



# COMPLETE CHEMISTRY PANEL

## INTERPRETATION GUIDE

Scroll down or click on the following parameters to quickly access content

**CHEM15/17:**

[GLU](#)

[BUN](#)

[CREA](#)

[BUN/CREA](#)

[P](#)

[Ca](#)

[TP total protein](#)

[ALB](#)

[GLOB](#)

[ALB/GLOB](#)

[ALT](#)

[ALP \(ALKP\)](#)

[GGT](#)

[TBIL](#)

[CHOL](#)

[AMYLASE](#)

[LIPASE](#)

-

Follow-up test

[UPC](#)



**CHARLIE**

IDEXX Representative

Species: Feline

Gender: Male

Year of Birth: 2001

Client: Carlos Animal Hospital

Veterinarian: Peter Parker, DVM ABVP

Instrument: ProCyte Dx Hematology Analyzer

Catalyst Dx Chemistry Analyzer

IDEXX VetLab UA Analyzer

**Chemistry**

8/7/2016 @ 2:11 PM



8/7/2016  
2:02 PM



8/17/2016  
11:18 PM

Parameter	8/7/2016 @ 2:11 PM	Reference Range	8/7/2016 2:02 PM	8/17/2016 11:18 PM
Glucose	6.99	3.94 - 8.83 mmol/L		6.94
<b>BUN</b>	<b>23.21</b>	<b>5.71 - 12.85 mmol/L</b>	H	<b>23.58</b>
Creatinine	194.48	70.72 - 212.16 µmol/L		<b>229.84</b>
BUN:Creatinine Ratio	30			25
Phosphorus	1.61	1 - 2.42 mmol/L		1.71
Calcium	2.45	1.95 - 2.82 mmol/L		2.42
Sodium			<b>167</b>	161
Potassium			<b>3.1</b>	<b>3.3</b>
Chloride			119	120
Total Protein	89	57 - 89 g/L		85
Albumin	34	23 - 39 g/L		35
<b>Globulin</b>	<b>55</b>	<b>28 - 51 g/L</b>	H	50
Alb:Glob Ratio	0.6			0.7
ALT	105	12 - 130 U/L		<b>131</b>
ALP	43	14 - 111 U/L		59
GGT	< 0	0 - 1 U/L		< 0
Bilirubin - Total	6.84	0 - 15.39 µmol/L		6.84
Cholesterol	4.94	1.68 - 5.82 mmol/L		4.24
SDMA	31	0 - 14 µg/dL		4.24

*Sample report*

# Glucose

---

## Description

- Glucose is the basic nutrient for tissues and is obtained by diet, glycogenolysis and gluconeogenesis.
- Glucose is tightly regulated in the normal animal.
- Cellular uptake is stimulated by insulin.
- Neurons, RBCs and renal tubular epithelial cells do not require insulin for glucose uptake.

## Values Below Reference Range

Hypoglycemia

### Common Causes

- Beta cell tumor (insulinoma)
- Severe exertion (hunting dog hypoglycemia)
- Hepatic disease
- Sepsis
- Idiopathic or puppy hypoglycemia (small breed dogs)
- Hypoadrenocorticism
- Paraneoplastic syndrome
- Starvation (mild decrease only)
- Drugs that may decrease glucose measurement
  - Insulin
  - Beta blockers (propranolol)
  - Antihistamines
  - Spironolactone
  - Anabolic steroids (only in diabetic animals)
- Artifacts that may modify glucose values include
  - Lipemia (increased value)
  - Failure to spin and separate sample within 30 minutes of collection (decreased value)

### Related Findings

- Elevated liver enzymes (ALT, AST, ALP, GGT)
- Inflammatory leukogram with sepsis (elevated or decreased WBC with left shift)
- Hypoadrenocorticism-lymphopenia, low sodium and high potassium

- Weakness

#### **Other Laboratory Tests**

- CBC
- ALT, AST, ALP, GGT
- Electrolyte panel
- Amended insulin:glucose ratio

#### **Values Above Reference Range**

Hyperglycemia

#### **Common Causes**

- Diabetes mellitus
- Stress (cat)
- Post-prandial (mild increase only)
- Hyperadrenocorticism
- Renal failure
- Acute pancreatitis
- Diestrus
- Drugs which may elevate glucose values include
  - Glucocorticoids
  - Diuretics
  - Megestrol acetate
  - Progesterone
  - Epinephrine
  - Salicylates
  - Asparaginase

#### **Related Findings**

- Polyuria/Polydipsia
- Weight loss
- Vomiting
- Weakness

#### **Other Laboratory Tests**

- Fructosamine
- Urinalysis

#### **Reference**

Duncan JR, Prasse KW, Mahaffey EA. *Veterinary Laboratory Medicine*, 3rd ed. Iowa State University Press: Ames, Ia; 1994.

# Blood Urea Nitrogen (BUN)

---

## Description

- **Urea is formed exclusively in the liver from ammonia, and excreted mainly by the kidney. BUN readily diffuses into blood and all body water in similar concentration.**
- Some urea is passively reabsorbed from the tubules back into the blood, but most is excreted.
- Urea also contributes to the osmotic gradient in medulla, important in medullary washout syndrome.
- There is an inverse relationship between BUN and glomerular filtration, and between BUN and tubular urine flow rate.
- Not specific for primary renal disease
- Affected by larger number of extrarenal factors than is creatinine, such as other diseases, some chemicals and drugs, diet, and condition of serum (e.g., lipemia)

## Values Below Reference Range

### Common Causes

- Chronic hepatic insufficiency
  - Congenital portosystemic shunts, cirrhosis
- Severe PU-PD
  - Hyperadrenocorticism, diabetes insipidus, psychogenic
- Increased excretion
  - Overhydration, late pregnancy
- Drugs
  - Diuretics, corticosteroids, salt in diet, aminoglycosides, amphotericin B, growth hormone
- Artifacts (falsely decreased)
  - High concentrations of sodium fluoride and sodium citrate; chloramphenicol; lipemia (severe)
- Low-protein diet

