LETTER TO THE EDITOR

Therapeutic potential of β-lactam ceftriaxone for chronic pain in sickle cell disease

Ying He[1], Xiao Guo[1] and Zaijie Jim Wang[1]

[1]Departments of Pharmaceutical Sciences, Neurology & Rehabilitation, Bioengineering, Center for Biomolecular Sciences, and Sickle Cell Center, University of Illinois, Chicago, IL, USA

Correspondence:
Y. HE - yhe8uic@gmail.com
Z. J. WANG - zjwang@uic.edu

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Figure S1

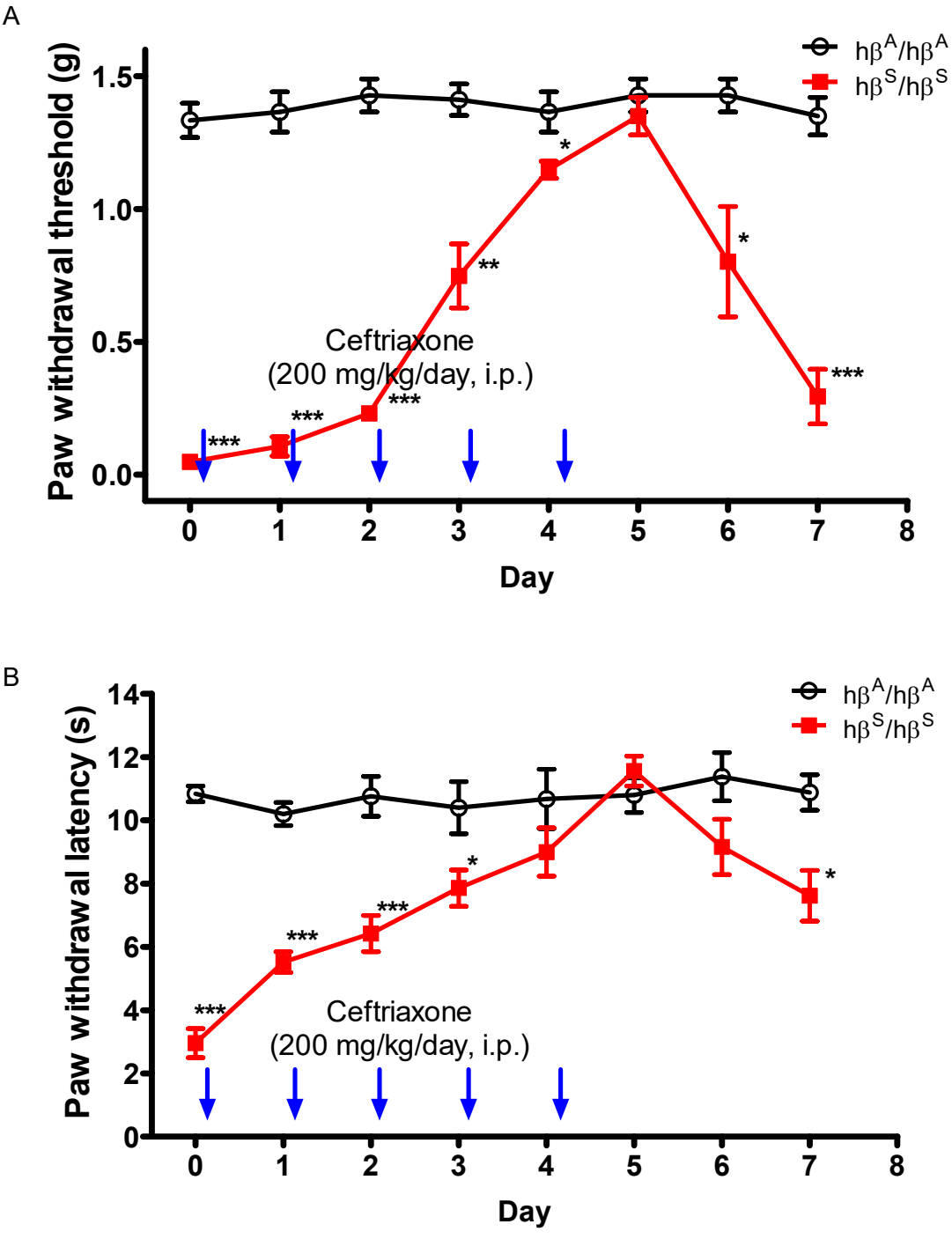
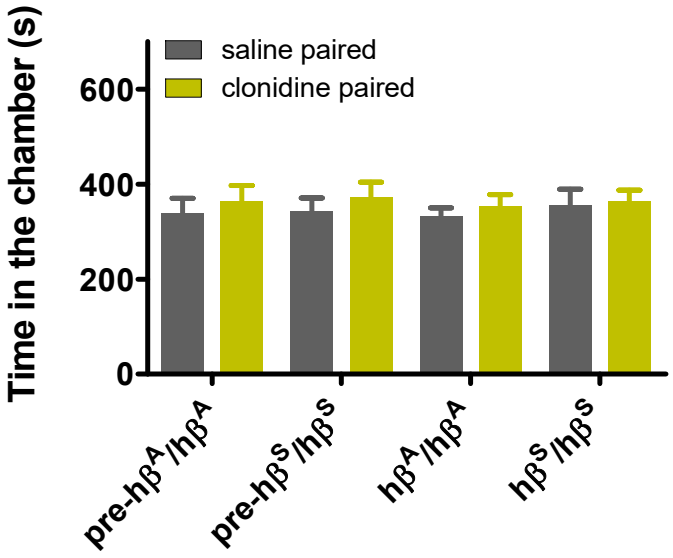


