MEDLAB BASICS · STUDY SHEET

Clinical Chemistry: Analytes and Disease Patterns

Glucose and HbA1c, lipids, proteins and electrophoresis, nonprotein nitrogen, bilirubin and jaundice, liver and other enzymes, cardiac and tumor markers, porphyrins

53 sources + more detail
medlabbasics.com/sheets/lab-chemistry-analytes.html
checked Nov 2026

1 Diabetes: the diagnostic criteria

ADA Standards of Care 2026, Tables 2.1 and 2.2. ASCP ranges: fasting glucose 74–100 mg/dL (4.1–5.6 mmol/L); HbA1c < 5.7% (< 39 mmol/mol).

TEST (NONPREGNANT ADULT)	DIABETES	PREDIABETES
HbA1c (NGSP-certified method, DCCT-standardized)	≥ 6.5% (≥ 48 mmol/mol)	5.7–6.4% (39–47 mmol/mol)
Fasting plasma glucose (no calories for at least 8 h)	≥ 126 mg/dL (≥ 7.0 mmol/L)	100–125 mg/dL (5.6–6.9 mmol/L): impaired fasting glucose
2-h plasma glucose, 75-g OGTT	≥ 200 mg/dL (≥ 11.1 mmol/L)	140–199 mg/dL (7.8–11.0 mmol/L): impaired glucose tolerance
Random plasma glucose with classic symptoms or a hyperglycemic crisis	≥ 200 mg/dL (≥ 11.1 mmol/L)	Not used

- Without unequivocal hyperglycemia, diabetes needs two abnormal results: two different tests on the same sample (for example HbA1c and fasting glucose) or the same test on two occasions.
- If two different tests disagree, the one above its cut point is repeated; a confirmed abnormal result makes the diagnosis even if the other test is normal.
- The OGTT load is the equivalent of 75 g anhydrous glucose dissolved in water, after a fast of at least 8 hours.

ADA 2026 Table 2.8.

GESTATIONAL DIABETES (24–28 WEEKS)	THRESHOLDS (MET OR EXCEEDED)	DIAGNOSIS
One-step: 75-g OGTT, fasting	Fasting 92 mg/dL (5.1 mmol/L); 1 h 180 (10.0); 2 h 153 (8.5)	Any one value
Two-step, step 1: 50-g load, nonfasting, 1 h	130, 135 or 140 mg/dL (7.2, 7.5 or 7.8 mmol/L), set locally	At or above: go to step 2
Two-step, step 2: 100-g 3-h OGTT, fasting (Carpenter-Coustan)	Fasting 95 (5.3); 1 h 180 (10.0); 2 h 155 (8.6); 3 h 140 (7.8)	At least two values (ACOG accepts one)

EXAM TIP The ASCP fasting glucose range ends at 100 mg/dL (5.6 mmol/L), the same value where ADA impaired fasting glucose begins. A fasting glucose of 100–125 mg/dL is prediabetes, not diabetes, and one result above a cut point is not a diagnosis unless the patient has symptoms or a crisis.

2 HbA1c, glucose specimens and hypoglycemia

ADA 2026 Section 2 and NGSP (updated October 2026).

CONDITION	EFFECT ON HBA1C	WHAT TO DO
Shortened red cell survival: hemolytic anemia, recovery from acute blood loss, increased red cell turnover	Falsely low with every method	Use plasma glucose criteria; consider fructosamine or glycated albumin
Iron deficiency anemia (including late pregnancy)	Higher; falls with iron treatment	Use glucose until iron deficiency is treated
Hemoglobin variant traits (HbS, HbC, HbE, HbD), raised HbF, carbamylated Hb in renal failure	Method-dependent bias	Check the method against the NGSP interference table; use a method without interference
HbSS, HbCC, HbSC disease	Unreliable (anemia, increased red cell turnover, transfusions)	Glycated albumin or glycated serum protein
Pregnancy, HIV, G6PD deficiency, erythropoietin therapy, hemodialysis	Altered relation to glycemia (dialysis: underestimates; G6PD G202A: about 0.7–0.8% lower)	Diagnose with plasma glucose