### Related Findings

- Urine specific gravity
  - Low in PU/PD, increased excretion, some drugs
- Serum bile acids
  - High in congenital and acquired hepatic atrophy (shunts and cirrhosis)
- Dexamethasone suppression and ACTH stimulation tests
  - Lack of suppression and/or high, respectively, with hyperadrenocorticism

### Other Laboratory Tests

- CBC
  - May see hemoconcentration
- Infectious disease tests
  - FeCoV, FeLV, FIV, *Ehrlichia*, Rocky Mountain spotted fever, *Coccidioides*
- Pre- and postprandial bile acids
  - To rule out chronic hepatic insufficiency due to congenital or acquired atrophy and shunts
- Hepatic biopsy
  - For definitive diagnosis of chronic hepatic insufficiency
- Adrenal cortical tests
  - For cortical hyperplasia or neoplasia
- Urinalysis
  - Specific gravity may help differentiate between decreased production and increased excretion
- Water deprivation test
  - May be useful in some cases to determine if tubules can concentrate the filtrate

### Values Above Reference Range

#### Common Causes

- Renal
  - Renal parenchymal disease due to glomerular disease, tubular dysfunction, necrosis, fibrosis
- Prerenal
  - Shock, dehydration, poor cardiac output, recent high-protein meal, gastrointestinal hemorrhage, catabolism of body tissues (fever, trauma)
- Postrenal
  - Urinary outflow tract disease due to bladder or ureteral rupture, or ureteral or urethral obstruction

#### Related Findings

- Creatinine
  - May be normal or low if nonrenal cause of increased BUN; increased with renal or postrenal causes of increased BUN
- Urine
  - Isosthenuric or inadequate concentration suggest primary renal disease
- Serum phosphorus
  - Elevated in primary renal disease
- Albumin
  - Albuminuria and hypoalbuminemia in glomerulopathy
- Parathyroid hormone increase

- Renal pseudohyperparathyroidism
- CBC
  - Hemogram may show mild to moderate nonregenerative anemia (decreased erythropoetin).
- Calcium
  - Levels increase acutely in some cases, and decrease in some cases of ethylene glycol toxicity

#### **Other Laboratory Tests**

- Creatinine
  - May help differentiate between primary renal and prerenal disease
- Urinalysis
  - Check specific gravity and protein, casts, bacteria, crystal
- Electrolytes
  - Help rule out hypoadrenocorticism and metabolic acidosis

#### **References**

Willard MD, Tvedten H, Turnwald GH. *Small Animal Clinical Diagnosis by Laboratory Methods*. WB Saunders: Philadelphia, Pa; 1994.

# Creatinine

---

## Description

- Creatinine originates from nonenzymatic conversion of creatine in muscles, occurring at a generally constant and uniform daily rate. Values are not significantly affected by mild catabolism or diet.
- Not altered by as many nonrenal factors as BUN, therefore, it is more specific in evaluating renal disease than is BUN.
- It is freely filtered by glomeruli. Clearance from plasma to urine is used to approximate glomerular filtration rate (GFR), which yields information on renal function.
- It is not reabsorbed by tubules; therefore, it is less affected by factors affecting urine flow rate than BUN.

## Values Below Reference Range

### Common Causes

- Pregnancy
  - Causes increased cardiac output, thereby increasing GFR
- Marked muscle wasting
  - Especially if BUN and serum phosphorus are high

### Other Laboratory Tests

- Bilirubin
  - High bilirubin values may cause artifactual decrease, depending on methodology used.

## Values Above Reference Range

### Common Causes

- Decreased GFR
  - Prerenal, renal or postrenal causes
- Acute myositis and severe muscle trauma
  - Uncertain significance
- False increase
  - Bilirubin >10 mg/dL, hemoglobin, lipemia, ascorbic acid, BSP, PSP, cephalosporins, barbiturates, acetoacetate, fructose, glucose
- Feeding cooked meat
  - Mild increase <1 mg/dL
- Nephrotoxic drugs
  - May increase serum creatinine

### Related Findings

- BUN
  - Concurrently increases with decreased glomerular filtration of renal, prerenal or postrenal causes
- Serum phosphorus



- May be increased in severe prerenal azotemia
- Urine specific gravity
  - Used to differentiate between prerenal, renal or postrenal causes; usually normal in prerenal azotemia and isosthenuric in renal azotemia

#### **Other Laboratory Tests**

- Serum phosphorus
- BUN
  - To help assess the status of the GFR and rule out acute muscle disease
- Urine specific gravity
  - To help distinguish between renal and prerenal azotemia
- Serum electrolytes
  - To help evaluate for hypoadrenocorticism, chronic blood loss, renal disease
- Serum calcium
  - To evaluate for hypercalcemic nephropathy
- Albumin
  - To evaluate if there is albumin loss
- Glucose
  - To rule out hyperosmolar diabetes mellitus
- Radiographs, ultrasound
  - To evaluate kidneys for focal or diffuse abnormalities
- Renal biopsy
  - When indicated by above tests, to arrive at a definitive diagnosis, plan treatment, establish prognosis

#### **Reference**

Willard MD, Tvedten H, Turnwald GH. *Small Animal Clinical Diagnosis By Laboratory Methods*. WB Saunders: Philadelphia, Pa; 1994.

# Blood Urea Nitrogen: Creatinine Ratio (BUN:CREA)

---

## Description

- Ratio of blood urea nitrogen value to creatinine value
- Originally thought to be of value in differential diagnosis of azotemia
- Possible differences in tubular resorption and diffusion rate and effects of diet and protein metabolism between the two compounds
- There are too many variables for the ratio to be used as a diagnostic parameter

## Values Below Reference Range

### Common Causes

- Acute myositis and severe muscle trauma
- Possibly high dietary protein ingestion
- Artificially elevated creatinine due to bilirubin, hemoglobin, lipemia, ascorbic acid, penicillins, cephalosporins, barbiturates

### Related Findings

Normal blood urea nitrogen

### Other Laboratory Tests

- Urine myoglobin
- Urinalysis: to classify azotemia if present

## Values Above Reference Range

### Common Causes

- Significant muscle loss and pregnancy (due to decrease creatinine)
- Gastrointestinal hemorrhage

### Other Laboratory Tests

- Urinalysis: helps differentiate between renal and nonrenal azotemia
- CBC: may show mild to moderate nonregenerative anemia in renal azotemia

## References

Duncan JR et al. *Duncan and Prasse's Veterinary Laboratory Medicine: Clinical Pathology*, 4th ed. Ames, Ia: Iowa State University Press; 2003.

