ISE BUFFER

Diagnostic reagent for determination of the concentration of Chloride, Potassium and Sodium.
Liquid. Store at +5/+25°C. For in Vitro Diagnostic Use (IVD). **Do not freeze.**
It is produced for Siemens Advia Series.

Ref No	Package
AD1000	2L

Changes made in the instructions for use are marked as grey.

INTENDED USE

The test is applied for the quantitative determination of chloride, potassium and sodium in serum, plasma and urine.

GENERAL INFORMATION

Chloride is the most important extracellular anion. Therefore, just as Na^+, it has an important role in maintaining water distribution, osmotic pressure and anion-cation balance in the extracellular fluid. In contrast to high extracellular fluid concentrations (103 mmol/L), the Cl^- concentration in the intracellular fluid of erythrocytes is 45 to 54 mmol/L. In the intracellular fluid of most other tissue cells, it is only about 1 mmol/L.

Cl- is the most abundant anion in gastric and intestinal secretions. Cl^- ions are almost entirely absorbed from the gastrointestinal system. It is filtered from plasma in the glomeruli and passively reabsorbed along with Na+ from the proximal tubules. In the thick ascending limb of the loop of Henle, Cl^- is actively reabsorbed by the Na^+-K^+-$2Cl^-$ (NKCC) pump, which helps the passive reabsorption of Na+. Loop diuretics such as furosemide prevent reabsorption of Cl- via the NKCC pump. Cl- concentrations are not homeostatically controlled and passively reflect the concentration of the main ions, Na^+ and HCO_3^-. Furthermore, its concentration decreases when pathologic concentrations of other anions (ketoacids, lactate, etc.) are present.[1]

Na^+ and K^+ deficiencies are often accompanied by Cl^- deficiency, but this is not specific. Two conditions are characterized by primary chloride loss: Unbuffered loss of gastric acid (for example, in patients with vomiting and pyloric stenosis or prolonged drainage of gastric secretion) and Cl^- losing diarrhea (a rare inherited condition). Both of these are associated with metabolic (non-respiratory) alkalosis.[2]

Cl- excess is usually associated with sodium excess and has no specific characteristics. It is also notable that intravenous (IV) administration of 0.9% (g/v) aqueous NaCl (often erroneously referred to as "physiologic" or "normal" but is neither) can cause hyperchloremic acidosis. This fluid is commonly used to support extracellular fluid volume but contains equal molar amounts (154 mmol/L) of Na+ and Cl-, with plasma concentrations of 140 and 100 mmol/L, respectively.[2]

Overuse of this fluid may cause acidosis by increasing plasma Cl- concentration despite bicarbonate.[3] This "dilutional acidosis" is the opposite of the "contraction alkalosis" that is sometimes seen in edematous patients treated with diuretics.[4]

Plasma Cl^- concentration reflects the relative amounts of water and chloride in the extracellular fluid but may not accurately reflect total body Cl^- status. In hypochloremia, a low urinary Cl^- concentration (<10 mmol/L) reliably indicates chloride deficiency, unless it is the result of excessive renal Cl^- excretion.[2]

Potassium is the most important intracellular cation. It is commonly found in foods of plant and animal origin. The tendency of potassium ions to move from the intracellular compartment to the extracellular compartment against the concentration gradient is counterbalanced by the action of Na+,K+-ATPase.[1] There is considerable evidence that a high dietary intake of potassium protects against hypertension and consequently the recommended intake is much higher than the minimum requirement of 4.7 g (120 mmol/L).[5]

Potassium homeostasis is complex and occurs mainly through the control of renal potassium excretion, which has reciprocal links with both sodium and hydrogen ion excretion. Aldosterone stimulates reabsorption of sodium in place of potassium in the distal nephron, and its secretion is directly stimulated through potassium excess and the effects of renin and angiotensin II. The kidneys retain less potassium than sodium and renal loss in good health is around 30 mmol/24 hours, with small losses through sweat and feces. Since most of the potassium in the body is intracellular, the plasma concentration may not reflect the total body potassium status.[1]

Potassium deficiency is very rare in healthy individuals on a normal diet. It may be a consequence of malnutrition, but it is often iatrogenic, secondary to treatment with diuretics or as a result of increased loss from the intestine in patients with diarrheal disease.[1] Potassium deficiency is not synonymous with hypokalemia, but most patients with hypokalemia are potassium deficient.

