

# Lab Test Interpretation Table\*

Normal Range**	Interpretation Tips
<b>Hematology</b>	
<p><b>White Blood Cell Count (WBC) &amp; Differential</b></p> <p>Leukocytes/WBC <math>4 - 10 \times 10^9/L</math></p> <p>Neutrophils</p> <ul style="list-style-type: none"> <li>- Absolute Neutrophil Count (ANC) = <math>2 - 7.5 \times 10^9/L</math></li> <li>- Calculated ANC = <math>WBC \times (\text{segs} + \text{bands}) / 100</math></li> <li>- Band neutrophils: <math>&lt; 0.7 \times 10^9/L</math></li> </ul> <p>Basophils <math>&lt; 0.2 \times 10^9/L</math></p> <p>Eosinophils <math>&lt; 0.7 \times 10^9/L</math></p> <p>Lymphocytes = <math>1 - 4 \times 10^9/L</math></p> <p>Monocytes = <math>0.1 - 0.8 \times 10^9/L</math></p>	<p>WBCs are measured as part of a complete blood count and differential (CBC &amp; diff). They protect the body from infection.</p> <p><b>Increased Counts</b></p> <ul style="list-style-type: none"> <li>- Leukocytosis and neutrophilia can be caused by infection, myeloproliferative disorders, inflammation, and medications. <ul style="list-style-type: none"> <li>o In cancer patients, supportive medications such as corticosteroids and colony stimulating factors can cause elevated counts. Treatment is not required unless they are associated with bone pain, which may improve with analgesic therapy.</li> <li>o When leukocytosis is accompanied by increased immature neutrophils (band neutrophils) and fever, infection is a likely cause. Band neutrophils often increase in number to fight infections (also called “a shift to the left”).</li> </ul> </li> <li>- Elevated lymphocyte counts are associated with increased risk of cytokine-release syndrome (see BC Cancer Protocol <a href="#">LYCHOPR</a>) or tumour lysis syndrome (see BC Cancer Protocol <a href="#">ULYVENETO</a>) and prophylaxis may be indicated. Consult respective protocol and/or tumor group chair for management recommendations.</li> </ul> <p><b>Decreased Counts</b></p> <ul style="list-style-type: none"> <li>- Leukocytopenia and neutropenia can result from nutritional deficiency, autoimmune disease, bone marrow infiltration (i.e., leukemia or myelodysplastic syndrome), radiation, and myelosuppression due to medications (including many cancer drugs). <ul style="list-style-type: none"> <li>o Many treatment protocols require dose adjustments or the addition of colony stimulating factors (e.g., filgrastim) if ANC drops below <math>1.5 \times 10^9/L</math>. Some protocols tolerate lower thresholds.</li> </ul> </li> <li>- Febrile neutropenia is defined as the presence of neutropenia plus concurrent fever (ANC <math>&lt; 1 \times 10^9/L</math> and single oral temperature of <math>\geq 38.3^\circ C</math> orally or a temperature of <math>\geq 38^\circ C</math> over 1 h). It is a medical emergency which requires treatment with antibiotics +/- supportive medications.</li> </ul>

