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DATASHEET

Cuprizone Model of Demyelination & Remyelination

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OVERVIEW

The cuprizone model is a well-characterized, toxin-induced model of CNS demyelination and remyelination relevant to multiple sclerosis (MS). Unlike EAE, it produces demyelination without a primary autoimmune response, so oligodendrocyte injury and repair can be studied directly.

Cuprizone Model

Cuprizone is a copper chelator that, given in the diet, causes reproducible oligodendrocyte loss and demyelination in defined brain regions, most notably the corpus callosum. Its withdrawal allows spontaneous remyelination, making the model well suited to neuroprotective and remyelinating therapies.

Demyelination is induced in C57BL/6 male mice on a cuprizone diet, with rapamycin co-administered as part of the model to drive consistent demyelination and reduce variability. Brains are sectioned at the corpus callosum and stained for MBP, with terminations at 7 and 13 weeks.

Species	C57BL/6 male mice, 8-10 weeks
Study Duration	7-13 weeks, unless otherwise specified
In vivo Assessments	Body weight (weekly)
Endpoints	Histology for myelin (MBP) MBP optical density (corpus callosum) Corpus callosum thickness

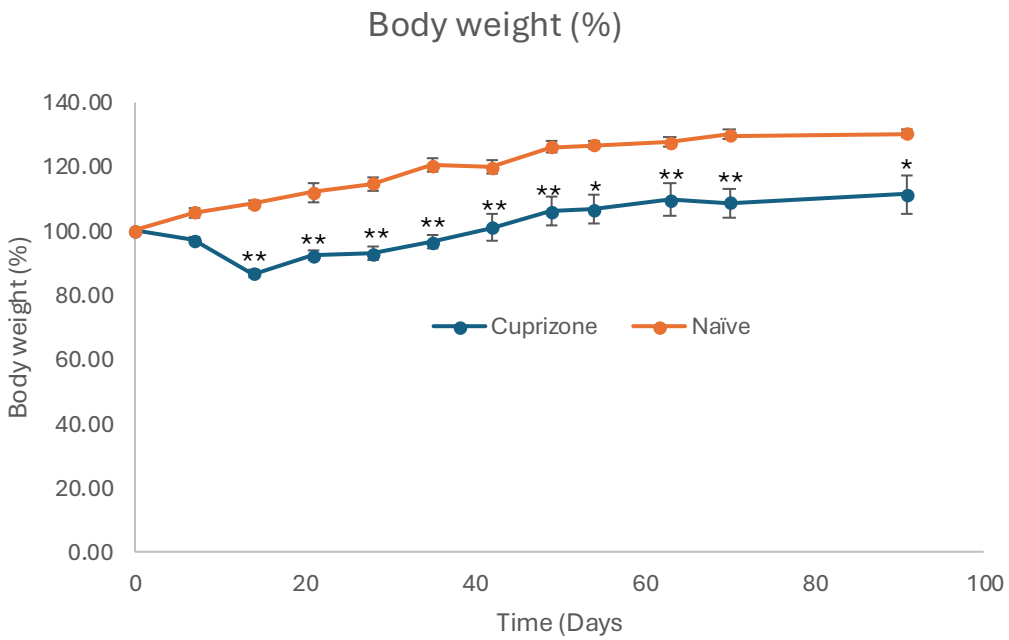

Figure 1: Body weight. Body weight is recorded weekly across normal-diet and cuprizone-diet groups.

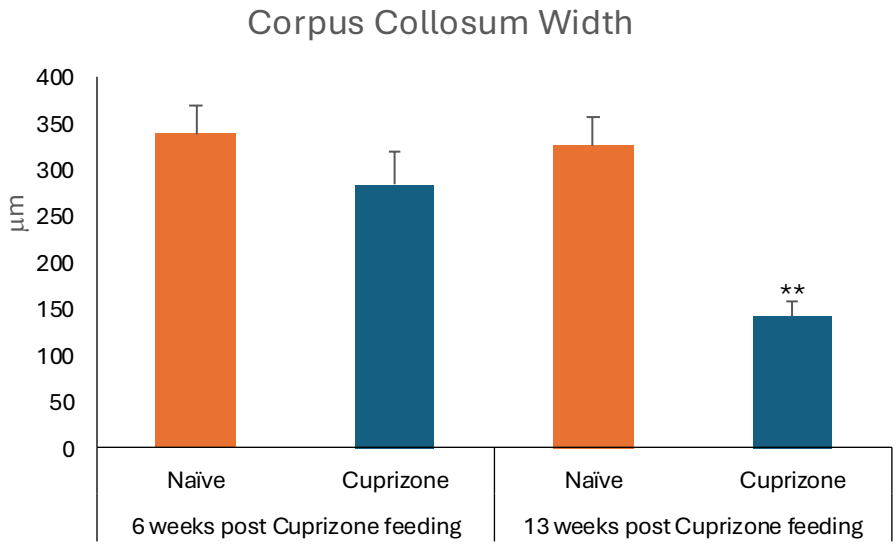

Figure 2: Corpus callosum thickness, from MBP-stained sections at 7 and 13 week terminations.