Potassium excess in the healthy state is rare and usually iatrogenic. However, high dietary intakes can lead to

hyperkalemia in the presence of renal failure. Potassium retention is most often a consequence of acute kidney injury or chronic kidney disease and is usually, but not always, associated with hyperkalemia.[1,6] Hyperkalemia can occur in the absence of excess potassium as a result of in vivo or in vitro loss of intracellular potassium into the intracellular compartment.[1]

In healthy individuals, urinary potassium excretion is largely dependent on dietary intake and hydration status. In other individuals, hemodynamic status, acid-base balance, disease processes and drug therapies may also affect potassium excretion. It has been reported that potassium excretion in men is approximately 25% higher than in women.[7,8]

Sodium is the major extracellular cation; its concentration in extracellular fluid is approximately 140 mmol/L, compared to 14 mmol/L in intracellular fluid. Cell membranes are normally relatively impermeable to sodium, and the concentration difference is maintained by the energy-demanding Na+,K+-ATPase pump.[1] Intestinal absorption of some nutrients is correlated with the absorption of sodium, which is the basis for the use of oral rehydration solutions containing glucose and sodium.[9] Sodium is generally present in higher amounts in animal products than in plant products. Sodium chloride is used as a preservative and flavor enhancer in many manufactured foods.[1] The kidneys can produce virtually sodium-free urine, and in healthy adults with a moderate requirement, the recommended daily intake to maintain sodium balance is less than 50 mmol. A maximum sodium intake of 100 mmol/day (2.3 g/day sodium or 5.8 g salt) is recommended.[10]

Sodium and water homeostasis are closely related because sodium is the main cation that contributes to the maintenance of plasma osmolality through its effect on renal water excretion. Consequently, body sodium content is the main factor determining the volume of extracellular fluid. There are obligatory losses in sweat (which can increase significantly with excessive sweating and may be higher in patients with cystic fibrosis with high sodium content in sweat) and in feces (increased in diarrheal diseases), but sodium balance is maintained primarily through control of renal excretion.[1] The most important factor that increases sodium reabsorption from the distal nephron in response to activation of the renin-angiotensin axis by stimuli reflecting a decrease in body sodium is aldosterone.[11]

Plasma sodium concentration reflects the relative amounts of sodium and water in the ECF. The plasma sodium concentration is often normal, especially in cases of mild sodium deficiency or excess. Since sodium is a particularly common nutrient, its deficiency due to reduced intake is rarely seen. More often, deficiency occurs as a result of increased loss from the kidneys, gastrointestinal tract or skin (e.g. in burn patients).

Loss of sodium without water is not seen and isotonic loss causes an early reduction in extracellular fluid (and hence plasma) volume, leading to peripheral circulatory failure and risk of acute kidney injury. Small reductions in extracellular fluid volume do not increase vasopressin secretion (although this can be greatly increased by increases >5%) and plasma sodium concentration may remain normal. In contrast to water deficiency, there are early increases in plasma urea and creatinine concentrations (urea usually precedes creatinine). Except in cases of kidney damage or the use of diuretics, the concentration of sodium in the urine becomes very low and is a reliable indicator of the presence of sodium depletion. Sodium overload is usually iatrogenic and occurs in the background of a reduced capacity of the kidneys to excrete sodium. In healthy individuals, high sodium intake causes thirst, extracellular fluid volume tends to increase and the kidneys respond by excreting the excess. The most common cause of sodium overload is increased aldosterone secretion, either primary or secondary to increased renin secretion.[1] Hypernatremia may be present, especially if the excess is acute, but most patients with sodium excess are normo- or even (paradoxically) hyponatremic. High sodium intake predisposes to hypertension; there is a very large literature on this topic and the public health implications of a reduction in dietary sodium intake.[12]

TEST PRINCIPLE

Cl, K and Na assays in the Advia Series is based on an indirect potentiometric procedure performed with an ion-selective electrode (ISE). When the sample is mixed with ISE buffer , a solution with constant pH value and ionic strength is provided. While this solution is passed through the ion-selective electrode, changes occur in the electrical potential. These electrical potential changes are measured against to the potential of the reference electrode to obtain the value of the sample.

REAGENT COMPONENTS

Formaldehyde : < %1.0
Phosphoric acid : < %1.0

REAGENT PREPARATION

Reagent is ready for use.

REAGENT STABILITY AND STORAGE

Reagents are stable at +5/+25°C till the expiration date stated on the label which is only for closed vials.

