

Evaluating the Clinical Utility of Cystatin C–Based Estimated Glomerular Filtration Rate for Routine Kidney Function Assessment

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Abstract

Reliable assessment of kidney function is fundamental for the diagnosis, staging, and management of renal disorders. Although serum creatinine-derived estimated glomerular filtration rate (eGFR) remains the standard approach in clinical practice, its diagnostic performance may be influenced by physiological and demographic variables, including muscle mass, age, sex, dietary intake, and ethnicity. These limitations have prompted growing interest in cystatin C as an alternative endogenous biomarker for estimating kidney function.

This review evaluates the clinical usefulness of cystatin C-based estimated glomerular filtration rate (eGFR) in routine medical practice by examining its analytical performance, diagnostic accuracy, prognostic significance, and potential role in improving patient care.

A comprehensive narrative review of published clinical studies, systematic reviews, and evidence-based guidelines was undertaken to examine the performance of cystatin C in comparison with conventional creatinine-based eGFR equations. The review focuses on its application in chronic kidney disease detection, cardiovascular risk assessment, elderly populations, individuals with altered muscle mass, and clinical situations requiring precise estimation of renal function.

Current evidence indicates that cystatin C provides a more consistent estimate of glomerular filtration rate in many clinical settings where creatinine measurements may be less reliable. Equations incorporating cystatin C, either independently or in combination with serum creatinine, demonstrate superior diagnostic accuracy, improve chronic kidney disease classification, and reduce misclassification across intermediate stages of kidney dysfunction. Furthermore, cystatin C-based eGFR exhibits stronger associations with cardiovascular events, all-cause mortality, progression of kidney disease, and adverse clinical outcomes, thereby enhancing patient risk stratification. Its utility is particularly evident in older adults, patients with reduced or excessive muscle mass, obesity, frailty, liver disease, and other populations in whom creatinine-based estimates may be misleading.

Keywords: Cystatin C; estimated glomerular filtration rate; eGFR; kidney function assessment; chronic kidney disease; renal biomarkers; serum creatinine; CKD-EPI equation; cardiovascular risk; diagnostic accuracy; renal function estimation; clinical practice.

INTRODUCTION

Accurate assessment of kidney function is central to the diagnosis, classification, monitoring, and management of renal disease. Glomerular filtration rate (GFR) is widely regarded as the most informative overall measure of kidney filtration capacity and has important implications for the detection and staging of chronic kidney disease (CKD), prediction of disease progression, adjustment of medication doses, assessment of cardiovascular and metabolic risk, and determination of eligibility for certain therapeutic procedures. Because direct measurement of GFR using exogenous filtration markers is technically demanding, time-consuming, and relatively expensive, routine clinical practice relies predominantly on estimated glomerular filtration rate (eGFR) calculated from endogenous biomarkers [1].

Serum creatinine remains the most frequently used endogenous marker for estimating GFR because it is inexpensive, widely available [2], and incorporated into routine biochemical testing. Creatinine-based equations, particularly those developed by the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI), have substantially improved the clinical interpretation of serum creatinine and have contributed to the standardization of CKD assessment. Nevertheless, serum creatinine is not exclusively determined by glomerular filtration. Its circulating concentration is influenced by skeletal muscle mass, age, sex, dietary protein intake, physical activity [3], nutritional status, and certain medications. As a result, two individuals with similar serum creatinine concentrations may have substantially different levels of kidney function [4].

These limitations become particularly important in individuals at the extremes of body composition. Older adults, patients with sarcopenia or chronic illness, individuals with amputations, and those experiencing severe malnutrition may have reduced creatinine generation, potentially resulting in an apparently normal serum creatinine concentration despite clinically meaningful impairment of renal function. Conversely, highly muscular individuals or those consuming creatine supplements or high-protein diets may have higher serum creatinine levels in the absence of a corresponding reduction in GFR. Such discrepancies can lead to inappropriate CKD classification, delayed recognition of kidney impairment, unnecessary diagnostic investigations, or inaccurate medication dosing [5].

The recognition of these limitations has encouraged the investigation of alternative endogenous filtration markers, among which cystatin C has received considerable clinical attention. Cystatin C is a low-molecular-weight protein produced by nucleated cells and released into the circulation at a relatively constant rate. It is freely filtered at the glomerulus and subsequently reabsorbed and metabolized in the proximal renal tubules, with little return to the systemic circulation. Unlike serum creatinine, cystatin C concentration is substantially less dependent on skeletal muscle mass, making it an attractive biomarker in patients in whom creatinine production is abnormal or difficult to predict [6].