Kaneko JJ et al. *Clinical Biochemistry of Domestic Animals*, 5th ed. San Diego, Ca: Academic Press; 1997.

Stockham SL et al. *Fundamentals of Veterinary Clinical Pathology*, 1st ed. Ames, Ia: Iowa State University Press; 2002.

Willard, MD et al. *Small Animal Clinical Diagnosis by Laboratory Methods*, 4th ed. St. Louis, Mo: Elsevier; 2004.

# Phosphorus

---

## Description

- Important component of ATP
- Depletion may affect brain, RBC and skeletal muscle cells
- Severe hypophosphatemia (< 1.0 mg/dL) is most often seen with diabetic ketoacidosis

## Values Below Reference Range

Hypophosphatemia

### Common Causes

- Increased urinary excretion
  - Diabetes mellitus ketoacidosis
  - Hypercalcemia of malignancy
- Phosphate-binding antacids
- Renal failure (horse)
- Translocation from extracellular fluid (ECF) to intracellular fluid (ICF)
  - Bicarbonate administration
  - Insulin therapy, hyperinsulinism
  - IV glucose administration

### Other Diagnoses

- Decreased intestinal absorption
  - Decreased dietary intake
  - Malabsorption syndrome
  - Phosphate-binding agent
  - Steatorrhea
  - Vitamin D deficiency
- Increased urinary excretion
  - Diuretics
  - Eclampsia, milk fever
  - Fanconi syndrome (renal tubular defects)
  - Hyperadrenocorticism
  - Hyperaldosteronism
  - Primary hyperparathyroidism
- Translocation from extracellular fluid (ECF) to intracellular fluid (ICF)

- Respiratory and metabolic alkalosis
- Vomiting and/or diarrhea

#### **Other Laboratory Tests**

- Cytology and/or histology of tissue specimens
- Fecal floatation
- FeLV/FIV
- Serum PTH
- Total T<sub>4</sub>, Free T<sub>4</sub>, T<sub>3</sub> suppression test
- TLI, cobalamin, folate
- Urinalysis
- Vitamin D metabolites (especially cholecalciferol)

#### **Values Above Reference Range**

Hyperphosphatemia

#### **Common Causes**

- Hypervitaminosis D
  - Cholecalciferol rodenticides
- Renal failure
  - Prerenal and postrenal azotemia
- Rhabdomyolysis
- Young, growing animals

#### **Other Diagnoses**

- Drugs
- Hemolysis, in vitro
- Hyperthyroidism
- Diet excess
- Neoplasia with osteolytic bone lesions
- Jasmine toxicity
- Laboratory error
- Nutritional secondary hyperparathyroidism
- Phosphate enema, phosphate-containing fluids
- Primary hypoparathyroidism

#### **Other Laboratory Tests**

- Serum PTH
- Urinalysis

- Total T<sub>4</sub>, Free T<sub>4</sub>, T<sub>3</sub> suppression test
- Skeletal radiographs

### References

Duncan JR, Prasse KW, Mahaffey EA. *Veterinary Laboratory Medicine*, 3rd ed. Ames, Ia: Iowa State University Press, 1994.

Tilley LP, et al. *The 5-Minute Veterinary Consult: Canine and Feline*. Williams & Wilkins: Baltimore, Md; 1997.

Willard MD, Tvedten H, Turnwald GH. *Small Animal Clinical Diagnosis by Laboratory Methods*. 3rd ed. WB Saunders: Philadelphia, Pa; 1999.

# Calcium

---

## Description

- Controlled by PTH and vitamin D, and their interactions with bone, kidneys, intestines and parathyroid glands
- Total calcium concentration: 50% ionized, 40% protein-bound and 10% complexed with bicarbonate, citrate, lactate, phosphate
- Ionized calcium is the active form and is available to tissues; protein-bound form is not.
- Only the ionized and complexed forms can be filtered by the kidneys.

## Values Below Reference Range

Hypocalcemia

### Common Causes

- Eclampsia
  - Dog, horse, ewe
- Parturient paresis
  - Cow
- Ethylene glycol toxicity
  - Dog, cat
- Hypoalbuminemia
- Iatrogenic
  - Following bilateral thyroidectomy/thyroid surgery
- Renal failure
  - Renal secondary hyperparathyroidism
- Laboratory error

### Other Diagnoses

- Alkalemia (especially in ruminants)
- Bicarbonate treatment for salicylate toxicity
- Blister beetle poisoning (horse)
- Factitious (e.g., EDTA)
- Hypercalcitonism
- Hypomagnesemic tetany (ruminant)
- Hypovitaminosis D associated with excess phosphorus
- Intestinal malabsorption (dog)

- Malabsorption syndrome
- Nutritional secondary hyperparathyroidism
- Pancreatitis (acute/necrotizing)
- Primary hypoparathyroidism
- Transport tetany (sheep)

### **Other Laboratory Tests**

- Pancreatic tests
  - Amylase, lipase, TLI
- CBC
- Serum and urine ethylene glycol concentrations
  - Peak between 1 and 6 hours after ingestion
- Serum magnesium
- Serum osmolality
- Serum PTH
- Urinalysis

### **Values Above Reference Range**

Hypercalcemia

### **Other Diagnoses**

- Ingestion of calciferol (cholecalciferol)-containing rodenticides
- Factitious
  - Lipemia, postprandial
- Granulomatous diseases such as blastomycosis
  - Rare
- Hemoconcentration; hyperalbuminemia
- Hypervitaminosis D
- Hypoadrenocorticism
- Iatrogenic (rare)
  - Excessive supplementation
  - Excessive oral phosphate binders
- Osteolytic bone lesions (rare)
  - Hypertrophic osteodystrophy
  - Multiple myeloma

- Osteomyelitis
- Plant toxicity
  - Jasmine, *Cestrum sp.*, *Solanum sp.*
- Primary hyperparathyroidism
- Renal failure
  - Horse, cow, uncommon in dog
- Young animal, large breed

#### **Other Laboratory Tests**

- Cytology and/or histology of tissue specimens
- Ionized calcium
- Serum PTH
- Serum PTH-related protein (PTHrp)
- Urinalysis

#### **Reference**

Duncan JR, et al. *Veterinary Laboratory Medicine; Clinical Pathology*. 3rd ed. Iowa State University Press: Ames, Ia; 1994.