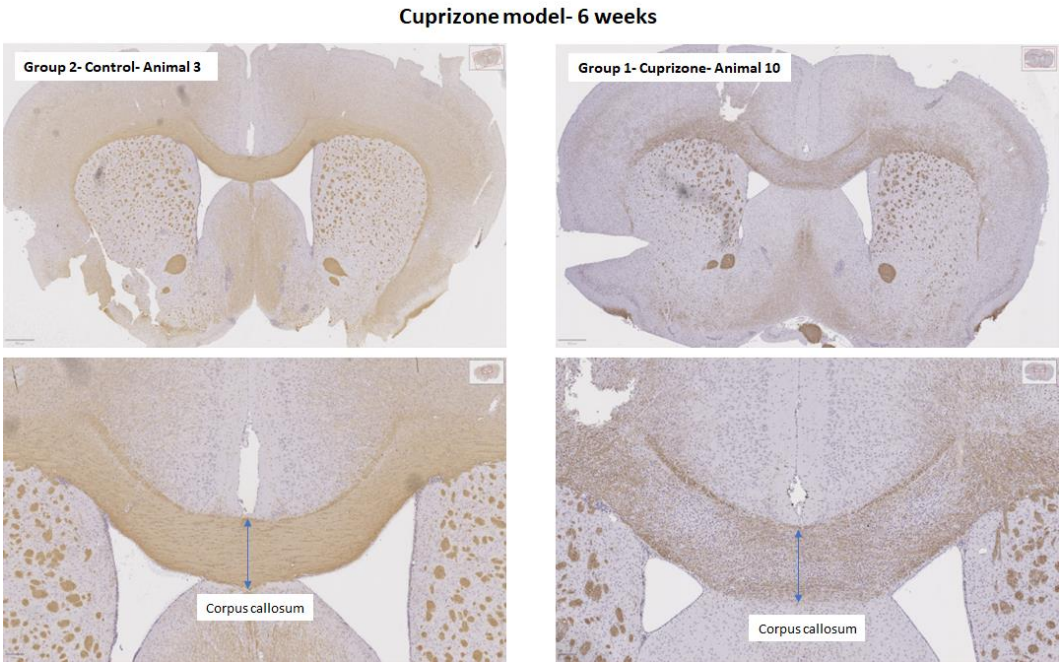

Figure 3: MBP staining, 6 weeks. Coronal sections (control vs cuprizone) stained for MBP; reduced corpus callosum staining indicates demyelination.

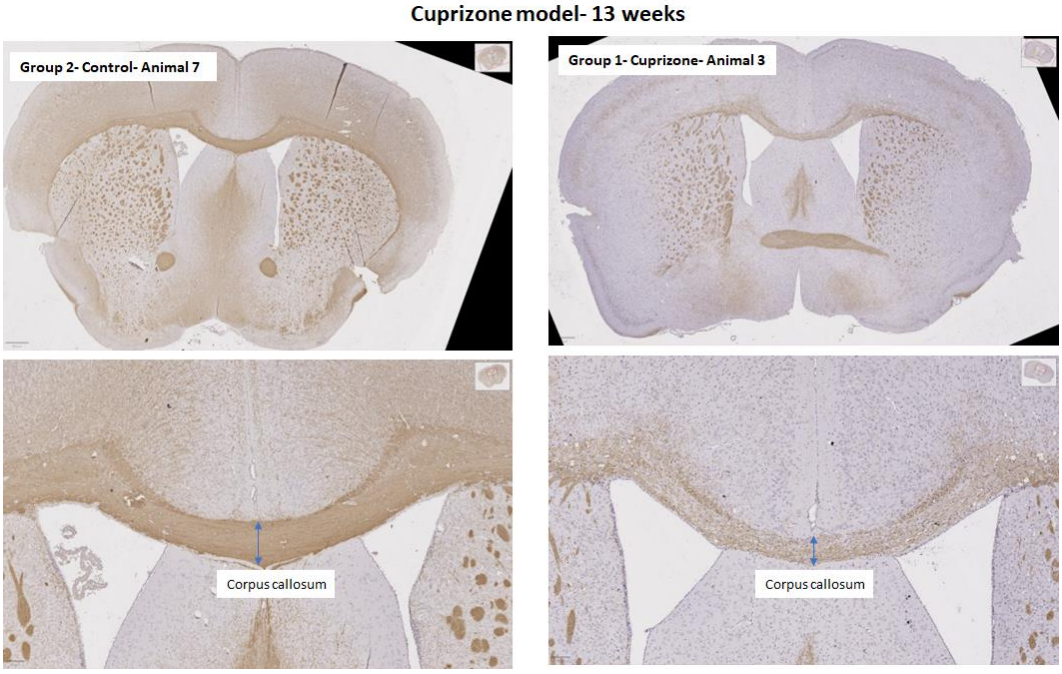

Figure 4: MBP staining, 13 weeks. Demyelination of the corpus callosum is more pronounced after 13 weeks of cuprizone.

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