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Transcriptomic Analysis Highlights Time-specific Embryonic Adaptation of Mice to the Lack of PrP

Submitted by Emmanuel Lemoine on Thu, 02/12/2015 - 12:59

Titre	Transcriptomic Analysis Highlights Time-specific Embryonic Adaptation of Mice to the Lack of PrP
Type de publication	Article de revue
Auteur	Khalife, Manal [1], Young, Rachel [2], Chadi, Sead [3], Le Guillou, Sandrine [4], Lefevre, Lucas [5], Tilly, Gaëlle [6], Bitton, Frédérique [7], Martin-Magniette, Marie-Laure [8], Soubigou-Taconnat, Ludivine [9], Balzergue, Sandrine [10], Vilotte, Marthe [11], Passet, Bruno [12], Béringue, Vincent [13], Renou, Jean-Pierre [14], Le Provost, Fabienne [15], Laude, Hubert [16], Vilotte, Jean-Luc [17]
Editeur	Taylor & Francis
Type	Article scientifique dans une revue à comité de lecture
Année	2010
Langue	Anglais
Date	2010/07/01
Numéro	3
Volume	4
Titre de la revue	Prion
ISSN	1933-6896
Résumé en anglais	The physiological function of the PrP remains largely elusive. Its invalidation does not affect mouse survival and induces subtle phenotypes. To potentially assess this conundrum, we first comparatively analyzed the adult brain transcriptome of wild-type mice with that of transgenic mice invalidated at this locus either at the zygotic (Zürich PrP0/0 mice) or adult stages (NFH-Cre-Lox mice). Only subtle differences could be evidenced in the adult brains following microarray and QPCR analyses. When performed at an early adult stage, neuronal Prnp disruption appeared to sequentially induce an oxidative stress response and a nervous system remodeling, but it involved a limited number of only slightly modified genes. In sharp contrast, analysis at early embryonic stages, 7.5 and 8.5 dpc, just after the suspected normal time set of the Prnp locus activation, led to a transient perturbation of the transcriptome involving a larger number of genes and pointing to potential pathways related to the PrP physiological function. Overall, our data suggests an early adaptation of the mouse to the potentially detrimental lack of PrP during embryogenesis while its presence is less influential or redundant at later developmental stages.
URL de la notice	http://okina.univ-angers.fr/publications/ua7712 [18]
DOI	10.4161/pri.12773 [19]
Lien vers le document	http://dx.doi.org/10.4161/pri.12773 [19]

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