- Glucose keeps falling in an unseparated specimen because cells consume it (glycolysis): separate plasma at once or keep the sample on ice, or collect in a tube with a glycolytic inhibitor. Very high WBC or red cell counts (leukemia, polycythemia) make the loss faster: pseudohypoglycemia.
- HbA1c needs no fasting, is stable before analysis and is a whole-blood test; glucose is plasma (or serum) and depends on sample type and source.
- Hypoglycemia outside diabetes treatment needs Whipple's triad: symptoms, a low plasma glucose (below 55 mg/dL, 3.0 mmol/L) and relief when glucose is given.

Measured from blood drawn at the time of hypoglycemia (Looi and Lawler 2025; Merck).

CAUSE OF HYPOGLYCEMIA	INSULIN	C-PEPTIDE AND PROINSULIN	KEY EXTRA TEST
Exogenous insulin (including surreptitious use)	High	Low (own secretion suppressed)	None needed; the C-peptide gap is the clue
Sulfonylurea or other secretagogue	High or not suppressed	High or not suppressed	Serum or urine screen for oral hypoglycemic agents
Insulinoma	High or not suppressed	High or not suppressed	Drug screen negative; beta-hydroxybutyrate low
Insulin autoimmune syndrome	Very high (often above 1000 pmol/L)	High	Insulin antibodies
Non-islet cell tumor (IGF-II)	Low	Low; ketones inappropriately low	IGF-II/IGF-I ratio (above 20 in 94% of cases in one series)

SAFETY A low glucose on a specimen that sat unspun for hours is probably glycolysis, not the patient. Do not report it as a true value without a comment; ask for a properly handled repeat. Call a true low glucose according to the laboratory's critical value list.

3 Lipids and lipoproteins

Merck, Overview of Lipid Metabolism.

LIPOPROTEIN	MADE IN	MAIN CARGO	MAIN APOLIPOPROTEIN
Chylomicrons (largest)	Intestine	Dietary triglyceride	Apo B-48 (C-II activates LPL; E clears remnants)
VLDL	Liver	Endogenous triglyceride	Apo B-100 (C-II, E)
IDL	From VLDL by LPL	Cholesterol-rich remnant	Apo B-100, E
LDL	From VLDL and IDL	Most cholesterol-rich of all	Apo B-100 (LDL receptor ligand)
HDL	Liver and intestine	Cholesterol carried back from tissues (reverse transport)	Apo A-I (LCAT esterifies its cholesterol)
Lp(a)	Liver	LDL-like particle	Apo B-100 linked to apo(a), which resembles plasminogen

- Total cholesterol, triglycerides and HDL cholesterol are measured; LDL cholesterol is measured directly or calculated.
- Friedewald (mg/dL): LDL = total cholesterol − HDL − triglycerides ÷ 5. In mmol/L divide triglycerides by 2.2. Valid only when triglycerides are below 400 mg/dL (4.5 mmol/L); above that, measure LDL directly. Current US practice prefers the Martin-Hopkins or Sampson/NIH equations.
- Non-HDL cholesterol = total cholesterol − HDL. It and apo B count all atherogenic particles (VLDL, IDL, LDL, remnants, Lp(a)) and predict risk better than LDL when triglycerides are high.
- Fasting is not routinely required. Repeat fasting when nonfasting triglycerides are high (Merck: above 400 mg/dL, 4.5 mmol/L; EAS/EFLM: above 440 mg/dL, 5 mmol/L) or with a known triglyceride disorder.
- Postpone risk testing during acute illness: triglycerides and Lp(a) rise and cholesterol falls. A panel drawn within 24 hours of a myocardial infarction is usually usable.

See the LDL Friedewald calculator. Conversion: cholesterol mg/dL × 0.02586 = mmol/L; triglyceride mg/dL × 0.01129 = mmol/L (the factors the Conventional and SI Units calculator uses).

