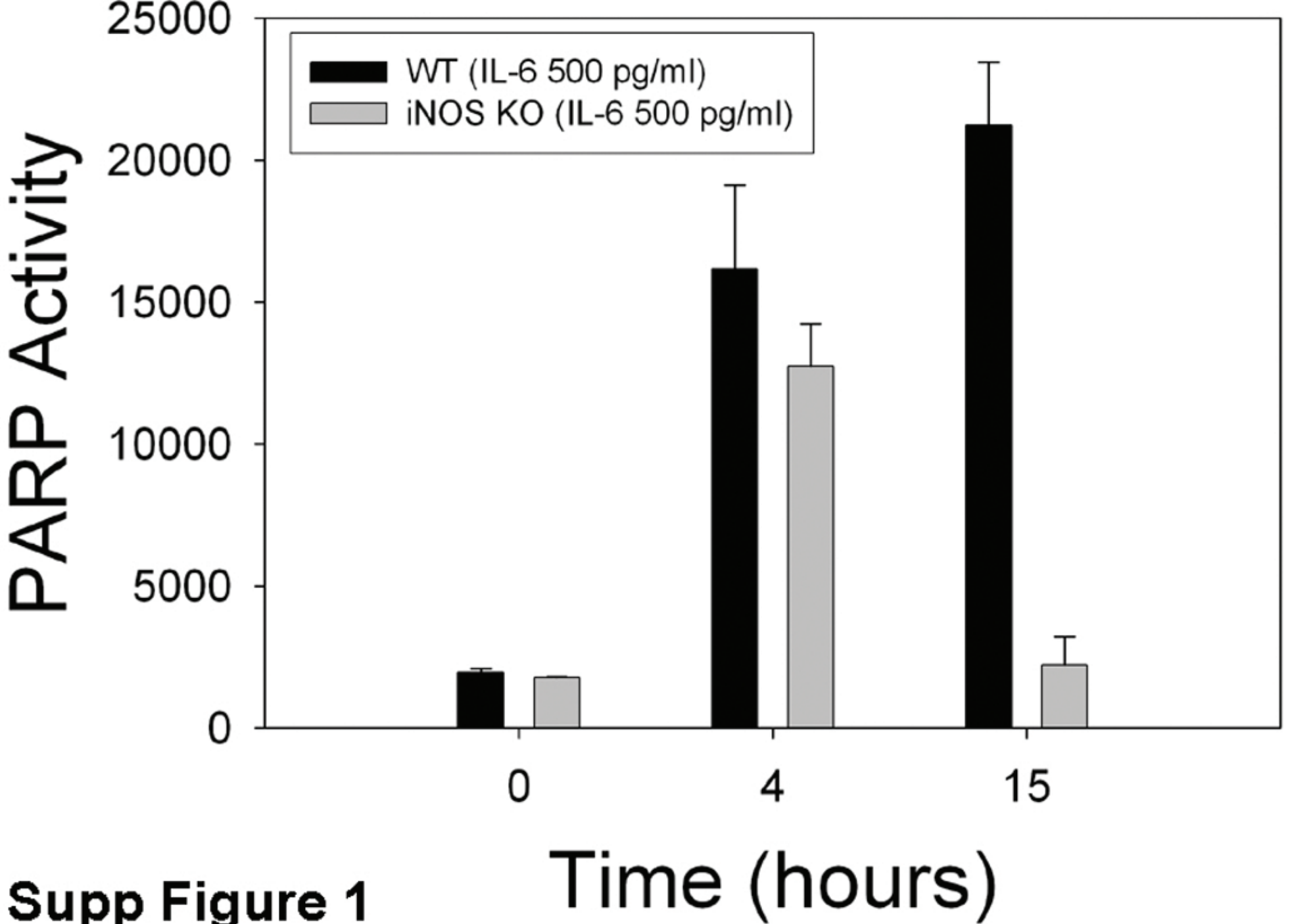
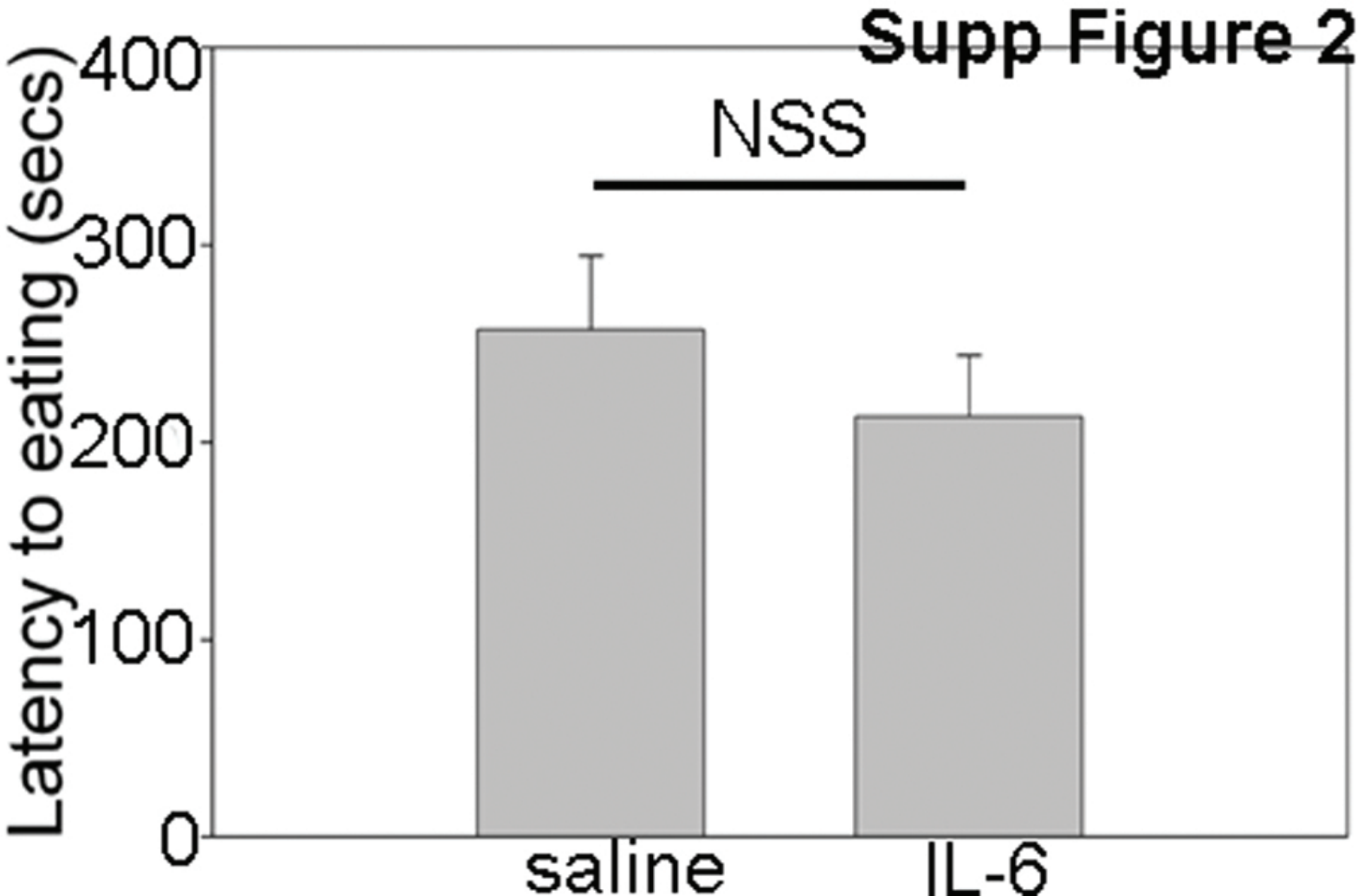