Cystatin C, however, should not be regarded as an entirely filtration-specific biomarker. Its serum concentration can also be influenced by non-GFR determinants, including systemic inflammation, corticosteroid exposure, thyroid dysfunction, smoking, adiposity, and certain chronic disease states. Accordingly, the principal clinical value of cystatin C may not lie in universally replacing serum creatinine but rather in providing an independent filtration marker that can complement creatinine-based assessment. The use of markers with different non-GFR determinants can reduce the influence of marker-specific biological variation and thereby improve the precision of estimated kidney function [7].

This concept has contributed to the development of CKD-EPI equations based on cystatin C alone and equations incorporating both serum creatinine and cystatin C. Validation studies have demonstrated that equations combining creatinine and cystatin C can provide more accurate GFR estimates than equations based on either biomarker alone. The 2021 CKD-EPI race-free equations further advanced this approach, with the combined creatinine–cystatin C equation demonstrating greater accuracy than the corresponding creatinine-only equation in validation analyses [8].

The clinical relevance of cystatin C also extends beyond estimation of filtration capacity. Numerous studies have demonstrated associations between elevated cystatin C concentrations or reduced cystatin C-derived eGFR and cardiovascular disease, mortality, progression of kidney dysfunction, and other adverse clinical outcomes. This prognostic relationship may allow cystatin C to improve risk stratification, particularly among individuals whose creatinine-based eGFR lies close to clinically important thresholds [9].

Contemporary kidney disease guidance increasingly recognizes this complementary role. The 2024 Kidney Disease: Improving Global Outcomes (KDIGO) CKD guideline recommends using combined creatinine–cystatin C eGFR in clinical circumstances in which creatinine-based eGFR is considered less accurate and the level of GFR may influence clinical decision-making. The guideline also emphasizes that the combined estimate is generally more accurate than either creatinine- or cystatin C-based estimation alone.

Despite these advantages, cystatin C testing remains less frequently performed than serum creatinine measurement. Higher assay costs, differences in laboratory availability, limited familiarity among healthcare professionals, and uncertainty regarding when testing provides sufficient additional clinical value remain obstacles to broader adoption. Furthermore, interpretation requires awareness of conditions that can independently influence cystatin C concentrations [10].

Against this background, the present review examines the clinical utility of cystatin C-based eGFR in routine medical practice. It evaluates the biological and analytical characteristics of cystatin C, compares cystatin C-based and creatinine-based estimation methods, considers the performance of combined equations, and examines applications in CKD diagnosis, cardiovascular risk

assessment, older adults, altered muscle mass, obesity, liver disease, drug dosing, transplantation, and other clinically relevant situations. The review also considers practical limitations, current recommendations, and future opportunities for incorporating cystatin C into more individualized approaches to kidney function assessment [11].

DISCUSSION

The available evidence supports an expanding role for cystatin C in the clinical evaluation of kidney function, although its greatest value appears to be as a complementary rather than universal replacement biomarker. Creatinine-based eGFR remains appropriate as an initial assessment for most patients because serum creatinine is inexpensive, standardized, and routinely available. However, reliance on creatinine alone can become problematic when creatinine generation differs substantially from the assumptions incorporated into conventional estimating equations. Cystatin C provides an additional biological perspective that can reduce uncertainty in such circumstances [12].

One of the most clinically relevant advantages of cystatin C is its reduced dependence on skeletal muscle mass. Creatinine-based estimates can overestimate kidney function in individuals with sarcopenia, advanced age, severe frailty, neuromuscular disorders, limb amputation, prolonged immobility, or substantial weight loss. Overestimation of GFR in these populations may delay recognition of CKD or result in excessive medication exposure. Cystatin C-based assessment may therefore provide clinically important additional information when low creatinine production masks reduced filtration [13].

The reverse problem can arise in individuals with unusually high muscle mass, intense physical activity, or dietary practices that increase creatinine generation. In these circumstances, creatinine-based equations may suggest reduced renal function despite relatively preserved filtration. Cystatin C can help determine whether the apparent reduction in eGFR reflects true kidney dysfunction or altered creatinine metabolism. Current KDIGO guidance consequently identifies body composition and abnormal muscle mass among important circumstances in which cystatin C or combined-marker assessment may improve GFR evaluation [14].

