Letter to the Editor

Use of neutrophil gelatinase-associated lipocalin (NGAL) in CF

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We read with interest the published review by Nazareth and Walshaw [1] in relation to renal disease in cystic fibrosis (CF).

The authors correctly note the use of markers including neutrophil gelatinase-associated lipocalin (NGAL) and kidney injury molecule 1 (KIM-1) as indicators of acute kidney injury (AKI). It is subsequently stated that ‘unfortunately NGAL is also produced in response to damaged epithelial cells in the lung, effectively excluding its use in the CF condition.’ However, the source of this reference, Devarajan [2] makes no such mention of NGAL in the lung.

There is sparse literature describing NGAL, also known as lipocalin-2, in the lung. Soni et al. [3] state variable degrees of NGAL gene expression that is demonstrated in human lung tissue. The authors describe its role as a growth factor with elevated levels identified in sputum of patients with asthma and chronic obstructive airways disease and in the bronchial secretions from patients with subclinical emphysema. There is no reference to cystic fibrosis.

A recent study by Zughaier et al. [4] sought to determine whether NGAL could serve as a maker of acute pulmonary exacerbation in CF. The study found that serum NGAL levels were elevated in patients with CF compared to healthy controls. Although, there was no difference in those with CF and stable disease compared to those with an exacerbation of CF, it is acknowledged that this may affect the positive and negative predictive values of NGAL and the clinical utility of NGAL in CF, compared to non-CF populations.

We are unaware of any reason that use of NGAL in CF is precluded. If NGAL is used as a urinary biomarker to detect renal impairment, interference with a urinary assay from lung expression is not expected as NGAL is a large molecule and unlikely to cross the glomerular barrier. Indeed, one French study (MUCO-IRC) [5] is currently recruiting patients with CF post lung transplant and investigating use of NGAL as a biomarker in chronic renal disease. Until data such as these are available, we believe it is premature to dismiss the use of NGAL as a marker of renal dysfunction in CF. Any biomarker with the potential for early detection of renal dysfunction should be assessed carefully before dismissing its utility in CF.

References