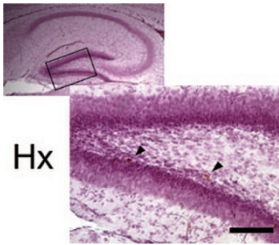
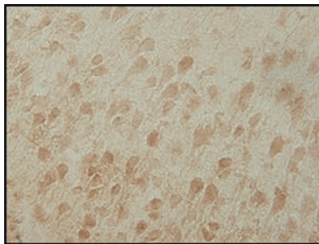


EDITOR'S FOCUS



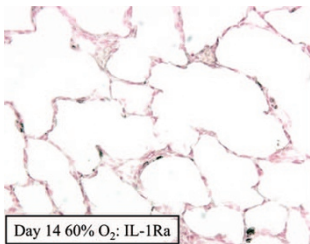
Employing prolonged exposure to sublethal postnatal hypoxia in mice sex-specific differences which included a greater hippocampal volume loss and disruption of forebrain myelination was more pronounced in males.

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Low dose lipopolysaccharide preconditioning reduces hypoxic-ischemia induced neuroinflammation and neuroprotects against long-term behavioral and pathological abnormalities in neonatal rats.

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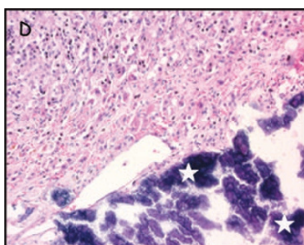
Interleukin-1 receptor antagonism protected against oxygen induced chronic neonatal lung injury.

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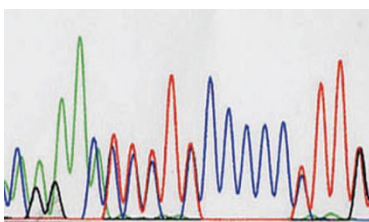
Functional magnetic resonance imaging during natural sleep in full term infants revealed five resting state networks in the absence of external stimuli encompassing sensory, parietal, temporal and prefrontal cortices and the bilateral basal ganglia.

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A defect in biosynthesis of the mitochondrially encoded subunits of the OXPHOS complex with no DNA mutations revealed a compensatory increase in ribosomal 12S and 16S in response to low translation. This defect led to lactic acidosis and bilateral calcifications in the adrenal medulla during the neonatal period.

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Insulin resistance in obese children was associated with a single nucleotide polymorphism (SNP) 276 G>T at the adiponectin gene and the n-6/n-3 long chain polyunsaturated fatty acids profile. Similar studies involving association of polymorphisms in the A allele of the uncoupling protein (UCP)-2 -866G>A proved protective against cardiovascular risk (see Labayen *et al.*, page 350), and the -174G>C interleukin-6 proved protective against juvenile onset type I diabetic late complications of retinopathy and nephropathy (see Myśliwska *et al.*, page 341).

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