The Association for Clinical Biochemistry

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ACB statement on laboratory assessment of kidney function

In 2006 clinical laboratories throughout the NHS introduced a blood test called estimated glomerular filtration rate (eGFR) as a measure of kidney function in adults. eGFR is calculated from serum creatinine concentration, which is a waste product from muscle that is excreted by the kidneys and reflects kidney function. The creatinine concentration in blood is also influenced by several other factors including age, gender and race that affect muscle bulk. eGFR adjusts the serum creatinine concentration for these influences and provides a way of reporting kidney function in physiological units, enabling kidney disease to be identified and staged according to the international disease classification system. Kidney disease is known to increase cardiovascular risk. Early detection and treatment of kidney disease should enable the slowing or prevention of worsening kidney function in many people. Since the introduction of eGFR approximately 4% of the UK population have been identified as having kidney disease: it is anticipated that improved blood pressure control and other interventions amongst these individuals will improve their outcomes. There has been an increase in referral for renal consultations in secondary care, but this is probably appropriate. There has also been a fall in the proportion of patients presenting for the first time at dialysis units with very advanced kidney disease, probably as a result of earlier detection.

The results of all diagnostic tests are subject to a degree of uncertainty that is largely a consequence of natural biological variation, both within and between individuals, and the limitations of measurement technology. This is true for both creatinine measurement and estimated GFR. This uncertainty is greatest where eGFR is close to that expected in healthy people. Unfortunately, the different methods used for creatinine measurement in laboratories exacerbate this problem, causing further (and potentially avoidable) variability in eGFR. The Association for Clinical Biochemistry (ACB) believes that variation in eGFR could be reduced by laboratories improving creatinine measurement across the UK.[1] This is in agreement with the National Institute for Health and Clinical Excellence (NICE) guideline on chronic kidney disease. This would be best achieved by laboratories adopting specific enzymatic assays for creatinine measurement.

Clinical users should be made aware that significant changes in estimated glomerular filtration rate might occur when a change in serum creatinine method is made. Examples of users likely to require well documented notification include renal physicians, oncologists, pharmacists, paediatricians and general practitioners.

[1] Technical footnote: NICE recommended that laboratories use assays that are specific and zero-biased compared with isotope-dilution mass spectrometry (IDMS) reference methods (www.nice.org.uk, clinical guideline 73, 2008). The availability of JCTLM listed reference measurement procedures and a standardised reference material (SRM 967) for creatinine means that good interlaboratory agreement is achievable if specific assays are used. In 2009 most major reagent and instrument manufacturers claimed that their assays were traceable to IDMS, although it must be appreciated that this does not necessarily mean that they all produce IDMS equivalent results. Furthermore, the non-specificity of the Jaffe reaction means that this can never hold true across all patient samples using such methods. Ideally laboratories should move to enzymatic assays for creatinine measurement: as a minimum, the use of traditional kinetic Jaffe assays should cease and be replaced with ‘compensated’ Jaffe methods. The universal adoption of more specific creatinine assays would also improve the early detection of acute kidney injury.