Supp Figure 1


Supp Figure 2
NSS
Latency to eating (secs)
400
300
200
100
0
saline
IL-6

SUPPLEMENTARY MATERIAL

Supplementary Methods: Novelty-induced food intake was utilized as a measure of animal anxiety to determine whether IL-6 induced any change in this measure. The testing apparatus consisted of a Plexiglas box (60 x 60 x 50 cm). Forty-eight hours before testing, all food was removed from the home cage. For testing, 3-4 food pellets were placed in the center of the testing box and an animal was placed into a randomly chosen corner of the testing box and a stopwatch was immediately started. The latency to begin eating was measured and was defined as chewing the food, not simply sniffing or playing with a pellet. If a rat had not eaten within 360 seconds, the test was stopped and the animal was assigned a latency score of 360 seconds and the animal was returned to the home cage. The data for novelty-induced food intake inhibition were analyzed using Student t-test.

Supplementary Figure Legends

Figure 1: iNOS KO mice exhibit only an early but not a late peak of PARP activity following IL-6 administration in spinal cord organotypic cultures. Mouse spinal cord organotypic cultures were generated from wildtype (WT) or iNOS knockout (KO) mice and IL-6 was added at 500 pg/ml. At 4 and 15 hours after IL-6 administration, tissue lysates were generated and PARP activity was assessed. The iNOS KO mice cultures exhibited an early increase in PARP activity that was no different from WT controls. However, at 15 hours and at other, later time points (data not shown), iNOS KO mice failed to activate PARP ($P < 0.05$), further confirming that the second peak of PARP activity (described in Figure 5A of the main manuscript) is dependent upon iNOS activation.

Figure 2: Intracerebroventricular infusion of IL-6 (2000 pg/ml) does not induce cognitive/behavioral changes in adult rats. The figure depicts the latencies to eating in the IL-6-treated and control rats. Although the control rats had a trend towards a greater latency to eating, no significant differences between the two groups were seen, $P > 0.05$

SUPPLEMENTARY TABLE

Demographic and clinical characteristics of idiopathic transverse myelitis and control patients

Characteristic	Transverse Myelitis	Control
Age (in years) (mean ± SEM)	48.7 ± 3.5 ($n = 21$)	49.9 ± 3.8 ($n = 16$)
Gender (% women)	58.0 % ($n = 25$)	62.5 % ($n = 16$)
Time to follow up (months)	6.5 ± 0.79 ($n = 22$)	18.5 ± 3.8 ($n = 16$)

(mean ± SEM)		
Percent with gadolinium enhancing lesions	78% (*n* = 14)	12.5 % (*n* = 16)
WBC in CSF (mean ± SEM)	10.8 ± 4.5 (*n* = 20)	2.1±1.2 (*n* = 11)
Protein in CSF (mg/dl) (mean ± SEM)	58.7 ± 11.4 (*n* = 16)	39.2 ± 11.5 (*n* = 10)
Protein in serum (g/dl) (mean ± SEM)	6.5 ± 0.4 (*n* = 8)	7.0 ± 0.5 (*n* = 4)
Acute EDSS (mean ± SEM)	6.4 ± 0.5 (*n* = 22)	3.8 ± 0.7 (*n* = 16)