Once opened vials are stable for 30 days at +5/+25°C in optimum conditions. On board stability is strongly related to auto analyzers' cooling specification and carry-over values.

Reagent stability and storage data have been verified by using Clinical and Laboratory Standards Institute (CLSI) EP25-A protocol.[13]

SAMPLE REQUIREMENTS

Serum, plasma and urine collected by standard procedure can be used. Li heparin collection tubes should be preferred for plasma. Multiple sample freezing and thawing should be avoided.

Chloride activity stability in serum and plasma[14,15]:

7 days at +20/+25ºC
7 day at +2/+8ºC
1 year at -20ºC

Potassium activity stability in serum and plasma[14,15]:

7 days at +20/+25ºC
7 days at +2/+8ºC
1 year at -20ºC

Sodium activity stability in serum and plasma[14,15]:

14 days at +20/+25ºC
14 days at +2/+8ºC
1 year at -20ºC

Chloride stability in urine[15,16] :

7 days at +20/+25ºC
7 days at +2/+8ºC
7 days at -20ºC

Potassium stability in urine[14,15] :

45 days at +20/+25ºC
60 days at +2/+8ºC
1 year at -20ºC

Sodium stability in urine[14,15]:

45 days at +20/+25ºC
45 days at +2/+8ºC
1 year at -20ºC

Annotation:

Serum, plasma and other fluids should be separated from cells within 3 hours to prevent changes in ionic balance due to cell metabolism and pH changes.[17]

CALIBRATION AND QUALITY CONTROL

Calibration: Use Advia Chemistry ISE Standard Set for calibration. **It requires calibration every day.** Other situations requiring calibration:
• Changes in reagent lot number,
• Out-of-limit quality control results,
• Calibration is required if ISE electrodes or hydraulic components are replaced.

Control: Commercially available control material with established values determined by this method can be used. At least two level controls must be run once in every 24 hours. Each laboratory should determine its own quality control scheme and procedures. If quality control results are not within acceptable limits, calibration is required.

REFERENCE INTERVALS / MEDICAL DECISION LEVELS

Reference ranges for chloride, potassium and sodium are as follows:

Chloride Reference Ranges[18,19]

Serum/Plasma	99-109 mmol/L
Urine	110-250 mmol/day

Potassium Reference Ranges[18,19]

Serum	3.5-5.5 mmol/L
Plasma (Men)	3.5-4.5 mmol/L
Plasma (Women	3.4-4.4 mmol/L
Urine	25-125 mmol/day

Sodium Reference Ranges[18,19]

Serum/Plasma	132-146 mmol/L
Urine	40-220 mmol/day

Annotation:

- Dietary differences are possible for serum, plasma and urine in chloride reference values.
- Sodium is a critical analyte in the treatment of patients with acute water balance and acid-base problems. Therefore, sodium values <125 and >155 mmol/L should be reported to the doctor immediately.[17]

Each laboratory should investigate the transferability of the expected values to its own patient population and if necessary, determine its own reference range.

Reference interval has been verified by using CLSI EP28-A3c protocol.[20]

To convert the results from mmol/L to mmol/day in 24-hour urine excretion;

24 hour urine = [(V x c) / 1000] mmol/day

V = 24 hour urine volume (mL)
c = analyte concentration (mmol/L)

Unit Conversion:
mEq/L = mmol/L

PERFORMANCE CHARACTERISTICS

Measuring Interval
According to CLSI EP34-ED1:2018, “Measuring Interval” refers to the interval where the analyte concentration is measured with intended accuracy in terms of medical and laboratory requirements without dilution, concentrating or any kind of pre-treatment that is between the analyte’s lower limit of quantitation (LLoQ) and upper limit of quantitation (ULoQ).[21]

The analytic measuring interval determined for chloride in serum and plasma is between 15 and 200 mmol/L. The

analytic measuring interval for chloride in urine is linear between 15 and 400 mmol/L.

The analytic measuring interval for potassium determined in serum and plasma is between 1 and 10 mmol/L. In urine, the analytic measuring interval is linear between 3 and 300 mmol/L.

The analytic measuring interval for sodium determined in serum and plasma is between 100 and 200 mmol/L. In urine, the analytic measuring interval is linear between 10 and 400 mmol/L.

LoD and LoQ values have been verified by using CLSI EP17-A2:2012 protocol.[22] Linearity Studies data have been verified by using CLSI EP06-A:2003 protocol.[23]

Precision

Running system has been developed according to 20x2x2 "The Single Site" protocol. Repeatability and Within-Laboratory Precision/Within-Device values have been obtained according to the running results.