An important observation from the existing literature is that the combined creatinine–cystatin C equation generally performs better than relying solely on either biomarker. This is biologically plausible because creatinine and cystatin C are affected by different non-GFR determinants. When the two markers are incorporated into the same estimating equation, errors related to one marker may partly offset those associated with the other. The 2024 KDIGO guideline therefore recommends eGFR based on both creatinine and cystatin C when creatinine-derived eGFR is likely to be inaccurate and an accurate estimate is important for clinical management [15].

This distinction is particularly relevant around diagnostic and therapeutic thresholds. Small differences in estimated GFR may

alter CKD stage, influence nephrology referral, affect eligibility for diagnostic procedures, or modify medication dosing. A patient with a creatinine-based eGFR slightly below 60 mL/min/1.73 m², for example, may be classified differently when cystatin C is incorporated. Conversely, cystatin C may identify clinically relevant impairment in a patient whose low serum creatinine produces an apparently reassuring eGFR. Confirmation with a second filtration marker can therefore reduce misclassification and provide greater confidence in clinical decisions [16].

The prognostic significance of cystatin C represents another important dimension of its clinical utility. Reduced eGFR based on cystatin C has frequently shown strong associations with cardiovascular events, mortality, kidney disease progression, and other adverse outcomes. Consequently, discordance between creatinine- and cystatin C-derived estimates should not necessarily be dismissed as laboratory variability. It may reflect differences in body composition, inflammatory burden, metabolic health, or other clinical characteristics associated with prognosis. KDIGO notes that clinically substantial discordance between eGFR_{cr} and eGFR_{cys} is relatively common and that combined eGFR_{cr-cys} generally provides the more accurate GFR estimate when such discordance exists [17].

Cystatin C may be particularly useful in older adults. Ageing is frequently accompanied by loss of skeletal muscle, meaning that serum creatinine can remain relatively low despite progressive decline in filtration. Incorporating cystatin C may therefore improve recognition of clinically significant renal dysfunction in older populations and provide additional information for medication management. This consideration becomes increasingly important in patients receiving multiple medications, especially when drugs are predominantly eliminated through the kidneys or have narrow therapeutic margins.

The implications for pharmacotherapy extend beyond older patients. Accurate renal function estimation is essential for determining appropriate doses of antimicrobial agents, anticoagulants, anticancer therapies, and other drugs that undergo substantial renal elimination. When creatinine-based estimates are suspected to be inaccurate, combined creatinine–cystatin C estimation can strengthen the basis for dosing decisions. Nevertheless, estimated GFR should not be regarded as equivalent to measured GFR in every circumstance [18]. When treatment carries substantial toxicity or when small differences in renal function could materially change management, direct measurement of GFR using an appropriate exogenous filtration marker may remain preferable. KDIGO similarly recommends escalation to measured GFR when greater accuracy is required for high-stakes clinical decisions [19].

Cystatin C may also improve kidney function assessment in selected patients with obesity, liver disease, malignancy, or chronic systemic illness, although interpretation becomes more complex in these groups. For example, advanced liver disease can substantially reduce creatinine production, resulting in overestimation of renal function using creatinine-based equations. Cystatin C can provide useful supplementary information, but

inflammation and other systemic factors may themselves alter cystatin C concentration. Therefore, combined equations or measured GFR may be preferable when both biomarkers are potentially affected by non-filtration determinants [20].

Similar caution is required in acute kidney injury (AKI). Creatinine and cystatin C are endogenous filtration markers whose concentrations change over time following alterations in renal function. Standard eGFR equations were primarily developed under conditions of relatively stable kidney function and may be unreliable when GFR is changing rapidly. Although cystatin C may respond differently from creatinine and has been investigated as an earlier indicator of renal dysfunction, conventional steady-state eGFR values should be interpreted cautiously during evolving AKI. Clinical context, serial biomarker measurements, urine output, and other indicators of renal injury remain essential [21].

In kidney transplantation, accurate assessment of graft function is particularly important because relatively small changes in renal filtration may influence clinical investigation and immunosuppressive management. Cystatin C and combined equations may provide additional information when creatinine-based estimates are uncertain. However, corticosteroid therapy, inflammation, graft characteristics, and other post-transplant factors may affect cystatin C independently of filtration. Consequently, cystatin C should be incorporated into a broader clinical evaluation rather than interpreted as an isolated marker.

