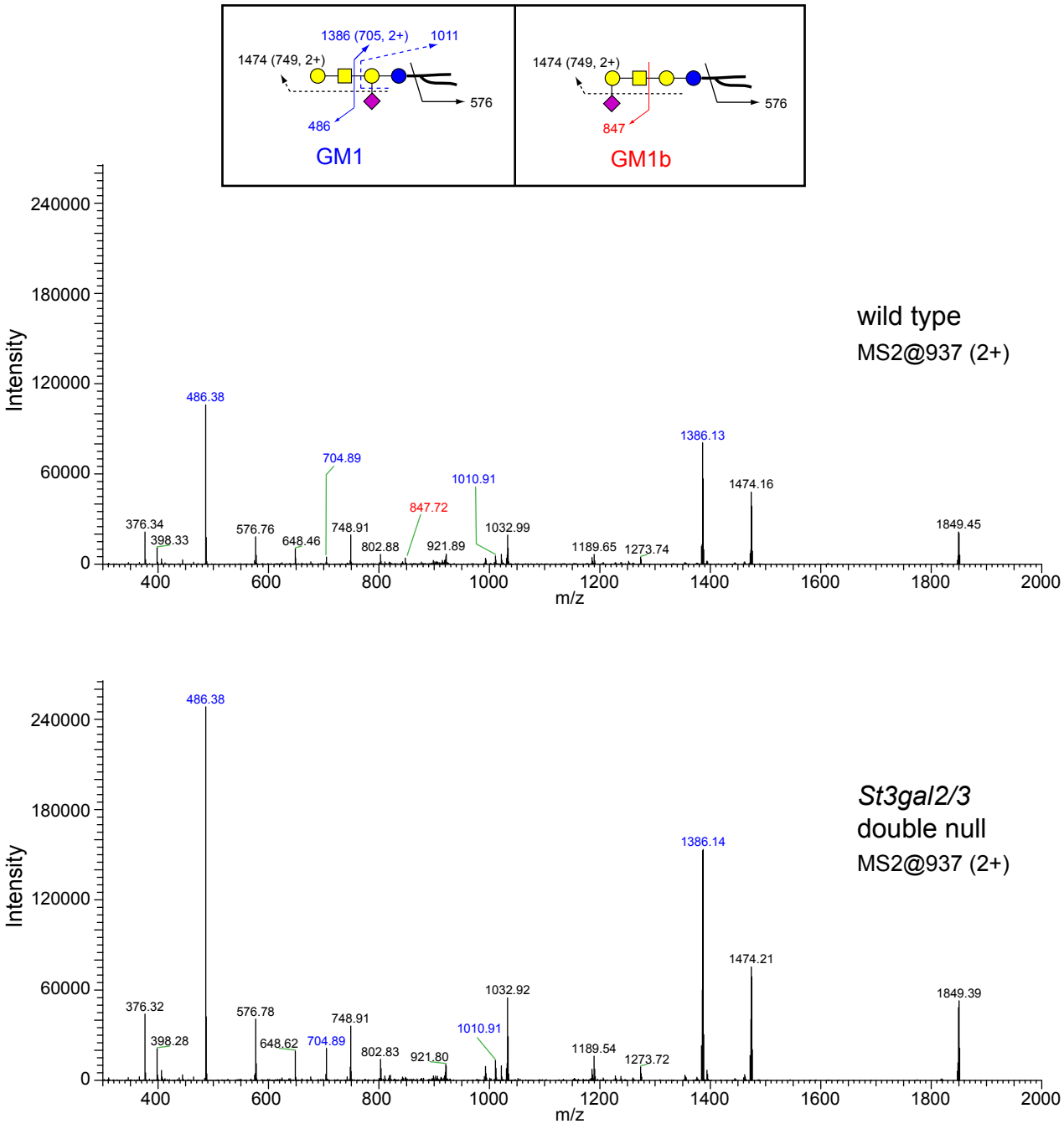

Fig. S2. GM1b (cisGM1), a minor species in wild type mice, is undetectable in St3gal2/3-double null mice. Wild type and mutant mice both express ample GM1 ions (blue), although their signal intensity is higher in the mutant. Wild type mice expresses an ion (red) that is characteristic of the minor ganglioside GM1b, which has a terminal sialic acid. This ion is not detected in the mutant.

