Aluminum Ingestion Promotes Colorectal Hypersensitivity in Rodents

Titre	Aluminum Ingestion Promotes Colorectal Hypersensitivity in Rodents
Type de publication	Article de revue
Auteur	Esquerre, Nicolas [1], Basso, Lilian [2], Dubuquoy, Caroline [3], Djouina, Madjid [4], Chappard, Daniel [5], Blanpied, Catherine [6], Desreumaux, Pierre [7], Vergnolle, Nathalie [8], Vignal, Cécile [9], Body-Malapel, Mathilde [10]
Editeur	Elsevier
Type	Article scientifique dans une revue à comité de lecture
Année	2019
Langue	Anglais
Date	2019
Numéro	1
Pagination	185-196
Volume	7
Titre de la revue	Cellular and Molecular Gastroenterology and Hepatology
ISSN	2352-345X
Mots-clés	Mast Cells [11], PAR2 [12], Risk Factors [13], Visceral Hypersensitivity [14]
Résumé en anglais	Background & Aims: Irritable bowel syndrome (IBS) is a multifactorial disease arising from a complex interplay between genetic predisposition and environmental influences. To date, environmental triggers are not well known. Aluminum is commonly present in food, notably by its use as food additive. We investigated the effects of aluminum ingestion in rodent models of visceral hypersensitivity, and the mechanisms involved. Methods: Visceral hypersensitivity was recorded by colorectal distension in rats administered with oral low doses of aluminum. Inflammation was analyzed in the colon of aluminum-treated rats by quantitative PCR for cytokine expression and by immunohistochemistry for immune cells quantification. Involvement of mast cells in the aluminum-induced hypersensitivity was determined by cromoglycate administration of rats and in mast cell-deficient mice (Kit). Proteinase-activated receptor-2 (PAR2) activation in response to aluminum was evaluated and its implication in aluminum-induced hypersensitivity was assessed in PAR2 knockout mice. Results: Orally administered low-dose aluminum induced visceral hypersensitivity in rats and mice. Visceral pain induced by aluminum persisted over time even after cessation of treatment, reappeared and was amplified when treatment resumed. As observed in humans, female animals were more sensitive than males. Major mediators of nociception were up-regulated in the colon by aluminum. Activation of mast cells and PAR2 were required for aluminum-induced hypersensitivity. Conclusions: These findings indicate that oral exposure to aluminum at human dietary level reproduces clinical and molecular features of IBS, highlighting a new pathway of prevention and treatment of visceral pain in some susceptible patients.

URL de la notice	http://okina.univ-angers.fr/publications/ua18917 [15]
DOI	10.1016/j.jcmgh.2018.09.012 [16]
Lien vers le document	https://www.cmghjournal.org/article/S2352-345X [17](18)30134-6/fulltext
Titre abrégé	Cell Mol Gastroenterol Hepatol
Identifiant (ID) PubMed	30534582 [18]

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