Tilley LP, et al. *The 5 Minute Veterinary Consult; Canine and Feline*. Williams & Wilkins: Baltimore, Md; 1997.

Willard MD, et al. *Small Animal Clinical Diagnosis by Laboratory Methods*. 3rd ed. WB Saunders: Philadelphia, Pa; 1999.



# Total Protein

---

## Description

- Sum of albumin and globulin (serum) and fibrinogen (plasma)
- The proteins in the blood consist of albumin, fibrinogen, coagulation proteins, and globulins (to include immunoglobulins and acute-phase proteins).
- Total protein is typically measured on serum and consists of albumin, fibrinogen and globulins. The primary difference between plasma protein and serum total protein is the absence of coagulation proteins and fibrinogen in the serum.
- Most proteins are produced by the liver, and immunoglobulins by lymphoid tissue (plasma cells).

## Values Below Reference Range

Hypoproteinemia

### Common Causes

- Decreased production:
  - Intestinal malabsorption
  - Malnutrition
  - Liver disease, atrophy, fibrosis
  - Immunodeficiency disease
  - Failure of passive transfer
  - Exocrine pancreatic insufficiency
- Increased loss:
  - Renal disease
  - Protein-losing glomerulopathy/renal disease
  - Severe exudative skin disease
  - External hemorrhage
  - Protein-losing enteropathies
  - Infiltrative intestinal neoplasia

### Related Findings

- Proteinuria
- Elevated renal function tests and liver enzymes
- Decreased TLI

### Other Laboratory Tests

- Renal function tests (BUN, creatinine, urinalysis, urine protein:creatinine ratio)
- Liver function/enzyme tests (ALT, AST, albumin, bile acids)
- CBC

- Intestinal/Skin biopsies
- Albumin/Globulin ratio
- Protein electrophoresis
- Body fluid analysis

### Values Above Reference Range

Hyperproteinemia

#### Common Causes

- Dehydration
- Chronic inflammation
- Abnormal globulins (*Ehrlichia*, lymphoma, plasma cell myeloma)
- Intravascular hemolysis
- Hyperbilirubinemia
- Lipemia

#### Related Findings

- Hemoconcentration
- Hemolysis
- Polyclonal, monoclonal gammopathies

#### Other Laboratory Tests

- Serum protein electrophoresis—may have polyclonal or monoclonal gammopathy
- Albumin—may be low or high
- Fibrinogen—may be high, especially in large animals
- CBC—may see hemoconcentration
- Survey radiographs
- Bence Jones protein
- Bone or soft tissue biopsy if plasma cell myeloma suspected
- Infectious disease tests—FeCoV, FeLV, FIV, *Ehrlichia*, Rocky Mountain spotted fever, *Coccidioides*

### References

Willard MD, et al. *Small Animal Clinical Diagnosis by Laboratory Methods*. WB Saunders: Philadelphia, Pa; 1994.

# Albumin

---

## Description

- Important regulator of osmotic equilibrium
- Produced by the liver
- Carrier for several compounds (especially calcium)

## Values Below Reference Range Hypoalbuminemia

### Common Causes

- Decreased production
  - Liver disease, atrophy, fibrosis/cirrhosis
  - Primary or secondary intestinal malabsorption
  - Malnutrition (dietary or parasitic)
  - Exocrine pancreatic insufficiency
- Increased loss
  - Renal disease, proteinuria
  - Severe exudative skin disease/trauma
  - External hemorrhage
  - Protein-losing enteropathies
  - Infiltrative enteropathies (neoplastic, Johnes)

### Related Findings

- Liver enzymes and function tests
  - May be elevated (inflammation, neoplasia, necrosis)
- Renal function tests
  - May be elevated (renal disease) or low (hepatic insufficiency)
- Urine
- May contain increased protein
- Serum calcium
- May be low

### Other Laboratory Tests

- Hepatic function tests, enzymes and biopsy
- Renal function tests, biopsy
- Urinalysis, including protein/creatinine ratio
- Abdominal/Thoracic tap for fluid analysis (if applicable)
- TLI
- Coagulation tests
- Intestinal biopsy

## Values Above Reference Range Hyperalbuminemia

### Common Causes

- Dehydration
- Laboratory error

#### **Related Findings**

- Hemoconcentration (dehydration)
- Calcium
- May be falsely high
- Hyperbilirubinemia
- Overproduction of albumin does not occur

#### **Other Laboratory Tests**

- Bilirubin
- Total protein
- Hemogram

#### **Reference**

Willard MD, et al. *Small Animal Clinical Diagnosis by Laboratory Methods*. Philadelphia, Pa: W.B. Saunders and Co., 1994.

# Albumin:Globulin Ratio (A:G Ratio)

---

## Description

- This arithmetic value (ratio of serum albumin to globulin concentrations) has been used to aid in the interpretation of total protein values.
- The ratio will remain normal if both fractions are uniformly altered. It occurs with hyperproteinemia (dehydration) and hypoproteinemia (e.g., overhydration, acute blood loss, exudative skin disease, gastrointestinal disease).
- The ratio will be decreased or increased if an alteration of one fraction predominates.