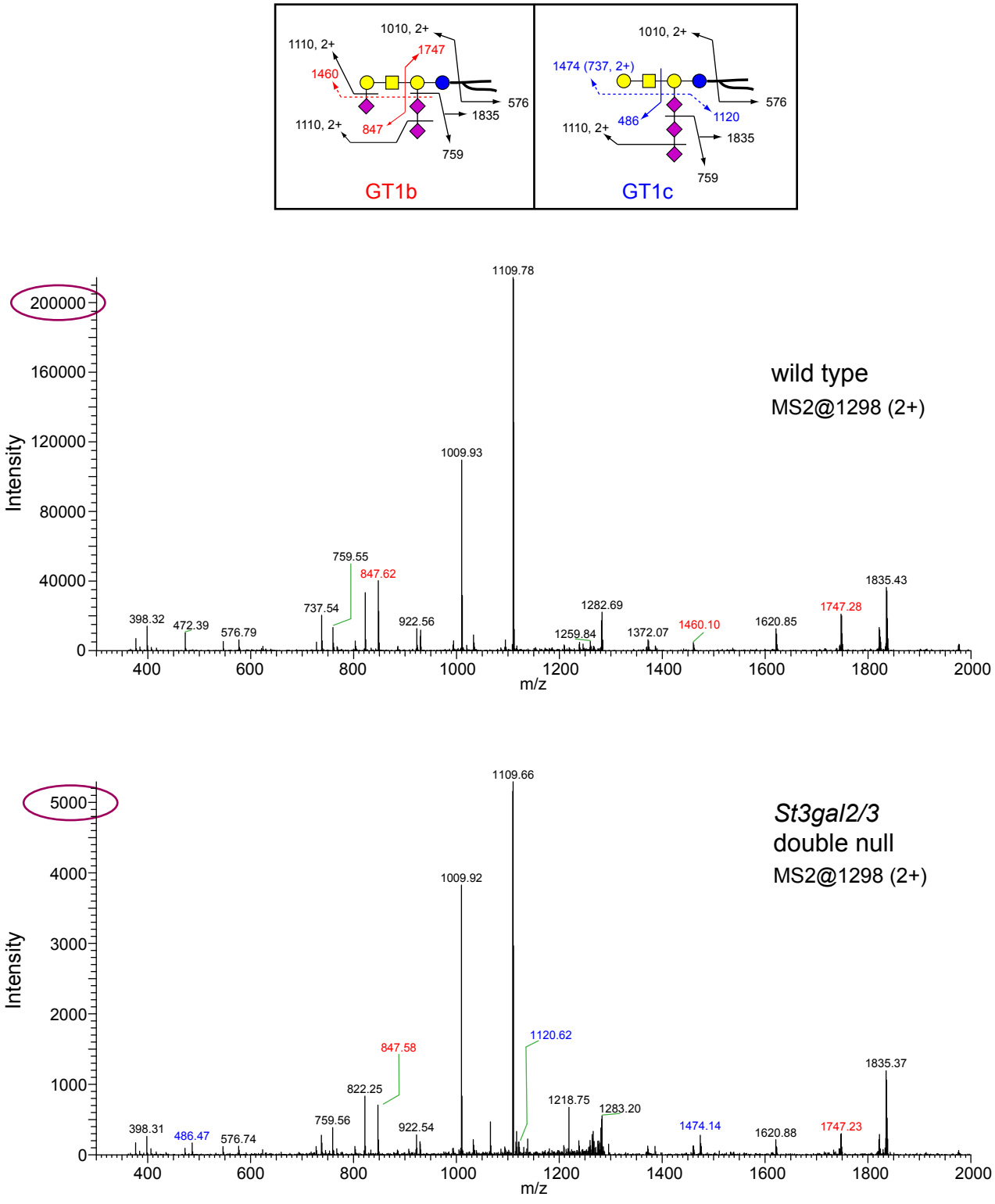

Fig. S3. GT1 species are depleted in St3gal2/3-double null mice. The intensity of ions common to trisialogangliosides are greatly diminished in the mutant mice. Nevertheless, ions selective for GT1c (blue) are apparent in the mutant, but are not detected in wild type mice.