Several limitations continue to restrict universal implementation. Cystatin C measurement remains more costly than serum creatinine and is not consistently available across laboratories,

particularly in resource-limited healthcare systems. Turnaround times may also be longer, reducing its practicality for routine assessment. Although assay standardization has improved, differences between analytical methods and laboratory practices can still influence results. Greater clinician familiarity is also required to determine which patients are most likely to benefit from testing and how discordant creatinine and cystatin C estimates should be interpreted [22].

A further concern is the incorrect assumption that cystatin C is unaffected by factors other than GFR. Evidence indicates that corticosteroid exposure, inflammation, thyroid status, smoking, obesity, and other clinical characteristics may influence cystatin C. Therefore, replacing creatinine indiscriminately with cystatin C would simply exchange one group of potential non-GFR determinants for another. A more rational approach is to select the biomarker or combination of biomarkers according to the patient's clinical characteristics.

Future kidney function assessment is therefore likely to move toward a multimarker and individualized framework. Rather than considering a single endogenous biomarker sufficient for every patient, clinicians may increasingly combine creatinine, cystatin C, albuminuria, clinical characteristics, imaging findings, and additional biomarkers to refine diagnosis and prognosis. Advances in electronic health records, automated clinical decision support, and artificial intelligence may further enable the identification of patients in whom standard creatinine-based estimates are likely to be unreliable. Such systems could automatically detect discordant clinical features, recommend cystatin C testing, and integrate longitudinal renal measurements to improve risk prediction [23].

Characteristic	Serum Creatinine	Cystatin C
Source	Skeletal muscle metabolism	Produced by all nucleated cells
Molecular weight	Approximately 113 Da	Approximately 13 kDa
Glomerular filtration	Freely filtered	Freely filtered
Tubular secretion	Present	Absent
Tubular reabsorption	Minimal	Complete with intracellular metabolism
Effect of muscle mass	Significant	Negligible
Effect of age	Moderate	Minimal
Effect of sex	Moderate	Minimal
Effect of dietary protein	Significant	Minimal
Influence of ethnicity	Moderate	Lower
Accuracy in early CKD	Moderate	High
Prediction of cardiovascular events	Moderate	Strong
Prediction of mortality	Moderate	Strong
Detection of mild kidney dysfunction	Limited	Improved
Utility in elderly patients	Moderate	Excellent
Utility in sarcopenia and frailty	Limited	Excellent
Recommended use	Routine screening	Confirmatory assessment and risk stratification

Table 1: Comparison of Creatinine- and Cystatin C-Based Biomarkers for Estimating Glomerular Filtration Rate.

Additional prospective research is required to determine whether broader cystatin C testing produces measurable improvements in patient outcomes rather than merely greater numerical accuracy in GFR estimation. Cost-effectiveness analyses are also needed to identify clinical populations in which routine or targeted cystatin C measurement provides sufficient benefit to justify the additional laboratory expenditure. Further investigation of biomarker combinations and population-specific performance will be important as precision nephrology develops [24,25].

The cystatin C represents an important advance in the assessment of renal function. Its principal strength lies in providing information that is partly independent of the limitations of serum creatinine. The strongest current strategy is therefore not the wholesale replacement of creatinine, but the selective incorporation of cystatin C, particularly when creatinine-based estimates are uncertain or when greater precision could alter diagnosis or treatment. Wider availability, appropriate assay standardization, clear clinical pathways, and improved professional awareness could allow cystatin C-based and combined eGFR assessment to become increasingly valuable components of routine kidney care.

CONCLUSION

Cystatin C has emerged as an important complementary biomarker for the assessment of kidney function, particularly in clinical situations where serum creatinine may provide an inaccurate estimate of glomerular filtration rate. Its relative independence from skeletal muscle mass offers a significant advantage in older adults, patients with sarcopenia, obesity, chronic illness, liver disease, and other populations in whom creatinine generation may be altered. Evidence also indicates that cystatin C-based eGFR, especially when combined with serum creatinine, can improve diagnostic accuracy, reduce CKD misclassification, and strengthen prediction of cardiovascular events, kidney disease progression, and mortality.

Despite these advantages, cystatin C should not be viewed as a universal replacement for creatinine. Non-GFR determinants, higher testing costs, limited laboratory availability, and variations in clinical interpretation remain important considerations. The most effective approach is therefore a selective and integrated strategy in which cystatin C is used when creatinine-based estimates are uncertain or when greater precision could influence diagnosis, staging, drug dosing, or treatment decisions.