**Note:** See interpretive guides for Albumin and Globulin.

## Values Below Reference Range

Decreased A/G ratio

### Common Causes

- Decreased albumin
- Increased globulin

## Values Above Reference Range

Increased A/G ratio

### Common Causes

- Increased albumin
- Decreased globulin

## References

Duncan JR et al. *Duncan and Prasse's Veterinary Laboratory Medicine: Clinical Pathology*, 4th ed. Ames, Ia: Iowa State University Press; 2003.

Kaneko JJ et al. *Clinical Biochemistry of Domestic Animals*, 5th ed. San Diego, Ca: Academic Press; 1997.

Stockham SL et al. *Fundamentals of Veterinary Clinical Pathology*, 1st ed. Ames, Ia: Iowa State University Press; 2002.

Willard, MD et al. *Small Animal Clinical Diagnosis by Laboratory Methods*, 4th ed. St. Louis, Mo: Elsevier; 2004.

# Alanine Aminotransferase (ALT)

---

## Description

- ALT is a cytosolic enzyme that catalyzes the reversible transamination of L-alanine and 2-oxoglutarate to pyruvate and glutamate.
- The enzyme is almost exclusively found within hepatocytes, but also in very small amounts in muscle.
- As a result, serum increases are highly specific for hepatocyte injury in dogs and cats.
- The hepatocyte level of ALT in horses, ruminants and pigs is very low, and hence ALT is of limited value in these species.

## Values Below Reference Range

### Common Causes

The hepatocyte level of ALT in horses, ruminants and pigs is very low, and ALT is of limited value in these species.

## Values Above Reference Range

### Common Causes

- Primary hepatocellular and biliary system diseases
  - Infectious diseases: ICH, FIP, leptospirosis, septicemia, hepatic abscess, cholangiohepatitis
- Toxic insult
  - Aflatoxicosis, pyrrolyzidine alkaloids, idiosyncratic drug reactions
- Neoplasia
  - Lymphosarcoma, bile duct carcinoma, metastatic neoplasia
- Obstructive disease
  - Cholangitis, pancreatitis, neoplasia, bile duct fibrosis
- Trauma
- Chronic active hepatitis
- Storage disorders
  - Copper storage disease
- Metabolic disorders
  - Diabetes mellitus, hyperadrenocorticism, ketosis, hepatic lipidosis, feline hyperthyroidism
- Drug therapy
  - Corticosteroids, barbiturates
- Severe skeletal myopathy

### Related Findings

- Other hepatic enzymes

- AST, ALP, GGT, GLDH may also be elevated
- Bilirubin
  - May be increased due to cholestasis, portosystemic shunts
- Cholesterol
  - Elevated with cholestatic disease
- Glucose
  - High in cases of diabetes mellitus
- CBC
  - May have evidence of inflammatory disease, anemia, leukemia, stress

### **Other Laboratory Tests**

- Hepatic biopsy
  - For definitive diagnosis of underlying primary hepatobiliary disease
- Adrenocortical function tests
  - To diagnose hyperadrenocorticism
- Amylase, lipase, TLI
  - To assess for the possibility of pancreatitis

### **References**

Willard MD, Tvedten H, Turnwald GH. *Small Animal Clinical Diagnosis by Laboratory Methods*. 2nd ed. WB Saunders: Philadelphia, Pa; 1994.

Duncan JR, Prasse KW, Mahaffey EA. *Veterinary Laboratory Medicine: Clinical Pathology*. 3rd ed. Iowa State University Press: Ames, Ia; 1994.

# Alkaline Phosphatase (ALKP)

---

## Description

- Alkaline phosphatase refers to a group of enzymes that catalyze the hydrolysis of phosphate esters at an alkaline pH in vitro.
- Little is known about their intracellular function, but these are membrane-bound and present in most tissues in the body.
- High activities are present in the liver, bone, intestine, kidney and placenta.
- Intestinal, renal and placental isoenzymes have extremely short half-lives, and are not responsible for significant increases in serum ALP.
- Changes in the serum level of ALP can be attributed to bone and hepatic isoenzymes.
- The hepatic ALP is found mainly in canalicular cell membranes, and is released as a result of biliary disease and increased hydrostatic pressure in the biliary system.
- In the dog, a steroid-induced isoenzyme is produced in the liver on exposure to glucocorticoids, contributing to serum ALP increases.
- In cats, the hepatic isoenzyme has a short half-life, and even minor increases should be considered significant.
- In ruminants and horses, the serum ALP levels are highly variable, and are of limited diagnostic use. GGT, GLDH and AST are of more use in large animals.

## Values Below Reference Range

### Common Causes

- Not clinically significant
- Artifact—assay performed on EDTA or oxalate plasma

## Values Above Reference Range

### Common Causes

- Cholestasis
  - Intra- or extra-hepatic cholestasis
- Primary hepatocyte injury
  - See ALT
- Bile duct obstruction
  - Ascending infection
  - Neoplasia
  - Pancreatitis
  - Fibrosis
  - Abscessation



- Intestinal foreign body
- Corticosteroid excess (dogs only)
  - Hyperadrenocorticism
  - Exogenous glucocorticoids
- Increased osteoblastic activity in bone
  - Young animals—increased osteoblastic activity leads to increases up to 3–4 times the basal level expected in mature animals.
- Bone disease
  - Bone neoplasia
  - Rickets
  - Fractures
  - Hyperparathyroidism
- Drug therapy
  - Anticonvulsants/Barbiturates
- Miscellaneous
  - Neoplasia

### **Related Findings**

- Hepatic enzymes (ALT, AST, GGT)
  - Increased with primary hepatocellular or cholestatic disease
- Bilirubin
  - Often increased due to cholestasis
  - Note that elevations of ALP will precede elevations in bilirubin.
- Cholesterol
  - Increased with cholestasis or endocrine disease, but may be decreased with hepatic insufficiency or portosystemic shunts
- Amylase, lipase, TLI
  - May be increased in cases of pancreatitis
- Hemogram
  - May indicate the presence of inflammation, or a stress leukogram in cases of corticosteroid excess

### **Other Laboratory Tests**

- Urinalysis
  - To check for bilirubinuria, and as part of the screen for hyperadrenocorticism
- Other hepatic enzymes

- The pattern of change often helps to further define the likely cause
- Hepatic biopsy
- Adrenocortical function tests
  - To assess for possible hyperadrenocorticism

### **References**

Willard MD, Tvedten H, Turnwald GH. *Small Animal Clinical Diagnosis by Laboratory Methods*. 2nd ed. WB Saunders: Philadelphia, Pa; 1994.

