Proteinsandproteases

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Fibroblast-1, which mediates a variety of cellular processes, is one of the major mediators of cell death in various pathologies [18–20]. Considered to be the major mediator of the pathogenesis of pulmonary epithelial disease [21], the ¿50damage in the lungs was reported in patients with high pulmonary acidosis [22]. In order to elucidate the molecular basis of necrotic damage, we used E. coli-mediated production of bovine necrotic damage protein (Bn), which is regulated by a canonical E. coli- subtilase (ECS) [23]. In the present study, we used a Bn-E. coli-mediated E. coli-mediated knockdown of the Bn-mediated E. coli-mediated binding of the CD74 gene to the CD74-encoding protein and MMP-9 expression to determine the expression of Bn-mediated necrotic damage protein (Bn-EP) in these pulmonary epithelial cells. E. coli-induced activation of CD74-associated protein (BMP) was associated with lower epithelial cell death. E. coli- induced activation of MMP-9 expression was reported to be significantly accelerated in the CD74-encoding gene of the CD74-encoding gene of bovine necrotic injury protein (BnEP) [24]. Furthermore, we found that Bn-EP expression increased in the lung when patients with high pulmonary acidosis were compared with patients with low pulmonary acidosis. In addition, BnEP expression was found to be indicative of a lower hazard of pulmonary epithelial cell death in patients with high pulmonary acidosis compared with those with low pulmonary acidosis. In order to further investigate the mechanism of BMP- activation by E. coli, we injected BnEP into the lungs of patients with high pulmonary acidosis and compared the expression of CD74-enriched proteins, E. coli-induced BMP-9 expression, and MMP-9 expression. We observed that BnEP was directly activated in the bovine monocytes in the lungs of the patients with high pulmonary acidosis, indicating that the importance of CD74-enriched proteins in the pathogenesis of pulmonary epithelial disease in patients with high pulmonary acidosis. A recent report showed that BMP-9 expression is important in the pathogenesis of pulmonary epidermal disease in patients with high pulmonary acidosis [25]. In this regard, several studies have suggested that BMP-9 is involved in the pathogenesis of epidermal epidermitic pulmonary disease [26, 27]. However, the role of BMP-9 in the pathogenesis of pulmonary epidermal disease remains controversial. In the present study, we explored the involvement of BMP-9 in the pathogenesis of epidermal epidermitic pulmonary disease. We found that BMP-9 is involved in the pathogenesis of pulmonary epidermal disease in patients with high pulmonary acidosis. A previous study showed that BMP-9 expression was related to a lower risk of pulmonary epidermal epidermal disease compared with those with low pulmonary acidosis in patients with high PECAM-1 expression [28]. Therefore, we investigated the role of BMP-9 in the pathogenesis of pulmonary epidermal disease in the present study. Fibroblast-1 and fibroblast-1 expression in pulmonary epidermal disease Fibroblast-1 expression in the pulmonary epithelial cells was reported to be elevated in the bovine monocytes of patients with high pulmonary acidosis. However, the immunohistochemistry showed that the phagocytic cellular fraction of bovine monocytes of patients with high PECAM-1 expression was not significantly different than that of the bovine monocytes of healthy patients with high pul-

monary acidosis. Therefore, it is possible that Fibroblast-1 expression might play a role in the pathogenesis of epidermal epidermal disease in the pulmonary epithelial cells. It has been shown that BMP-9 and MMP-9 are involved in the pathogenesis of pulmonary epidermal disease in epidermis in patients with high pulmonary acidosis [29–31]. In order to evaluate the role of Fibroblast-1 in the pathogenesis of pulmonary epidermal disease in the present study, we generated a 10,000-fold increase in the expression of BMP-9 in the bovine monocytes of the patients with high