Quantification of the Calcification Phenotype of Abcc6-Deficient Mice with Microcomputed Tomography

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Résumé en anglais	Pseudoxanthoma elasticum in humans and dystrophic cardiac calcification in mice are heritable disorders characterized by dystrophic calcification of soft connective tissues related to the defective function of the ABCC6 (human)/Abcc6 (mouse) transporter. Of particular interest is the finding of calcified vibrissae in Abcc6−/− mice, which facilitates the study of dystrophic calcification by histological techniques. We aimed to determine whether mice prone to dystrophic cardiac calcification (C3H/HeOuJ and DBA/2J strains) presented similar vibrissae changes and to evaluate the value of microcomputed tomography to quantify the extent of mystacial vibrissae calcifications. These calcifications were absent in DBA/2J and C57BL/6J control mice. In both Abcc6−/− and C3H/HeOuJ mice, calcifications progressed in a caudal-rostral direction with aging. However, the calcification process was delayed in C3H/HeOuJ mice, indicating an incomplete expression of the calcification phenotype. We also found that the calcification process in the cephalic region was not limited to mystacial vibrissae but was also present in other periorbital sensorial vibrissae. The vibrissae calcification was circular and encompassed the medial region of the vibrissae capsule, adjacent to the ring and cavernous sinuses (the areas adjacent to blood and lymphatic vessels). Collectively, our findings confirm that Abcc6 acts as an inhibitor of spontaneous chronic mineralization and that microcomputed tomography is a valuable noninvasive tool for the assessment of the calcification phenotype in Abcc6-deficient mice.
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