

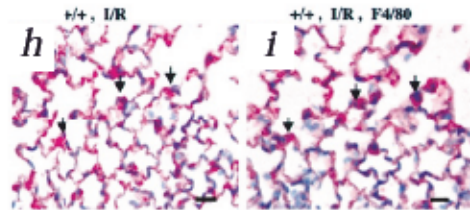
ERRATA

Egr-1, a master switch coordinating upregulation of divergent gene families underlying ischemic stress

SHI-FANG YAN, TOMOYUKI FUJITA, JIESHENG LU, KENJI OKADA, YU SHAN ZOU, NIGEL MACKMAN, DAVID J. PINSKY & DAVID M. STERN

Nature Med. 6, 1355-1361 (2000)

In Fig. 1, panels h and i are incorrect. The correct panels are shown here.



We regret this error.

Genetic models for CNS inflammation

TREVOR OWENS, HARTMUT WEKERLE & JACK ANTEL

Nature Med. 7, 161-166 (2001)

Table 4 was incorrect. The corrected Table 4 is shown here.

Table 4 MS versus EAE

Treatment	MS		EAE	Transgenics or knockouts
	RR	SP		
IFN- β	Alleviated ⁶²	No or minimal benefit ⁶²	Inhibited/alleviated ⁶²	NR
IFN- γ	Exacerbated ⁶³	Transient worsening only ⁴	Inflammatory (intra-CNS) ^{23,24} Suppressive (intraventricular) ²⁵	Inflammatory ^{22,28} in transgenics
IFN- γ blockade	Alleviated ^{62,6}	NR	Exacerbated ^{36,40}	Knockouts ^{35,36} generate a novel disease
TNF- α	NR	NR	Inhibited ¹⁵	Inflammatory ^{30,38} in transgenics
TNF- α blockade	Exacerbated ⁶⁷ sTNFR1-IgG	Exacerbated ⁶⁸ Anti-TNF- α	Inhibited ⁶	Gene knockouts show delayed or inhibited disease ^{13,14}
TGF- β	NR	No effect on MS Nephrotoxic ⁶⁹	Inhibited/alleviated ^{35,36}	Inflammatory ⁴⁴ in transgenics

⁴Treatment with IFN γ inducer Poly-ICLC (ref. 64) ⁶IFN- β suppresses IFN- γ production⁶², and this has been invoked as one possible cause of therapeutic benefit. IFN- β may also act via inhibition of leukocyte extravasation. RR, relapsing/remitting. SP, secondary progressive.

We regret this error.