Normal Range**	Interpretation Tips
<p><b>Platelets (Thrombocytes)</b> 150 – 400 x 10<sup>9</sup>/L</p>	<p>Platelets are measured as part of a complete blood count (CBC). They are involved in blood clotting.</p> <p><b>Increased Counts</b></p> <ul style="list-style-type: none"> <li>- Thrombocytosis can be caused by bone marrow disorders, various cancers, inflammatory disease, or surgical removal of the spleen. <ul style="list-style-type: none"> <li>o In patients with myeloproliferative disorders, thrombocytosis is generally caused by the malignancy.</li> <li>o In patients without myeloproliferative disorders, it is prudent to notify the prescriber if thrombocytosis occurs. Treatment is generally not required unless the patient is symptomatic.</li> </ul> </li> </ul> <p><b>Decreased Counts</b></p> <ul style="list-style-type: none"> <li>- Thrombocytopenia can be caused by bone marrow disorders, bone marrow infiltration (i.e., leukemia, lymphoma), viral infections, or cancer drug therapy and radiation. <ul style="list-style-type: none"> <li>o Many protocols require a dose reduction or delay if platelet count is &lt; 100 x 10<sup>9</sup>/L. Some protocols tolerate lower thresholds.</li> <li>o If the thrombocytopenia is disease-related, no dose change may be necessary.</li> </ul> </li> </ul>
<p><b>Red Blood Cells (RBCs, Erythrocytes)</b> Females: 3.5 – 5 x 10<sup>12</sup>/L Males: 4.2 – 5.4 x 10<sup>12</sup>/L</p> <p><b>Hemoglobin (Hgb)</b> Females: 115 – 155 g/L Males: 135 – 170 g/L</p>	<p>RBCs are measured as part of a complete blood count (CBC). They use hemoglobin (Hgb) to help deliver oxygen to body tissues.</p> <p><b>Increased Counts</b></p> <ul style="list-style-type: none"> <li>- Erythrocytosis and hemoglobinemia can occur to compensate for low oxygen levels (heart disease, lung disease, high altitude), or may be caused by other conditions such as polycythemia vera, dehydration, and kidney tumours that produce excess erythropoietin.</li> </ul> <p><b>Decreased Counts</b></p> <ul style="list-style-type: none"> <li>- Decreased Hgb and RBCs can result from chronic anemia, blood loss (hemorrhage, hemolysis), nutritional deficiency, bone marrow disorders, cancer, or medications (including many cancer treatment drugs).</li> </ul>
<b>Liver Function Tests (LFTs): for Synthetic Ability</b>	
<p><b>Albumin</b> 35 – 50 g/L</p>	<p>Albumin is a protein synthesized by the liver and can be an indicator of the liver's synthetic ability.</p> <p>However, because it has a long half-life of 20-30 days, and levels often remain normal even in acute</p>

Normal Range**	Interpretation Tips
	<p>disease, it is not always useful in assessing acute hepatic injury.</p> <ul style="list-style-type: none"> <li>- Decreased albumin levels can occur in chronic diseases such as cirrhosis, cancer and malnutrition.</li> </ul>
<p><b>Prothrombin Time (PT)</b> 8.7 – 11.5 seconds</p> <p><b>International Normalized Ratio (INR)</b> 0.8 – 1.2 seconds (normal)</p> <p><b>Partial Thromboplastin Time (PTT)</b> 60 – 70 seconds</p> <p><b>Activated Partial Thromboplastin Time (aPTT)</b> 30 - 40 seconds</p>	<p>The liver is responsible for synthesizing a number of different clotting factors. Unlike albumin, PT offers a good reflection of acute changes in liver function because of the short half-life of clotting factors.</p> <p>PT may be reported as a standardized INR value or tested in conjunction with partial thromboplastin time (PTT) or activated partial thromboplastin time (aPTT).</p> <ul style="list-style-type: none"> <li>- Liver damage can significantly prolong clotting time (e.g., PT) and increase the risk of bleeding. <ul style="list-style-type: none"> <li>o PT may rise to 50 sec or greater in acute liver failure.</li> <li>o PT is usually 2 to 5 times the upper limit of normal (ULN) in cirrhosis.</li> </ul> </li> <li>- INR, PT, PTT and aPTT times may deliberately be kept longer, at 1.5 to 2.5 times the normal value, for patients on anticoagulant therapy or patients with artificial heart valves.</li> <li>- Other factors that can prolong PT include vitamin K deficiency (an essential co-factor in the clotting cascade), inherited clotting factor deficiencies and certain types of leukemia.</li> </ul>
<b>Liver Function Tests (LFTs): for Hepatocellular Injury</b>	
<p><b>Alanine aminotransferase (ALT)</b> Females: &lt;36 U/L Males: &lt;50 U/L</p>	<p>ALT is an enzyme found primarily in hepatocytes, but also in the skeletal muscle, heart and kidneys. It is a useful marker of hepatic injury.</p> <ul style="list-style-type: none"> <li>- ALT is released into the blood when the liver is damaged or inflamed. Liver cancer or liver metastases may elevate ALT.</li> <li>- ALT is higher than AST (aspartate aminotransferase) in most types of liver disease (i.e., AST/ALT ratio &lt; 1).</li> <li>- Very high ALT levels (&gt; 1000 U/L) are most commonly due to viral hepatitis, ischemic hepatitis, or liver injury due to drug or toxin.</li> </ul>
<p><b>Aspartate Aminotransferase (AST)</b> &lt;36 U/L</p>	<p>AST is a less specific indicator of hepatic injury than ALT because it is found in the liver, heart, red blood cells, skeletal muscle, kidneys, brain and pancreas.</p> <ul style="list-style-type: none"> <li>- AST elevation generally indicates liver damage if ALT, bilirubin and ALP (alkaline phosphatase) are also elevated. Liver cancer or liver</li> </ul>