Duncan JR, Prasse KW, Mahaffey EA. *Veterinary Laboratory Medicine: Clinical Pathology*. 3rd ed. Iowa State University Press: Ames, Ia; 1994.

# Gamma-Glutamyl Transferase (GGT)

---

## Description

- GGT is involved in glutathione metabolism and is mainly associated with the microsomal membranes of hepatocyte canalicular surfaces, bile duct epithelium and renal convoluted tubular epithelial cells.
- The activity of serum GGT parallels the activity of ALP.
- For detection of hepatobiliary disease, GGT has a higher specificity and lower sensitivity than ALP in dogs. The opposite is seen in cats, except for hepatic lipidosis.
- GGT is less influenced than ALP by secondary hepatic disease and enzyme-inducing drugs; generally GGT will only be mildly increased.
- GGT does not tend to increase after increased osteoblastic activity in young, growing animals, bone disease and acute hepatic necrosis, as does ALP.
- GGT is more useful in horses and ruminants due to its narrow reference range and the high variability of ALP activity in these species.
- The use of ALP and GGT together increases the predictive value for hepatic disease.

## Values Below Reference Range

### Common Causes

Not clinically relevant

## Values Above Reference Range

Increased serum GGT

### Common Causes

- Intrahepatic (bile canaliculi) and extrahepatic (common bile duct) cholestasis
- Corticosteroid excess (dogs only)
  - Hyperadrenocorticism
  - Exogenous glucocorticoid drugs
- Drug therapy
  - Anticonvulsant/Barbiturates
- Primary hepatocyte injury
  - See ALT
- Nephrotoxicoses (e.g., aminoglycosides) will cause increased urine GGT

## Related Findings

- Other hepatic enzymes may also be increased with primary cholestatic (ALP) or hepatocellular (ALT, AST) disease.
- Bilirubin is often increased due to cholestasis.
- Cholesterol is increased with cholestasis or endocrine disease, but may be decreased with hepatic insufficiency or portosystemic shunts.

- Amylase, lipase and TLI may be increased in cases of pancreatitis.
- A hemogram may indicate the presence of inflammation, or a stress leukogram in cases of corticosteroid excess. The latter may also cause an increase in glucose concentration.
- Bile acids may be increased if the hepatic function is impaired.
- BUN, albumin and glucose serum concentrations may be decreased with chronic hepatobiliary disease.

#### **Other Laboratory Tests**

- Adrenocortical function tests
- CBC
- Hepatic cytology and histologic examinations
- Hepatic imaging
- Other liver chemical parameters, hepatic function tests/parameters
- Urinalysis
- Urine GGT:creatinine ratio

#### **References**

Duncan JR, et al. *Veterinary Laboratory Medicine: Clinical Pathology*. 3rd ed. Iowa State University Press: Ames, Ia; 1994.

Tilley L.P, et al. *The 5 Minute Veterinary Consult: Canine and Feline*. Williams & Wilkins: Baltimore, Md; 1997.

Willard MD, et al. *Small Animal Clinical Diagnosis by Laboratory Methods*. 3rd ed. WB Saunders: Philadelphia, Pa; 1999.

# Bilirubin

---

## Description

- Bilirubin is a waste product resulting from catabolism of heme in a variety of hemoproteins, mainly erythrocyte hemoglobin.
- A small amount is also produced by the breakdown of hepatic cytochrome P-450, tryptophan pyrrolase and myoglobin.
- Hemoglobin is broken down to heme, iron and globin mainly within macrophages. The heme molecule is further degraded to biliverdin and then to bilirubin (unconjugated) before release back into the circulation.
- On leaving the cell, the unconjugated bilirubin binds to albumin, is transported to the liver and bound by receptors within the hepatocyte membrane. Bilirubin disassociates from albumin, and is taken into the cell by carrier-mediated transport. The bilirubin is then conjugated with glucuronic acid, which renders bilirubin water-soluble. Conjugated bilirubin is secreted into bile canaliculi to be excreted into the intestinal tract via the bile duct. Resident bacteria convert conjugated bilirubin to urobilinogen, which is then excreted via the feces.
- The serum levels of bilirubin are proportional to the rate of hemoglobin breakdown, subsequent removal from the serum by hepatocytes and excretion into the intestinal tract.
- Most methods of bilirubin assay are based on the diazo reaction. With the addition of reagent, color develops rapidly (direct reaction). After addition of alcohol, further color development occurs (indirect reaction). The direct-reacting component is equivalent to conjugated bilirubin, and the indirect reaction gives a measurement of the total serum bilirubin. The difference between these two results gives a derived value of unconjugated bilirubin.
- The proportions of direct and indirect bilirubin fractions have been investigated in order to differentiate between hemolytic or cholestatic disease. However, the proportions vary greatly and are of limited clinical use.

## Values Below Reference Range

### Common Causes

Not clinically significant.

## Values Above Reference Range

Bilirubinemia

### Common Causes

- **Liver disease**
  - Hepatocellular injury
  - Toxic injury
  - Ascending bacterial cholangitis
  - Neoplasia
- Cholestatic disorders
  - Pancreatitis
  - Neoplasia
- Bile duct rupture
- Reduced hepatic mass
  - Porto-systemic shunt
  - Cirrhosis
  - Neoplasia
- Acute hemolytic disease
- Artefact
  - Hemolysis of sample
  - Lipemia

### **Related Findings**

- PCV, red cell indices and erythrocyte morphology
  - May indicate the presence of hemolytic disease
- Liver enzymes
  - Will be elevated in cases of hepatocyte injury and cholestasis, see ALT and ALP
- Amylase, Lipase
  - may be elevated in cases of pancreatitis
- Bilirubinuria
  - Will precede and accompany elevations in conjugated bilirubin

### **Other Laboratory Tests**

- Hepatic biopsy
  - For definitive diagnosis of hepatocellular disease or chronic insufficiency
- Coombs test
  - To assess for the presence of anti-erythrocyte antibodies as part of immune-mediated hemolytic anemia
- Abdominocentesis
  - To rule out the presence of bile peritonitis

### **References**

Willard MD, Tvedten H, Turnwald GH. *Small Animal Clinical Diagnosis by Laboratory Methods*. 2nd ed. WB Saunders: Philadelphia, Pa; 1994.