WORKED EXAMPLE	MG/DL	MMOL/L
Total cholesterol, HDL, triglycerides	220, 50, 150	5.69, 1.29, 1.69
VLDL estimate (TG ÷ 5 or ÷ 2.2)	30	0.77
LDL (Friedewald)	220 − 50 − 30 = 140	5.69 − 1.29 − 0.77 = 3.63
Non-HDL	220 − 50 = 170	5.69 − 1.29 = 4.40

4 Proteins and serum protein electrophoresis

- Albumin is made in the liver and is about 60% of serum protein. Globulin = total protein − albumin; A/G ratio = albumin ÷ globulin, normally a little above 1.
- Example: total protein 7.0 g/dL, albumin 4.0 g/dL: globulin 3.0 g/dL, A/G 1.3. A reversed ratio (globulin above albumin) points to low albumin, high globulin or both.
- Dehydration raises total protein and albumin together; liver disease, malnutrition, malabsorption, nephrotic syndrome, protein-losing enteropathy and burns lower them.

testing.com protein electrophoresis table.

FRACTION (ANODE TO CATHODE)	MAIN PROTEINS	LOW IN	HIGH IN
Albumin	Albumin	Malnutrition, nephrotic syndrome, liver disease, inflammation	Dehydration
Alpha-1	Alpha-1 antitrypsin, alpha-1 acid glycoprotein	Alpha-1 antitrypsin deficiency, severe liver disease	Inflammation
Alpha-2	Alpha-2 macroglobulin, haptoglobin, ceruloplasmin	Hemolysis (haptoglobin used up), severe liver disease	Nephrotic syndrome, acute or chronic inflammation
Beta (1 and 2)	Transferrin, C3, LDL	Malnutrition	Iron deficiency (transferrin), high cholesterol, some monoclonal proteins
Gamma	Immunoglobulins	Inherited and secondary immunodeficiency	Polyclonal: infection, autoimmune disease, cirrhosis. Monoclonal: myeloma, MGUS, Waldenström, lymphoma

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4 Proteins and serum protein electrophoresis CONTINUED

PATTERN	WHAT THE TRACING SHOWS
Acute phase (inflammation)	Albumin down; alpha-1 and alpha-2 up
Chronic inflammation	Broad (polyclonal) rise in gamma
Nephrotic syndrome	Albumin markedly low; alpha-2 and beta relatively high; gamma not raised
Cirrhosis	Albumin low; polyclonal gamma rise with beta-gamma bridging (no dip between beta and gamma) from raised polyclonal IgA
Monoclonal gammopathy	A narrow, tall band (M-spike), usually in gamma, sometimes beta; type it by immunofixation
Hypogammaglobulinemia	Gamma faint or nearly absent
Alpha-1 antitrypsin deficiency	Alpha-1 band nearly absent, rest normal

EXAM TIP Beta-gamma bridging is the cirrhosis clue; a single narrow spike is the myeloma clue. A spike is called monoclonal only after immunofixation shows a single light-chain type (with its one heavy chain, or as free light chain alone). Plasma, or serum that clotted incompletely, leaves fibrinogen, a coagulation protein absent from properly clotted serum, which runs as a discrete extra band near the beta-gamma region and shows no immunoglobulin on immunofixation.

5 Nonprotein nitrogen: urea, creatinine, uric acid, ammonia

MedlinePlus (BUN, uric acid), testing.com (creatinine, ammonia), Merck (urinary indices, gout), Elitek label. BUN mg/dL × 0.357 = mmol/L; creatinine mg/dL × 88.4 = µmol/L.