According to the protocol in use, 2 separate runs per day have been made for 20 days (no obligation for being consecutive days). This protocol has been applied to each low and high samples separately and 80 results have been obtained for each one. Statistically, the results have been obtained using 2-factor Nested-ANOVA model.[24]

Repeatability (Within Run) and Repeatability (Day to Day) SD (standard deviation) and CV% values of chloride, sodium and potassium have been given in the table 1 and 2 respectively.

Table 1. Chloride Repeatability (Within Run) results obtained from samples in two different concentrations

Mean Concentration	SD	CV%	n
100 mmol/L (serum)	0.6	0.6	80
129 mmol/L (serum)	0.9	0.7	80
153 mmol/L (urine)	1.1	0.7	80
271 mmol/L (urine)	1.8	0.7	80

Note: This working system has been named "Within-Run Precision" in the previous CLSI - EP05-A2 manual.[25]

Table 2. Chloride Repeatability (Day to Day) results obtained from samples in two different concentrations

Mean Concentration	SD	CV%	n
100 mmol/L (serum)	1.0	1.0	80
129 mmol/L (serum)	1.2	0.9	80
153 mmol/L (urine)	1.7	1.1	80
271 mmol/L (urine)	2.1	0.78	80

Note: This working system has been named "Total Precision" in the previous CLSI - EP05-A2 manual.[25]

Table 3. Potassium Repeatability (Within Run) results obtained from samples in two different concentrations

Mean Concentration	SD	CV%	n
4.06 mmol/L (serum)	0.03	0.7	80
5.98 mmol/L (serum)	0.09	1.5	80
48 mmol/L (urine)	1.21	2.5	80
133 mmol/L (urine)	1.57	1.2	80

Table 4. Potassium Repeatability (Day to Day) results obtained from samples in two different concentrations

Mean Concentration	SD	CV%	n
4.06 mmol/L (serum)	0.07	1.7	80
5.98 mmol/L (serum)	0.11	1.8	80
48 mmol/L (urine)	1.45	3.0	80
133 mmol/L (urine)	1.66	1.3	80

Table 5. Sodium Repeatability (Within Run) results obtained from samples in two different concentrations

Mean Concentration	SD	CV%	n
133 mmol/L (serum)	0.8	0.6	80
145 mmol/L (serum)	1.3	0.9	80
59 mmol/L (urine)	0.8	1.4	80
218 mmol/L (urine)	1.2	0.6	80

Table 6. Sodium Repeatability (Day to Day) results obtained from samples in two different concentrations

Mean Concentration	SD	CV%	n
133 mmol/L (serum)	2.1	1.6	80
145 mmol/L (serum)	2.7	1.9	80
59 mmol/L (urine)	1.5	2.5	80
218 mmol/L (urine)	4.6	2.1	80

Method Comparison

As a result of the statistical evaluation of the method comparison data:

Chloride

Serum
Passing-Bablok equation:[26]
y= 0.98x + 1.2 mmol/L
r= 0.98

Urine
Passing-Bablok equation:[26]
y= 0.99x + 2.1 mmol/L
r= 0.99

Potassium

Serum
Passing-Bablok equation:[26]
y= 0.99x + 0.04 mmol/L
r= 0.99

Urine
Passing-Bablok equation:[26]
y= 0.98x + 0.18 mmol/L
r= 0.99

Sodium

Serum
Passing-Bablok equation:[26]
y= 1.02x - 2.4mmol/L
r= 0.99

Urine
Passing-Bablok equation:[26]
y= 1.03x - 3.8 mmol/L
r= 0.99

Interference

Endogenous interferant and analyte concentrations that have been used in the Sodium scanning tests has been determined according to "CLSI EP37-ED1:2018" and "CLSI EP07-ED3:2018" manuals.[27,28]

The total acceptable error rate, which is going to be used to detect whether the observed differential value obtained from Sodium interference scanning test is appropriate, is determined as ±10%.[29]

In Chloride test results, no significant interaction has been observed in the determined endogenous interferant and analyte concentrations or between interferants and analyte.

Lipemia	: ≤ 500 mg/dL
Bilirubin	: ≤ 25 mg/dL
Hemoglobin	: ≤ 500 mg/dL

In Potassium test results, no significant interaction has been observed in the determined endogenous interferant and analyte concentrations or between interferants and analyte.