Normal Range**	Interpretation Tips
	<p>metastases may elevate AST.</p> <ul style="list-style-type: none"> <li>- Very high AST levels (&gt; 1000 U/L) are most commonly due to viral hepatitis, ischemic hepatitis or liver injury due to drug or toxin.</li> <li>- Elevated AST in relation to ALT (i.e., AST/ALT ratio &gt; 2), along with high GGT, can occur with alcoholic hepatitis.</li> <li>- Isolated AST elevation (without ALT elevation) may indicate cardiac or muscle disease. This is often accompanied by an elevated serum creatine kinase (CK), which can also indicate cardiac or muscle injury.</li> </ul>
<p><b>Lactate Dehydrogenase (LD, LDH)</b> &lt;225 U/L</p>	<p>LDH is an enzyme involved in energy production and is present in most tissues, but particularly in the heart, blood cells, kidneys, liver, lungs, and skeletal muscle.</p> <ul style="list-style-type: none"> <li>- LDH is released into the blood when cells are damaged or destroyed, making it a non-specific marker for tissue damage. <ul style="list-style-type: none"> <li>o Elevated LDH (i.e., &gt; 2 x ULN) is associated with a greater risk of Tumor Lysis Syndrome (TLS) in patients with aggressive hematologic cancers, or other cancers with a large tumour burden or high sensitivity to treatment.</li> </ul> </li> <li>- LDH can be used in oncology as a non-specific marker to monitor for tumour progression or as a prognostic factor for various cancers (e.g., lymphoma).</li> </ul>
<b>Liver Function Tests (LFTs): for Cholestasis</b>	
<p><b>Alkaline Phosphatase (Alk Phos, ALP)</b> Females: 35 – 120 U/L Males: 40 – 145 U/L</p>	<p>ALP is an enzyme found in several tissues throughout the body, but primarily in the liver, bile duct and bone.</p> <ul style="list-style-type: none"> <li>- ALP levels &gt; 3 times ULN are generally associated with cholestasis. Bile accumulation increases liver synthesis of ALP and GGT; levels tend to normalize within 2-4 weeks after the cholestasis is resolved. <ul style="list-style-type: none"> <li>o Biliary obstruction secondary to cancers, metastases, or hepatic infiltration by hematological cancers can markedly elevate ALP.</li> </ul> </li> <li>- ALP levels ≤ 3 times ULN in the presence of other elevated liver enzymes generally indicate a hepatocellular source. <ul style="list-style-type: none"> <li>o Liver cancer, liver metastases, and hepatic infiltration by hematological cancers can elevate ALP.</li> </ul> </li> </ul>