Figure S1. Mechanical (**A**) and thermal (**B**) sensitivities before and after the treatment with ceftriaxone (200 mg/kg/day, *i.p.* × 5 days). *** $P < 0.001$ vs. "$h\beta^A/h\beta^A$" group; n = 8/group

Figure S2

A



B

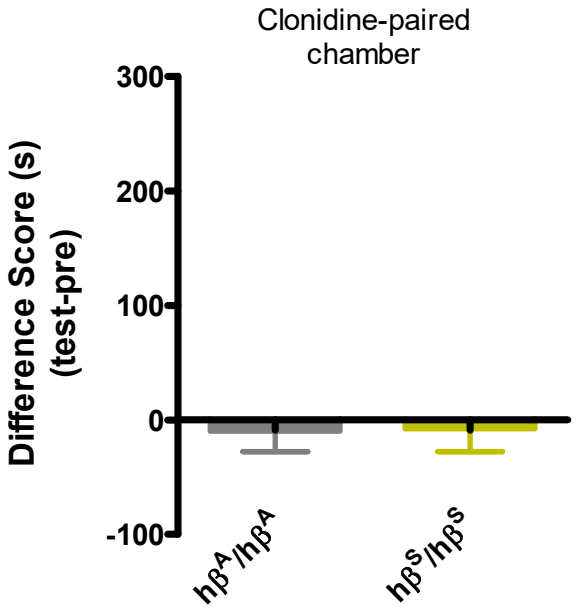
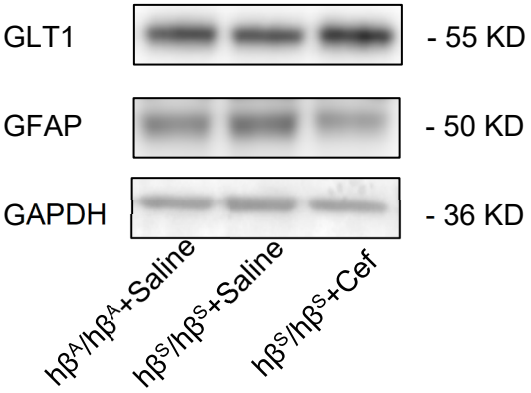
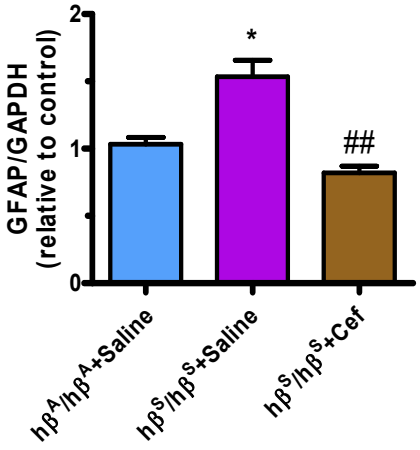


Figure S2. When tested on Day 30, clonidine (1 µg, *i.t.*) did not induce CPP in ceftriaxone-treated TOW $h\beta^S/h\beta^S$ mice or $h\beta^A/h\beta^A$ mice. (**A**) $h\beta^S/h\beta^S$ and $h\beta^A/h\beta^A$ mice spent similar amount of time in saline- or clonidine-paired chambers. (**B**) Different scores (test time – preconditioning time spent in the clonidine chamber) confirmed the absence of chamber preference.

Figure S3





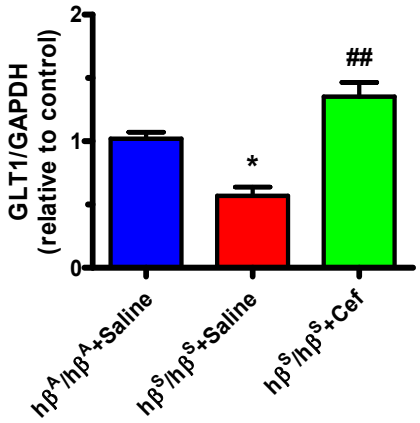


Figure S3. Western blotting analysis of GFAP and GLT1 in the dorsal root ganglion of TOW $h\beta^S/h\beta^S$ mice and control $h\beta^A/h\beta^A$ mice. Ceftriaxone reversed the up-regulation of GFAP and the down-regulation of GLT1 in TOW hβS/hβS mice. * $P < 0.05$, vs. $h\beta^A/h\beta^A$ mice+saline group. ## $P < 0.01$ vs. $h\beta^S/h\beta^S$ +saline group, n = 3/group.