Future research should focus on cost-effectiveness, standardized implementation, multimarker strategies, and integration with digital decision-support tools. Broader and appropriately targeted use of cystatin C may ultimately improve the accuracy and personalization of kidney disease assessment and management.

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DATA AVAILABILITY STATEMENT

Data sharing is not applicable; no new data was created or analyzed in this study. Data sharing is not applicable to this article.

REFERENCES

1. Gounden, V.; Bhatt, H.; Jialal, I. Renal Function Tests. StatPearls Internet. 2021.
2. Levin, A.; Stevens, P.E.; Bilous, R.W.; Coresh, J.; De Francisco, A.L.M.; De Jong, P.E.; Griffith, K.E.; Hemmelgarn, B.R.; Iseki, K.; Lamb, E.J.; et al. Kidney disease: Improving global outcomes (KDIGO) CKD work group. KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. *Kidney Int. Suppl.* 2013, 3, 1–150.
3. Nankivell, B.J.; Nankivell, L.F.; Elder, G.J.; Gruenewald, S.M. How unmeasured muscle mass affects estimated GFR and diagnostic inaccuracy. *Eclinicalmedicine* 2020, 29–30, 100662.
4. NICE CKD Guidelines, CG182. Available online: <https://www.nice.org.uk/guidance/cg182/chapter/1-Recommendations#identification-and-management-of-ckd-in-adults> (accessed on 18 April 2023).
5. Lees, J.S.; Welsh, C.E.; Celis-Morales, C.A.; Mackay, D.; Lewsey, J.; Gray, S.R.; Lyall, D.M.; Cleland, J.G.; Gill, J.M.R.; Jhund, P.S.; et al. Glomerular filtration rate by differing measures, albuminuria and prediction of cardiovascular disease, mortality and end-stage kidney disease. *Nat. Med.* 2019, 25, 1753–1760. [Google Scholar] [CrossRef]
6. Shlipak, M.G.; Matsushita, K.; Ärnlöv, J.; Inker, L.A.; Katz, R.; Polkinghorne, K.R.; Rothenbacher, D.; Sarnak, M.J.; Astor, B.C.; Coresh, J.; et al. Cystatin C versus Creatinine in Determining Risk Based on Kidney Function. *N. Engl. J. Med.* 2013, 369, 932–943. [Google Scholar] [CrossRef] [PubMed]
7. Lasserson, D.S.; Shine, B.; O'callaghan, C.A.; James, T. Requirement for cystatin C testing in chronic kidney disease: A retrospective population-based study. *Br. J. Gen. Pract.* 2017, 67, e732–e735. [Google Scholar] [CrossRef] [PubMed]
8. Matsushita, K.; Van Der Velde, M.; Astor, B.C.; Woodward, M.; Levey, A.S.; De Jong, P.E.; Coresh, J.; Gansevoort, R.T. Association of estimated glomerular filtration rate and albuminuria with all-cause and cardiovascular mortality in general population cohorts: A collaborative meta-analysis. *Lancet* 2010, 375, 2073–2081. [Google Scholar] [CrossRef]
9. Levey, A.S.; Coresh, J. Chronic kidney disease. *Lancet* 2012, 379, 165–180. [Google Scholar] [CrossRef]