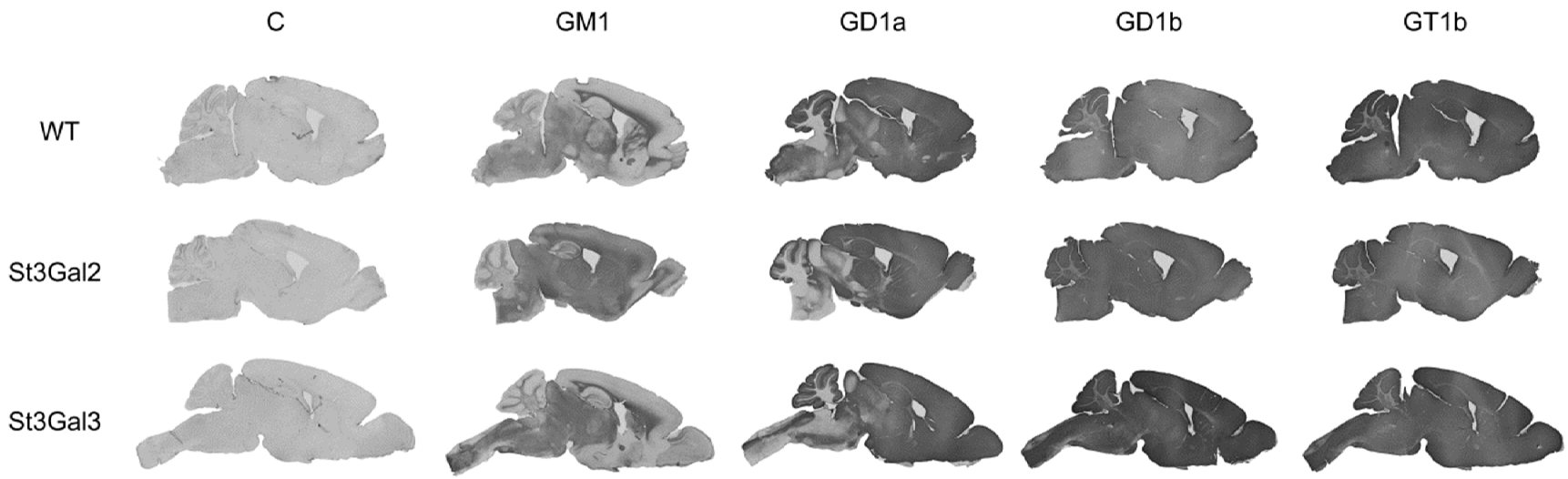

Fig. S4. Ganglioside immunohistochemistry on wild type, *St3gal2*-null and *St3gal3*-null mouse mid-sagittal brain sections. In *St3gal2*-null mice, note the expansion of GM1 immunostaining from white matter only and the diminished GD1a immunostaining in the caudal-most (left) brain structures.

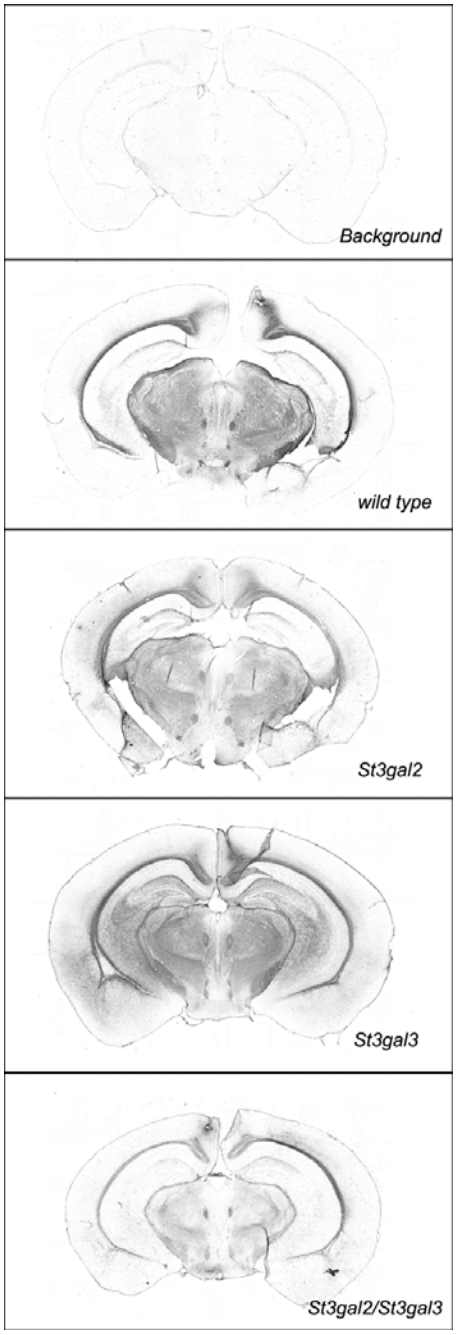

Fig. S5. Myelin-associated glycoprotein (MAG) immunohistochemistry on wild type and St3gal null mouse coronal brain sections. White matter tracts are stained throughout the brain, with intensities reduced in the *St3gal2/3*-double null mice (bottom).