Normal Range**	Interpretation Tips
	<ul style="list-style-type: none"> <li>- ALP levels <math>\leq</math> 3 times ULN in the absence of other elevated liver enzymes generally indicate a non-hepatic source. <ul style="list-style-type: none"> <li>o Bone cancers (e.g., osteosarcomas) or bone metastases can elevate ALP.</li> </ul> </li> <li>- Some tumours without liver or bone involvement (e.g., renal, Hodgkin lymphoma) can secrete ALP or cause it to leak into the bloodstream.</li> </ul>
<p><b>Total Bilirubin (T.Bili)</b> &lt;17 umol/L</p>	<p>Bilirubin is a waste product of heme metabolism, primarily derived from red blood cell breakdown. It is conjugated by the liver to facilitate its excretion as bile into the gut.</p> <p>Total serum bilirubin represents the sum of unconjugated bilirubin (indirect/albumin-bound) and conjugated bilirubin (direct/unbound) present in the blood, with the majority (almost 100%) being unconjugated.</p> <ul style="list-style-type: none"> <li>- Bilirubin is not a sensitive indicator of hepatic dysfunction since total serum levels may be normal in the presence of liver injury. The liver has a reserve capacity to remove at least twice the normal daily bilirubin load without developing hyperbilirubinemia.</li> <li>- Bilirubin may be elevated in inherited liver disorders, hepatocellular injury or cholestasis, conditions that block the bile ducts (e.g., gallstones, pancreatic cancer), or hemolysis. <ul style="list-style-type: none"> <li>o High bilirubin levels accompanied by elevated ALT or AST, may indicate hepatitis or cirrhosis.</li> <li>o High bilirubin levels accompanied by elevations specifically in ALP and GGT, may suggest a cholestatic disorder.</li> </ul> </li> <li>- Bilirubin in the urine generally indicates underlying liver disease. Only conjugated bilirubin is water-soluble, and if the liver is damaged or unable to secrete it as bile, it can get filtered into the urine.</li> </ul>
<p><b>Gamma Glutamyl Transpeptidase (GGT, GGTP or GTP)</b> Females: &lt;31 U/L Males: &lt;49 U/L</p>	<p>GGT is an enzyme found primarily in the liver and bile ducts, but also in many other tissues throughout the body.</p> <ul style="list-style-type: none"> <li>- Since GGT levels quickly elevate with bile accumulation, it is considered the most sensitive test for cholestatic disorders.</li> <li>- As GGT is not found in the bone, it is useful in determining whether ALP elevations are</li> </ul>

Normal Range**	Interpretation Tips
	<p>secondary to liver or bone pathology. If both ALP and GGT are elevated, the source is likely the liver.</p> <ul style="list-style-type: none"> <li>- Elevated GGT, combined with an AST/ALT ratio &gt; 2, is suggestive of alcoholic liver disease.</li> </ul>
Renal Function Tests	
<p><b>Serum Creatinine (SCr)</b>            Females: 45 – 90 umol/L            Males: 45 – 110 umol/L</p>	<p>Creatinine is a waste product of muscle breakdown which gets removed from blood by the kidneys. Many different variables can affect its levels (e.g., dehydration, nutritional status, muscle mass, ingestion of meat, certain medications).</p> <ul style="list-style-type: none"> <li>- Increased SCr levels may suggest a variety of conditions caused by renal infection/inflammation (e.g., glomerulonephritis, pyelonephritis), renal obstruction (e.g., kidney stones, prostate disease), or reduced renal blood flow (e.g., dehydration, atherosclerosis, congestive heart failure, shock, or diabetes).</li> </ul>
<p><b>Blood Urea Nitrogen (BUN, urea nitrogen, or urea)</b>            2 – 9 mmol/L</p>	<p>Urea is a nitrogen waste product of protein and amino acid metabolism. The liver releases urea into blood and it is filtered by the kidneys into urine.</p> <ul style="list-style-type: none"> <li>- Increased urea levels may be caused by dehydration, kidney disease, increased protein intake or increased protein breakdown from muscle damage or GI bleeds.</li> </ul>
<p><b>Creatinine Clearance (CrCl), Glomerular Filtration Rate (GFR)</b>            eGFR &gt; 60 mL/min</p>	<p>CrCl measures the kidneys' ability to remove creatinine from blood, which helps to estimate GFR, the amount of blood filtered per minute through the kidneys.</p> <p>The most accurate method of assessing GFR is to measure it using a nuclear renogram, but this method is not commonly used. It is more common to estimate GFR by calculating CrCl using the Cockcroft-Gault formula (see <a href="#">Renal Function Tests</a> [Clinical Pharmacy Guide]).</p> <ul style="list-style-type: none"> <li>- Decreased CrCl rates may suggest decreased renal function.               <ul style="list-style-type: none"> <li>o Some protocols require dose changes or delays based on CrCl.</li> </ul> </li> <li>- Certain protocols with medications primarily eliminated via the kidneys (e.g. CISplatin, CARBOplatin) have baseline thresholds for CrCl or GFR.</li> </ul>
<p><b>Urine protein random (&lt;0.3 g/L)</b>  <b>Urine protein 24-hour (&lt;0.15 g/L)</b></p>	<p>Urine protein tests detect the amount of protein being released into the urine.</p> <p>Urine protein can be measured by using a dipstick, obtaining a random laboratory urine sample, or</p>