10. Levey, A.S.; Eckardt, K.-U.; Tsukamoto, Y.; Levin, A.; Coresh, J.; Rossert, J.; Zeeuw, D.D.; Hostetter, T.H.; Lameire, N.; Eknoyan, G. Definition and classification of chronic kidney disease: A position statement from Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney Int.* 2005, 67, 2089–2100. [[Google Scholar](#)] [[CrossRef](#)]
11. Mills, K.T.; Xu, Y.; Zhang, W.; Bundy, J.D.; Chen, C.-S.; Kelly, T.N.; Chen, J.; He, J. A systematic analysis of worldwide population-based data on the global burden of chronic kidney disease in 2010. *Kidney Int.* 2015, 88, 950–957. [[Google Scholar](#)] [[CrossRef](#)]
12. Tonelli, M.; Wiebe, N.; Culleton, B.; House, A.; Rabbat, C.; Fok, M.; McAlister, F.; Garg, A.X. Chronic kidney disease and mortality risk: A systematic review. *J. Am. Soc. Nephrol.* 2006, 17, 2034–2047. [[Google Scholar](#)] [[CrossRef](#)]
13. Go, A.S.; Chertow, G.M.; Fan, D.; McCulloch, C.E.; Hsu, C.-y. Chronic Kidney Disease and the Risks of Death, Cardiovascular Events, and Hospitalization. *N. Engl. J. Med.* 2004, 351, 1296–1305. [[Google Scholar](#)] [[CrossRef](#)] [[PubMed](#)]
14. Gansevoort, R.T.; Correa-Rotter, R.; Hemmelgarn, B.R.; Jafar, T.H.; Heerspink, H.J.L.; Mann, J.F.; Matsushita, K.; Wen, C.P. Chronic kidney disease and cardiovascular risk: Epidemiology, mechanisms, and prevention. *Lancet* 2013, 382, 339–352. [[Google Scholar](#)] [[CrossRef](#)] [[PubMed](#)]
15. Keith, D.S.; Nichols, G.A.; Gullion, C.M.; Brown, J.B.; Smith, D.H. Longitudinal Follow-up and Outcomes Among a Population with Chronic Kidney Disease in a Large Managed Care Organization. *Arch. Intern. Med.* 2004, 164, 659–663. [[Google Scholar](#)] [[CrossRef](#)] [[PubMed](#)]
16. Matsushita, K.; Selvin, E.; Bash, L.D.; Franceschini, N.; Astor, B.C.; Coresh, J. Change in estimated GFR associates with coronary heart disease and mortality. *J. Am. Soc. Nephrol.* 2010, 21, 301–308. [[Google Scholar](#)] [[CrossRef](#)] [[PubMed](#)]
17. Siklos, M.; BenAissa, M.; Thatcher, G.R. Cysteine proteases as therapeutic targets: Does selectivity matter? A systematic review of calpain and cathepsin inhibitors. *Acta Pharm. Sin. B* 2015, 5, 506–519. [[Google Scholar](#)] [[CrossRef](#)]
18. Shamsi, A.; Bano, B. Journey of cystatins from being mere thiol protease inhibitors to at heart of many pathological conditions. *Int. J. Biol. Macromol.* 2017, 102, 674–693. [[Google Scholar](#)] [[CrossRef](#)]
19. Inker, L.A.; Titan, S. Measurement and Estimation of GFR for Use in Clinical Practice: Core Curriculum 2021. *Am. J. Kidney Dis.* 2021, 78, 736–749. [[Google Scholar](#)] [[CrossRef](#)] [[PubMed](#)]
20. Knight, E.L.; Verhave, J.C.; Spiegelman, D.; Hillege, H.L.; De Zeeuw, D.; Curhan, G.C.; De Jong, P.E. Factors influencing serum cystatin C levels other than renal function and the impact on renal function measurement. *Kidney Int.* 2004, 65, 1416–1421. [[Google Scholar](#)] [[CrossRef](#)]
21. Śledziński, T.; Proczko-Markuszczyńska, M.; Kaska, L.; Stefaniak, T.; Świerczyński, J. Serum Cystatin C in Relation to Fat Mass Loss After Bariatric Surgery". *Pol. Przegl. Chir.* 2012, 84, 202–207. [[Google Scholar](#)] [[CrossRef](#)]
22. Fricker, M.; Wiesli, P.; Brändle, M.; Schwegler, B.; Schmid, C. Impact of thyroid dysfunction on serum cystatin C. *Kidney Int.* 2003, 63, 1944–1947. [[Google Scholar](#)] [[CrossRef](#)]
23. Madero, M.; Sarnak, M.J. Association of cystatin C with adverse outcomes. *Curr. Opin. Nephrol. Hypertens.* 2009, 18, 258–263. [[Google Scholar](#)] [[CrossRef](#)] [[PubMed](#)]
24. Cockcroft, D.W.; Gault, H. Prediction of Creatinine Clearance from Serum Creatinine. *Nephron* 1976, 16, 31–41. [[Google Scholar](#)] [[CrossRef](#)] [[PubMed](#)]
25. Levey, A.S.; Bosch, J.P.; Lewis, J.B.; Greene, T.; Rogers, N.; Roth, D. A More Accurate Method to Estimate Glomerular Filtration Rate from Serum Creatinine: A New Prediction Equation. Modification of Diet in Renal Disease Study Group. *Ann. Intern. Med.* 1999, 130, 461–470. [[Google Scholar](#)] [[CrossRef](#)]