Kaneko JJ. *Clinical Biochemistry of Domestic Animals*. 4th ed. Academic Press: San Diego, Ca; 1989.

Duncan JR, Prasse KW, Mahaffey EA. *Veterinary Laboratory Medicine: Clinical Pathology*. 3rd ed. Iowa State University Press: Ames, Ia; 1994.

# Cholesterol

---

## Description

- The total amount of serum cholesterol is under close homeostatic control.
- It is influenced by dietary intake, production in the liver from fatty acids, tissue utilization and hepatic uptake, metabolism and excretion as bile acids into the gastrointestinal tract.

## Values Below Reference Range

Hypocholesterolemia

### Common Causes

- Decreased uptake
  - Low-fat diet
  - Intestinal malabsorption/maldigestion
  - Severy malnutrition
- Decreased production
  - Hepatic insufficiency such as with cirrhosis, portosystemic shunts

### Related Findings

- Low BUN, hypoalbuminemia
  - Also due to hepatic insufficiency, or severe intestinal disease with malassimilation
- Bilirubin
  - May be elevated with hepatic insufficiency
- CBC
  - Mild, nonregenerative anemia due to chronic disease

### Other Laboratory Tests

- Fecal examination, TLI, intestinal biopsy
  - To assess for possible maldigestion/malabsorption syndromes
- Pre- and postprandial bile acids
  - To assess for hepatic insufficiency as a result of congenital or acquired atrophy or portosystemic shunts

## Values Above Reference Range

Hypercholesterolemia

### Common Causes

- Hyperlipidemic states
  - Post-prandial
- Increased fat mobilization

- Diabetes mellitus, starvation/hyperlipidemia in horses, hyperadrenocorticism, steatitis in cats
- Decreased fat catabolism
  - Hypothyroidism
- Primary idiopathic hyperlipidemias
- Liver and biliary system diseases with cholestasis
  - See ALP, bilirubin
- Miscellaneous
  - Pancreatitis
  - Nephrotic syndrome
  - Glomerulonephritis

### **Related Findings**

- Glucose
  - Increased with diabetes mellitus, and occasionally with hyperadrenocorticism, pancreatitis
- Proteinuria, hypoalbuminemia and edema
  - Characteristic of nephrotic syndrome
- Pancreatitis
  - Inflammatory leukogram with pancreatitis,
- Hyperadrenocorticism
  - Stress leukogram with hyperadrenocorticism
- Bilirubin, ALT, AST, ALP, GGT
  - Increased with primary hepatocellular or cholestatic disorders
- Amylase, lipase, TLI
  - Elevated with pancreatitis

### **Other Laboratory Tests**

- Fructosamine, beta-hydroxybutyrate
  - To assess for the presence of diabetes mellitus
- Urinalysis
  - Proteinuria with nephrotic syndrome, glycosuria with diabetes mellitus
- T4
  - To definitively diagnose hypothyroidism
- Adrenocortical function tests
  - To assess for possible hyperadrenocorticism



## References

Willard MD, Tvedten H, Turnwald GH. *Small Animal Clinical Diagnosis by Laboratory Methods*. 2nd ed. WB Saunders: Philadelphia, Pa; 1994.

Kaneko JJ. *Clinical Biochemistry of Domestic Animals*. 4th ed. Academic Press: San Diego, Ca; 1989.

# Amylase

---

## Description

- Amylase is a calcium-dependent metalloenzyme that catalyzes the hydrolysis of complex carbohydrates.
- Amylase is synthesized in an active form.
- The pancreas secretes alkaline fluid that prevents acid-induced denaturation of the enzyme and provides an optimal environment for enzyme activity.
- Tissue sources: pancreas, stomach, kidney, small intestine, uterus, others
- Serum amylase activity does not decrease proportionally as the functional exocrine pancreatic tissue mass decreases in exocrine pancreatic insufficiency (EPI); therefore, amylase is of no value as a marker of EPI.

## Values Below Reference Range

### Decreased amylase

#### Common Causes

Acute pancreatitis (cats)

#### Other Laboratory Tests

- CBC
- Urinalysis
- Bilirubin
- Hepatic enzymes
- Glucose
  - May be increased with necrotizing pancreatitis
  - May be decreased with feline suppurative pancreatitis
- Calcium
- Cholesterol
- Tryglyceride
- Lipase
- Trypsin-like immunoreactivity (TLI)

## Values Above Reference Range

### Increased amylase

#### Common Causes

- Pancreatitis (3–4-fold increase in amylase concentration, dogs and horses)
  - Pancreatic acinar cell damage
  - Pancreatic duct obstruction

- Gastritis
- Gastritis neoplasia
- Hepatic disease
- Renal disease (up to 2.5-fold increase in amylase concentration, dogs)
- Decreased glomerular filtration rate (GFR), azotemia

### Related Findings

- Corticosteroids do not reliably increase serum amylase concentration.
- If the measured amylase concentration of abdominal fluid exceeds that of serum, differential diagnoses include pancreatic disease and intestinal rupture.