ANALYTE	WHAT IT REFLECTS	RAISED IN	PITFALL
BUN (ASCP 6–20 mg/dL, 2.1–7.1 mmol/L urea)	Urea, which the liver makes from ammonia	Dehydration, heart failure, GI bleeding, high protein intake, kidney disease, obstruction	Low in liver failure, low protein intake, overhydration
Creatinine (ASCP 0.8–1.2 mg/dL, 71–106 µmol/L)	Muscle breakdown product, cleared by the kidneys	Falling GFR	Depends on muscle mass: low muscle mass hides a low GFR
BUN/creatinine ratio (conventional units)	How urea handling compares with filtration	Above 20: prerenal azotemia (low perfusion)	Below 10: tubular injury (ATN)
Uric acid	End product of purine metabolism	Gout (urate above 6.8 mg/dL, 0.40 mmol/L, saturates), tumor lysis, renal failure, leukemia, lead	Rasburicase keeps destroying urate in the tube at room temperature
Ammonia	Protein digestion by gut bacteria; liver turns it into urea	Liver failure (hepatic encephalopathy), metabolic disorders	Falsely high from delay, warmth or a clotted (serum) tube, and from fist clenching or smoking at collection

SAFETY Ammonia: EDTA plasma, on ice, processed within about 15 minutes; a serum tube that clotted at room temperature is rejected. Uric acid on rasburicase: prechilled heparin tube, kept in an ice-water bath, assayed within 4 hours. A falsely low urate can hide tumor lysis.

Creatinine clearance and timed urine: see the Measured Creatinine Clearance and eGFR CKD-EPI 2021 calculators and the Laboratory Mathematics page.

6 Bilirubin and the jaundice table

- Heme (mostly from old red cells) → biliverdin → unconjugated bilirubin, carried on albumin, insoluble in water, never in urine.
- Liver conjugates it with glucuronic acid (UGT1A1) → conjugated bilirubin, water soluble, excreted in bile; it can appear in urine when the plasma level is high.
- Gut bacteria reduce bilirubin to urobilinogens; most leave in feces (stool color), some are reabsorbed and re-excreted by the liver, and a small part (2–5%) reaches the urine.
- Diazo method: direct bilirubin is what reacts without an accelerator (conjugated); total is measured with an accelerator (caffeine-benzoate); indirect = total − direct.

Merck (Jaundice; Laboratory Tests of the Liver; Inborn Disorders); Guerra Ruiz 2021 for urobilinogen. Bilirubin mg/dL × 17.1 = µmol/L.

TYPE	SERUM BILIRUBIN	URINE BILIRUBIN	URINE UROBILINOGEN	OTHER CLUES
Prehepatic (hemolysis)	Unconjugated (indirect over 85% of total); usually under 3.5 mg/dL (about 60 µmol/L)	Negative	Increased	Haptoglobin low, LD and retics up, AST/ALT normal
Gilbert syndrome (uptake and conjugation defect)	Unconjugated, 2–5 mg/dL (34–86 µmol/L), rises with fasting	Negative	Normal	No anemia, no reticulocytosis, other liver tests normal
Hepatic (hepatocellular injury)	Both fractions rise	Positive	Normal to increased	AST and ALT usually above 500 U/L
Posthepatic (obstruction: stone, pancreatic cancer)	Conjugated (direct over 50% of total)	Positive (dark urine)	Normal to low; absent if obstruction is complete	Pale stools; ALP and GGT over 3 × normal, AST/ALT under 200 U/L
Dubin-Johnson, Rotor	Conjugated	Positive	Normal	Liver enzymes normal

EXAM TIP Indirect bilirubin is calculated, not measured: total 3.0 mg/dL (51 µmol/L) − direct 0.3 mg/dL (5 µmol/L) = 2.7 mg/dL (46 µmol/L), 90% unconjugated: think hemolysis or Gilbert. Bilirubin in the urine always means conjugated bilirubin.

7 Liver enzyme patterns

Merck, Laboratory Tests of the Liver and Gallbladder; Jaundice; Cirrhosis.

