

Overview

Useful For

Diagnosis and management of a variety of disorders including bone, parathyroid, and kidney disease

Method Name

Only orderable as part of profile. For more information see:

-RTRP2 / Tubular Reabsorption of Phosphorus, Random Urine and Serum

-TRPP / Tubular Reabsorption of Phosphorus, Serum/Plasma/Urine

Photometric, Ammonium Molybdate

NY State Available

Yes

Specimen

Specimen Type

Serum

Specimen Required

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Patient Preparation: Fasting 12 hours, required

Collection Container/Tube:

Preferred: Serum gel

Acceptable: Red top

Submission Container/Tube: Plastic vial

Specimen Volume: 0.5 mL serum

Collection Instructions:

1. Serum gel tubes should be centrifuged within 2 hours of collection.
2. Red-top tubes should be centrifuged and the serum aliquoted into a plastic vial within 2 hours of collection.

Specimen Minimum Volume

Serum: 0.25 mL

Reject Due To

Gross	Reject
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hemolysis	
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Specimen Stability Information

Specimen Type	Temperature	Time	Special Container
Serum	Refrigerated (preferred)	7 days	
	Frozen	365 days	

Clinical & Interpretive

Clinical Information

Of the phosphorus contained in the body, 88% is localized in bone in the form of hydroxyapatite. The remainder is utilized during intermediary carbohydrate metabolism and bound to physiologically important substances such as phospholipids, nucleic acids, and adenosine triphosphate (ATP). Phosphorus exists in blood in the form of inorganic phosphate and organically bound phosphoric acid. The small amount of extracellular organic phosphorus is found exclusively in the form of phospholipids. Serum contains approximately 2.5 to 4.5 mg/dL of inorganic phosphate (the fraction measure in routine biochemical assays). Serum phosphate concentrations are dependent on dietary intake and regulation by hormones such as parathyroid hormone and 1,25 vitamin D, and systemic acid base status and may vary widely.

Hypophosphatemia may have 4 general causes: shift of phosphate from extracellular to intracellular, renal phosphate wasting, loss from the gastrointestinal tract, and loss from intracellular stores.

Hyperphosphatemia is usually secondary to an inability of the kidneys to excrete phosphate and is common in patients with chronic kidney disease stage 4 or greater. Acute hyperphosphatemia can occur as a result of tissue breakdown such as rhabdomyolysis. Other possible contributory factors are increased intake, especially in combination with chronic kidney disease, or a shift of phosphate from tissues into the extracellular fluid.

Reference Values

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Males

<1 year: Not established

1-4 years: 4.3-5.4 mg/dL

5-13 years: 3.7-5.4 mg/dL

14-15 years: 3.5-5.3 mg/dL

16-17 years: 3.1-4.7 mg/dL

> or =18 years: 2.5-4.5 mg/dL

Females

<1 year: Not established

1-7 years: 4.3-5.4 mg/dL

8-13 years: 4.0-5.2 mg/dL
14-15 years: 3.5-4.9 mg/dL
16-17 years: 3.1-4.7 mg/dL
> or =18 years: 2.5-4.5 mg/dL

Interpretation

Hypophosphatemia is relatively common in hospitalized patients. Serum concentrations of phosphate between 1.5 and 2.4 mg/dL may be considered moderately decreased and are not usually associated with clinical signs and symptoms. Levels below 1.5 mg/dL may result in muscle weakness, hemolysis of red cells, coma, bone deformity, and impaired growth.

The most acute problem associated with rapid elevations of serum phosphate levels is hypocalcemia with tetany, seizures, and hypotension. Soft tissue calcification is also an important long-term effect of high phosphorus levels.

Phosphorus levels below 1.0 mg/dL are potentially life-threatening and are considered a critical value in the Mayo Health System.

Cautions

Phosphorus has a very strong biphasic circadian rhythm. Values are lowest in the morning, peak first in the late afternoon and peak again in the late evening. The second peak is quite elevated and results may be outside the reference range.

Clinical Reference

1. Delaney MP, Lamb EJ. Kidney disease. In: Rifai N, Horvath AR, Wittwer CT, eds: Tietz Textbook of Clinical Chemistry and Molecular Diagnostics. 6th ed. Elsevier; 2018:1280-1283
2. Agarwal R, Knochel JP. Hypophosphatemia and hyperphosphatemia. In: Brenner BM, ed. The Kidney. 6th ed. WB Saunders Company; 2000:1071-1125
3. Yu GC, Lee DBN. Clinical disorders of phosphorus metabolism. West J Med. 1987;147(5):569-576
4. Koumakis E, Cormier C, Roux C, Briot K. The causes of hypo- and hyperphosphatemia in humans. Calcif Tissue Int. 2021;108(1):41-73. doi:10.1007/s00223-020-00664-9

Performance**Method Description**

Inorganic phosphate forms an ammonium phosphomolybdate complex with ammonium molybdate in the presence of sulfuric acid. The concentration of phosphomolybdate formed is directly proportional to the inorganic phosphate concentration and is measured photometrically. (Package insert: Phosphate (Inorganic) ver 2. Roche Diagnostics; V11.0 07/2019)

PDF Report

No

Day(s) Performed

Monday through Sunday

Report Available

Same day/1 to 3 days

Specimen Retention Time

1 week

Performing Laboratory Location

Mayo Clinic Laboratories - Rochester Main Campus

Fees & Codes**Fees**

- Authorized users can sign in to [Test Prices](#) for detailed fee information.
- Clients without access to Test Prices can contact [Customer Service](#) 24 hours a day, seven days a week.
- Prospective clients should contact their account representative. For assistance, contact [Customer Service](#).

Test Classification

This test has been cleared, approved, or is exempt by the US Food and Drug Administration and is used per manufacturer's instructions. Performance characteristics were verified by Mayo Clinic in a manner consistent with CLIA requirements.

LOINC® Information

Test ID	Test Order Name	Order LOINC® Value
PHOSS	Phosphorus (Inorganic), S	2777-1

Result ID	Test Result Name	Result LOINC® Value
PHOSS	Phosphorus (Inorganic), S	2777-1