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LSC 3 | 12:00 - 1:00PM



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## Role of carboxypeptidase B2 (TAFI)

Carboxypeptidase B2 (TAFI) is a metalloprotease that removes C-terminal basic amino acids, thereby inactivating pro-inflammatory mediators such as complement C3a and C5a as well as reducing the rate of fibrinolysis. Specific inhibitors have been developed that increase the rate of fibrinolysis. Mice that CPB2 deficient (Cpb2<sup>-/-</sup>) have no phenotype until challenged. In these mice fibrin, C3a and C5a have been shown to be physiological substrates in models in which thrombosis is not directly induced. We have recently compared Cpb2<sup>-/-</sup> mice to mice deficient in the other plasma basic carboxypeptidase, CPN, (Cpn<sup>-/-</sup> mice) in a model of hemolytic uremic syndrome and found that the Cpb2<sup>-/-</sup> mice have exacerbated disease compared to Cpn<sup>-/-</sup> mice which have worse disease than wild type. This data suggests that CPN may be responsible for inactivating C3a and C5a generated by turnover of the alternative complement pathway, CPB2 is responsible for inactivating inflammatory mediators at local sites of damage and repair.

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