Lipemia	: ≤ 500 mg/dL
Bilirubin	: ≤ 25 mg/dL
Hemoglobin	: ≤ 500 mg/dL

In Sodium test results, no significant interaction has been observed in the determined endogenous interferant and analyte concentrations or between interferants and analyte.

Lipemia	: ≤ 500 mg/dL
Bilirubin	: ≤ 25 mg/dL
Hemoglobin	: ≤ 500 mg/dL

It should be noted that endogenous interferants, as well as various medicines and metabolites, anticoagulants (e.g. Heparin, EDTA, citrate, oxalate) and preservatives (e.g. sodium floride, iodoacetate, hydrochloride acide) such as additives, materials that may contact with samples during collection and processing (serum separator devices, sample collection containers and contents, catheters, catheter wash solutions, skin disinfectants, hand cleaners and lotions, glass washing detergents, powder gloves), dietary substances known to affect some specific tests (caffeine, beta-carotene, poppy seeds, etc.), or some substances present in a sample that cause foreign proteins (heterophilic antibodies, etc.), autoimmune response (autoantibodies, etc.), or due to malignancy (for example, interference by paraproteins with phosphate testing and indirect ion selective electrode methods) may show some negative effects that will cause various attempts and some misjudgements.[28]

These performance characteristics have been obtained using an autoanalyzer. Results may vary slightly when using different equipment or manual procedures.

WARNINGS AND PRECAUTIONS

IVD: For in Vitro Diagnostic use only.
Do not use expired reagents.
Reagents with two different lot numbers should not be interchanged.
For professional use.
Follow Good Laboratory Practice (GLP) guidelines.
Contains sodium azide.

CAUTION: Human source samples are processed with this product. All human source samples must be treated as potentially infectious materials and must be handled in accordance with OSHA (Occupational Safety and Health Administration) standards.

Danger

EUH032	:Releases a very toxic gas if contacts with acid.
H317	:May cause allergic skin reaction.

Precaution

P280	:Use protective gloves / clothes / glasses / mask.
P264	:Wash your hands properly after using.
P272	:Contaminated work clothes should not be allowed to be used outside of the workplace.

Intervention

P302+P352	:Wash with plenty of water and soap if it contacts with skin.
P333+P313	:Seek medical help if it irritates your skin or develops rash.
P362+P364	:Remove contaminated clothes and wash properly before using.

Disposal

P501	:Dispose the vials and contents according to the local regulations.

REFERENCES

1. Rifai, N., Chiu, R. W., & Young, I., et al., (2023) Tietz Textbook of Laboratory Medicine (7th ed.), Chapter 37: Electrolytes and Blood Gases, p.415-415.e25, Elsevier, St. Louis, Missouri 63043
2. Rifai, N., Chiu, R. W., & Young, I., et al., (2023) Tietz Textbook of Laboratory Medicine (7th ed.), Chapter 46: Nutrition: Laboratory and Clinical Aspects, p.458-501.e7, Elsevier, St. Louis, Missouri 63043
3. Yunos NM, Bellomo R, Storey D, et al. Bench-to-bedside review: chloride in critical illness. Crit Care 2010;14:226–36.
4. Garella S, Chang BS, Kahn SI. Dilution acidosis and contraction alkalosis: review of a concept. Kidney Int 1975;8:279–83.