PATTERN	AST AND ALT	ALP AND GGT	BILIRUBIN AND PT
Acute hepatocellular necrosis (viral, toxin or drug, ischemic, autoimmune)	Marked, above 500 U/L	Normal to mildly raised	PT prolongs if function is severely impaired
Chronic hepatocellular disease, MASLD, cirrhosis	Mild, under 300 U/L (can be normal)	Often normal	Albumin low and PT long in cirrhosis
Alcohol-related liver disease	AST:ALT above 2, both usually under 300 U/L	GGT raised (induced)	Variable
Cholestasis or obstruction	Mild, under 200 U/L (a passing stone can spike them briefly)	ALP and GGT over 3 × normal; ALP rises 1–2 days after obstruction	Conjugated bilirubin up; PT corrects with vitamin K
Infiltration (tumor, granulomas, amyloid)	Mild	Mild to moderate ALP rise	Usually normal
Ischemic (hypoxic) hepatitis	Marked	Mild	LD typically raised as well

EXAM TIP A high ALP with a normal GGT is not from the liver: think bone (Paget disease, growth in children), pregnancy (placenta) or intestine. GGT does not rise from bone, childhood or pregnancy, so it is the quick way to place an ALP.

8 Cardiac markers

Merck (Acute MI, ACS, Heart Failure), MedlinePlus (CK isoenzymes), Gallacher 2021, Nishikimi 2025.

MARKER	USE	INTERPRETATION	PITFALLS
hs-troponin I or T	Marker of choice for myocardial injury and MI	Elevated means above the assay's (sex-specific) 99th percentile upper reference limit; sample at presentation and 1–2 h later (0 and 3–6 h with a standard assay). A rise of more than 20 to 50% makes MI much more likely	Stable elevation in kidney disease and heart failure; cTnT from diseased skeletal muscle; macrotroponin
CK-MB	Older infarction marker	Rises 3–6 h after MI, peaks 12–24 h, normal again 12–48 h	Defibrillation, myocarditis, cardiac surgery, CK-BB and macro-CK
BNP	Heart failure diagnosis and monitoring	Common threshold 100 pg/mL (100 ng/L)	Lower in obesity; up to 30% of acute HFpEF below 100; raised by neprilysin inhibitors
NT-proBNP	Same; made from the same precursor as BNP	Kidney disease and age raise natriuretic peptides	Not a neprilysin substrate: use it to follow a patient on sacubitril/valsartan

SAFETY A troponin above the 99th percentile that is rising or falling is called to the clinician at once. A stable, unchanging elevation with no clinical story is still reported, but think of chronic injury, kidney disease, skeletal myopathy (troponin T) or macrotroponin before an assay error.

MORE Hypoglycemia workup in detail

- In treated diabetes, severity is graded by glucose: level 1 below 70 mg/dL (3.9 mmol/L) but at least 54 (3.0); level 2 below 54 mg/dL (3.0 mmol/L); level 3 needs another person's help.
- Outside diabetes treatment, blood is drawn during symptoms into a tube with a glycolytic inhibitor. If glucose is normal, hypoglycemia is excluded. If low with no obvious cause, insulin, C-peptide, proinsulin, insulin antibodies and a sulfonylurea screen are run on the same specimen.
- Supervised fast (up to 72 h; 48 h is usually enough): glucose at baseline, with symptoms and every 4–6 h (every 1–2 h once below 70 mg/dL). Insulin, C-peptide and proinsulin are drawn when glucose is below 55 mg/dL (3.05 mmol/L). The fast ends at 72 h or earlier at 45 mg/dL (2.5 mmol/L) or less with symptoms; end-of-fast tests are beta-hydroxybutyrate (low in insulinoma), a sulfonylurea level and the glucose response to IV glucagon.
- Postprandial patterns (for example after bariatric surgery) are tested with a mixed meal: glucose, insulin and C-peptide every 30 minutes for up to 4 hours.
- Home meters and continuous monitors are unreliable in the hypoglycemic range; fingersticks also read low with poor circulation to the digits.
- Healthy women can have fasting glucose as low as 50 mg/dL (2.8 mmol/L) without symptoms.

C-peptide and proinsulin mark the patient's own insulin secretion: both are suppressed when the insulin is injected and not suppressed when the pancreas is being driven (secretagogue, insulinoma). For diabetes classification in insulin-treated patients (ADA), a C-peptide of at least 600 pmol/L (1.8 ng/mL) is valid whatever the circumstances of testing, and a very low result (under 80 pmol/L, 0.24 ng/mL) does not need to be repeated.

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