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to address the question of whether the associations between epicardial and subcutaneous inflammatory profiles at the genome level were conserved at the proteome level, we compared protein expression levels in a subset of paired adipose tissue samples. as shown in figure 6, we detected the expression of 797 proteins in epicardial and subcutaneous adipose tissue samples, of which, 692 proteins were detected in both adipose depots. of these, 297 proteins were upregulated and 395 proteins were downregulated in epicardial versus subcutaneous adipose tissues. download figure download powerpoint figure 6. integrated coverage of proteins in paired samples of epicardial and subcutaneous adipose tissue (11 paired samples). bars indicate integrated coverage of proteins in 11 paired samples. the p2x4r was also expressed in iems. as shown in figure 5, iems expressed markers of smooth muscle cells ( calponin, smooth muscle myosin heavy chain, and sm22 $\alpha$ ) and were strongly immunoreactive for p2x4r. the presence of p2x4r in iems was confirmed by the high ca<sup>2+</sup> sensitivity of iem contractility and the reduction in contractility with the p2x4r selective antagonist a-317491, as shown in figure 6. in contrast, iems from p2x4r knockout (ko) mice showed no response to a-317491 and no immunoreactivity for p2x4r ( figure 5 ). the p2x4r expression in smooth muscle cells and its modulation by the bk-mediated contractility in iems have not been described in the human or mouse inner ear. to further characterize iems, we examined the expression of various markers of vascular smooth muscle cells ( table 5 ). the majority of iems were immunoreactive for calponin (76%), sm22 $\alpha$  (74%), and smooth muscle myosin heavy chain (63%), which are markers of vascular smooth muscle cells. iems were also immunoreactive for the macrophage marker cd68 (51%) and the monocyte/macrophage marker monocyte chemoattractant protein-1 (53%), suggesting that iems are composed of both vascular smooth muscle cells and macrophages. these results indicate that iems are vascular smooth muscle cells expressing contractile function, which are composed of vascular smooth muscle cells, macrophages, and neurons. to characterize the function of iems, we examined their contractile response to bk and a-317491, and the expression of p2x4r. as shown in figure 6, contraction of iems to the selective p2x4r agonist bzapf was abolished by a-317491 (1  $\mu$ mol/l) in wt mice. in contrast, iems from p2x4r ko mice showed no response to a-317491 and no immunoreactivity for p2x4r ( figure 5 ). in addition, a-317491 also significantly inhibited the iem contractility in wt mice. taken together, these data suggest that iem contractility is predominantly mediated by p2x4r. download figure download powerpoint figure 6. inhibition of iem contractility by p2x4r antagonist (a-317491). iems from wt or p2x4r ko mice were contracted to the selective p2x4r agonist bzapf (10  $\mu$ mol/l) in the presence or absence of a-317491 (1  $\mu$ mol/l). the data are expressed as means  $\pm$  sem. \*p

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after adjustment for age, sex, baseline framingham risk score, and chf status at study entry, af was associated with a more than 4-fold increased risk of chf (adjusted hazard ratio, 4.24; 95% confidence interval, 3.06 to 5.82; p 5ec8ef588b

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