5. Aaron KJ, Sanders PW. Role of dietary salt and potassium intake in cardiovascular health and disease – a review of the evidence. Mayo Clin Proc 2013;88:987–95.
6. Theisen-Toupal J. Hypokalemia and hyperkalemia. Hosp Med Clin 2015;434–50.
7. Massey LK, Wise KJ. Impact of gender and age on urinary water and mineral excretion responses to acute caffeine doses. Nutr Res 1992;12:605-612.
8. Bingham SA, Williams R, Cole TJ, Price CP, Cummings JH. Reference values for analytes of 24-h urine collections known to be complete. Ann Clin Biochem 1988;25:610-619
9. Schoeller DA, van Santen E, Peterson DW, et al. Total body water measurement in humans with 18O and 2H labeled water. Am J Clin Nutr 1980;33:2686–93.
10. National Acadamies of Sciences, Engineering and Medicine. Dietary reference intervals. The National Acadamies Press. Available from http://ods.od.nih.gov/health.information/Dietary_Reference_Intervals.aspx. Accessed May 2020
11. Hirschhorn N. The treatment of acute diarrhea in children. An historical and physiological perspective. Am J Clin Nutr 1980;33:637–63.
12. He JF, Tan M, Ma T, et al. Salt reduction to prevent hypertension and cardiovascular disease. J Am Coll Cardiol 2020;75: 632–47.
13. Clinical and Laboratory Standards Institute (CLSI). Evaluation of Stability of In Vitro Diagnostic Reagents; Approved Guideline. CLSI Document EP25-A. Wayne, PA: CLSI; 2009.
14. Guder WG, Narayanan S, Wisser H, et al. List of analytes preanalytical variables. Annex In: Samples: From the Patient to the Laboratory. Darmstadt, Germany: GIT Verlag; 1996:Annex 10–1, 20–1, 40–1.
15. US Pharmacopeial Convention, Inc. General notices. In: US Pharmacopeia National Formulary, 1995 ed (USP 23/NF 18). Rockville, MD: The US Pharmacopeial Convention, Inc; 1994:11.
16. Pesce AJ, Kaplan LA, editors. Methods in Clinical Chemistry. St Louis, MO: CV Mosby, 1987:75.
17. Pesce, A. J., & Kaplan, L. D. (2009). Methods in Clinical Chemistry: Kaplan and Pesce's: Clinical Chemistry: Theory, Analysis, Correlation: Vol. I (5th ed.), Chapter: Sodium and Potassium, p.1070-77. Elseviers.
18. Data on file at Archem.
19. Wu AHB, ed. Tietz Clinical Guide to Laboratory Tests, 4th ed. St. Louis, MO: WB Saunders Company; 2006:236.
20. Clinical and Laboratory Standards Institute (CLSI). Defining, Establishing and Verifying Reference Intervals in the Clinical Laboratory; Approved Guideline – Third Edition. CLSI Document EP28-A3c. Wayne, PA: CLSI; 2010.
21. Clinical and Laboratory Standards Institute (CLSI). Establishing and Verifying an Extended Measuring Interval Through Specimen Dilution and Spiking – 1st Edition. CLSI Document EP34. Wayne, PA: CLSI; 2018.
22. Clinical and Laboratory Standards Institute (CLSI). Evaluation of Detection Capability for Clinical Laboratory Measurement Procedures; Approved Guideline – Second Edition. CLSI Document EP17-A2. Wayne, PA: CLSI; 2012.
23. Clinical and Laboratory Standards Institute (CLSI). Evaluation of the Linearity of Quantitative Measurement Procedures: A Statistical Approach - 1st Edition. CLSI Document EP06-A. Wayne, PA: CLSI; 2003.
24. Clinical and Laboratory Standards Institute (CLSI). Evaluation of Precision of Quantitative Measurement Procedures; Approved Guideline – Third Edition. CLSI Document EP05-A3. Wayne, PA: CLSI; 2014.
25. Clinical and Laboratory Standards Institute (CLSI). Evaluation of Precision Performance of Quantitative Measurement Methods; Approved Guideline - Second Edition. CLSI Document EP05-A2. Wayne, PA: CLSI; 2004.
26. Bablok W et al. A General Regression Procedure for Method Transformation. J Clin Chem Clin Biochem 1988;26:783-790.
27. Clinical and Laboratory Standards Institute (CLSI). Supplemental Tables for Interference Testing in Clinical Chemistry - First Edition. CLSI Document EP37. Wayne, PA: CLSI; 2018.
28. Clinical and Laboratory Standards Institute (CLSI). Interference Testing in Clinical Chemistry - Third Edition. CLSI Document EP07. Wayne, PA: CLSI; 2018.
29. CLIA proficiency testing criteria for acceptable analytical performance, as printed in the Federal Register July 11, 2022;87(131:41194-242.

Archem Sağlık Sanayi ve Tic. A.Ş.
Mahmutbey Mah. Halkalı Cad. No:124 Kat:4
Bağcılar/İstanbul/Türkiye
Tel: + 90 212 444 08 92
Fax: +90 212 629 98 89
info@archem.com.tr **www.archem.com.tr**

CE

SYMBOLS

IVD	In Vitro Diagnostic Medical Device
LOT	Lot Number
R1	Reagent 1
R2	Reagent 2
GTIN	Global Trade Item Number
REF	Reference Number
GLP	Good Laboratory Practices
FOR USE WITH	Identifies Products to Be Used Together
PRODUCT OF TURKEY	Product of Turkey
	Manufacturer
	Expiration Date
	Temperature Limits
	Consult Instructions for Use
	Caution
	Number of Tests