### Other Laboratory Tests

- CBC
- Urinalysis
- Hepatic enzymes
- Glucose
  - May be increased with necrotizing pancreatitis
  - May be decreased with feline suppurative pancreatitis
- Calcium
- Cholesterol
- Tryglyceride
- Lipase
- Canine pancreatic lipase immunoreactivity (c-PLI)
- Trypsin-like immunoreactivity (TLI)

### References

- Willard MD, Twedt DC, Tvedten H, Turnwald GH, eds. Gastrointestinal, pancreatic and hepatic disorders. In: *Small Animal Clinical Diagnosis by Laboratory Methods*. 3rd ed. WB Saunders: Philadelphia, Pa; 1999.
- Brobst DF, Kaneko JJ, Harvey JW, Bruss ML, eds. Pancreatic function. In: *Clinical Biochemistry of Domestic Animals*. 5th ed. Academic Press: New York; 1997.
- Strombeck DR, Guilford WG, Center SA, Strombeck DR, Williams DA, Meyer DJ, eds. Small and large intestine: Normal structure and function. In: *Strombeck's Small Animal Gastroenterology*. 3rd ed. WB Saunders: Philadelphia, Pa; 1996.
- Williams DA, Guilford WG, Center SA, Strombeck DR, Meyer DJ, eds. The pancreas. In: *Strombeck's Small Animal Gastroenterology*. 3rd ed. WB Saunders: Philadelphia, Pa; 1996.

# Lipase

---

## Description

- Lipase activity is used as a marker of pancreatitis. The sensitivity and the specificity of the assay is dependent upon the methodology used. Increased specificity can be attained if the values are greater than three-fold of the upper end of the normal reference range, unless baseline data is established for the individual patient's lipase values. However, even following this guideline, there are significant numbers of pancreatitis cases that will not have a significant increase in lipase activity.
- Lipase may originate from pancreatic or nonpancreatic sources, such as the gastrointestinal mucosa, which makes it difficult to interpret increases of lipase activity less than three-fold above the high end of the reference range.
- Extra-pancreatic factors that influence lipase concentration include:
  - Glucocorticoids therapy in which lipase increases five-fold without concurrent increases in amylase activity
  - Renal disease or severe dehydration with which there is decreased lipase clearance by the kidneys (up to three-fold increase)
  - Liver disease

## Values Below Reference Range

### Common Causes

Not clinically significant.

## Values Above Reference Range

### Common Causes

- Acute pancreatitis—magnitude of increase doesn't reflect severity of disease
- Drug therapy—corticosteroids, heparin
- Hepatic disease
- Kidney disease
- Neoplasia—pancreatic adenocarcinoma, gastric carcinoma

### Related Findings

- In cats with acute pancreatitis, lipase concentration doesn't increase to levels seen in dogs and, in fact, some cats have an increase in lipase without concurrent increases in amylase concentration.

### Other Laboratory Tests

- CBC, biochemistry profile and urinalysis
- Abdominal ultrasound
- Spec cPL™ Test, canine pancreatic lipase immunoreactivity (cPLI)
- Feline pancreatic lipase immunoreactivity (fPLI)
- Histopathology

## Reference

Williams DA, ed. The Pancreas. *Strombecks' Small Animal Gastroenterology*. 3rd ed. WB Saunders: Philadelphia, Pa; 1996.

## Urine Protein:Creatinine Ratio (UPC)

---

### Description

- The urine protein:creatinine (UPC) ratio is a simple and rapid test for the detection and quantification of proteinuria. Unlike other qualitative and semi-quantitative tests, the UPC ratio is not affected by urine concentration and volume. The urine protein:creatinine ratio offers the accuracy of 24-hour urine protein measurements without the need to perform the 24-hour urine collection.
- The urine protein:creatinine ratio is obtained by dividing the protein concentration [UPRO] (mg/dL) by the creatinine concentration [UCRE] (mg/dL). The result is a unitless ratio.
- Appropriate interpretation of urine protein:creatinine ratio results requires that you first localize the protein loss to the kidneys and then determine persistence.

### Localization

Causes of proteinuria can be prerenal or postrenal. *It is imperative that prerenal and postrenal causes of proteinuria are ruled out before assessing your UPC value.* This can be done by examining the patient's history and clinical signs, biochemical profile, CBC and complete urinalysis (including urine sediment examination and determination of urine specific gravity).

- **Prerenal:** evaluate for Bence Jones proteinuria, myoglobinuria, hemoglobinuria
- **Postrenal:** evaluate urine sediment for signs of hemorrhage, inflammation, infection and neoplasia
- **Renal:** determine level of azotemia

### Persistence

Determination of persistence of proteinuria is necessary to rule out transient elevations of urine protein. Determine persistence of proteinuria as needed given the level of azotemia and other clinical signs. In questionable cases, the 2004 ACVIM Forum Consensus Statement (Small Animal): *Assessment and Management of Proteinuria in Dogs and Cats* recommends repeating the UPC ratio on three or more occasions, at least two weeks apart.

### Evaluation

Once you have addressed localization and determination of persistence, you should evaluate your urine protein:creatinine ratio results in light of the patient's level of azotemia. The following ranges are in accordance with the recommendations from the 2004 ACVIM Forum Consensus Statement (Small Animal): *Assessment and Management of Proteinuria in Dogs and Cats*.

#### **Nonazotemic and persistent proteinuria with inactive urine sediment (dogs and cats):**

- UPC <0.5 no significant proteinuria
- UPC ≥0.5 <1.0 requires further monitoring
- UPC ≥1.0<2.0 proteinuria
- UPC ≥2.0 significant proteinuria

#### **Azotemic dogs and persistent proteinuria with inactive urine sediment (dogs):**

- UPC <0.5 no significant proteinuria
- UPC ≥0.5 significant proteinuria

#### **Azotemic cats and persistent proteinuria with inactive urine sediment (cats):**

- UPC <0.4 no significant proteinuria
- UPC ≥0.4 significant proteinuria

Once renal disease is established, you should determine if any underlying cause is present and address that as needed. Potential beneficial ancillary tests include: radiology, ultrasonography, blood pressure evaluation, infectious disease testing, endocrine testing and autoimmune testing.

The urine protein:creatinine ratio can be used as a prognostic indicator, with higher ratios correlating to a worsening prognosis. However, it is worth noting that in severe cases of chronic renal disease, the urine P:C ratio may decrease. This is due to the

fact that as plasma creatinine increases and the number of functioning nephrons decreases, the amount of urinary protein loss is reduced.

**More info on [UPC](#)**