suggest a modified version of the previously published Table 1 underscoring the existence of this very interesting pattern (Table 1).


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Corin: a serine protease

To The Editor: We read with great interest the commentary in a recent issue of Kidney International, in which Klein1 provided an insightful discussion on potential roles of corin and atrial natriuretic peptide (ANP) in regulating sodium reabsorption in nephrotic syndrome.2 In this article, however, corin was indicated as a ‘serine/threonine’ protease.1 Unlike serine/threonine protein kinases, which phosphorylate the OH group of serine or threonine residues, corin is a serine protease, that is, proteolytic enzyme that cleaves peptide bonds within a polypeptide.3 The catalytic activity of corin is mediated by a reactive Ser residue that is part of the catalytic triad (Asp, His, and Ser) in its protease domain (Figure 1). As a trypsin-like serine protease, corin has a substrate specificity favoring Arg or of ‘interstitial nephritis accompanying glomerulonephritis and vasculitis’.


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The Authors Reply: We appreciate the interest of Airoldi et al.1 in our review ‘Acute interstitial nephritis’.2 The uncommon clinical cases on which they comment (interstitial vasculitis without glomerular involvement) have a great clinical and pathogenic interest. However, their inclusion as another type of acute interstitial nephritis (AIN) is, in our opinion, debatable. AIN is usually defined as a syndrome with different etiologies and is characterized by the presence of inflammatory infiltrates and edema within the interstitium, whereas glomeruli and vessels are distinctly normal.2,3 This definition is useful to distinguish true AIN from those interstitial infiltrates and edema that very frequently accompany many forms of glomerulonephritis and vasculitis. For instance, in the study of Hauer et al.,4 interstitial infiltrates were found in 92% of 173 patients with antineutrophil cytoplasmic antibody-associated vasculitis. The distinction between true AIN and interstitial inflammation accompanying other renal diseases (glomerulonephritis, vasculitis, and acute tubular necrosis), although difficult to establish in some cases, is convenient not only for academic purposes: prognosis and treatment of these entities is very different. For all these reasons, we would prefer ‘interstitial vasculitis’ as the most appropriate and precise diagnosis for the patients reported by Airoldi et al. To be included among the different causes of AIN, they could be classified within a subgroup (Figure 1) A molecular model of the corin protease domain. The protease domain of corin is shown in a three-dimensional structure similar to that of chymotrypsin. β-Sheets and α-helices are shown in yellow and red, respectively. The catalytic residues Asp, His, and Ser are shown in purple.
Lys residues. Consistently, corin cleaves human pro-ANP at Arg98, but not at 'serine 124', as described in the article.