Normal Range**	Interpretation Tips
	<p>analyzing a 24-hour urine collection done by the patient at home.</p> <ul style="list-style-type: none"> <li>- Normal urinary protein levels are low; persistently elevated levels can suggest renal damage.</li> <li>- Increased protein in urine (proteinuria) may be related to multiple myeloma, medications toxic to the kidneys, bladder cancer, or urinary tract infection.</li> </ul>
Blood Chemistry Tests	
<p><b>Electrolytes (Lytes)</b></p>	<p>Electrolytes are minerals that carry a positive or negative electric charge in body fluids. They are required to maintain fluid balance and generate the electrical signals used to regulate heart rhythm, muscle contraction and nerve function. Electrolyte levels are regulated in the body through hormonal mechanisms and renal excretion.</p> <p>An electrolyte panel test generally includes sodium, potassium, chloride, and bicarbonate. Additional electrolytes monitored in cancer patients include calcium, magnesium and phosphate.</p> <ul style="list-style-type: none"> <li>- Abnormal electrolyte levels can be caused by the cancer itself, drug treatment (e.g., abiraterone, CISplatin), or treatment-related side effects (e.g., renal toxicity, excessive vomiting or diarrhea).</li> <li>- Generally, acute or severe changes in electrolyte levels require more aggressive management. <ul style="list-style-type: none"> <li>o QT prolongation of cardiac rhythm can be caused by electrolyte abnormalities such as hypokalemia, hypocalcemia or hypomagnesemia.</li> <li>o Tumour Lysis Syndrome (TLS) can cause both electrolyte and metabolic abnormalities such as hyperkalemia, hyperphosphatemia, hypocalcemia, and hyperuricemia.</li> </ul> </li> <li>- Patients with renal dysfunction may be at higher risk for electrolyte abnormalities.</li> <li>- Dehydration or other medications (e.g., furosemide, hydrocortisone) may also cause electrolyte imbalances.</li> </ul>
<p><b>Sodium (Na)</b> 135 – 145 mmol/L</p>	<p>Sodium, primarily an extracellular cation, helps regulate the amount of body fluids and helps cells in the nerves and muscles function normally.</p> <ul style="list-style-type: none"> <li>- Patients with either hyponatremia or hypernatremia may be asymptomatic, or may experience neurological symptoms (e.g., nausea, vomiting, malaise, headache, muscle twitching, restlessness and seizures).</li> </ul>

Normal Range**	Interpretation Tips
<p><b>Potassium (K)</b> 3.5 – 5 mmol/L</p>	<p>Potassium, primarily an intracellular cation, helps cells in the nerves, skeletal and cardiac muscles function normally.</p> <ul style="list-style-type: none"> <li>- Patient with hypokalemia or hyperkalemia may be asymptomatic or may experience muscle weakness or cardiac arrhythmias (i.e., ECG changes); cardiac monitoring may be required.</li> </ul>
<p><b>Calcium (Ca)</b> (2.1 – 2.6 mmol/L) <b>Ionized calcium</b> (1.12 – 1.31 mmol/L)</p>	<p>Calcium cations are essential for cell signaling, blood coagulation, maintenance of bones and teeth, and proper muscle, nerve and heart function. The vast majority of calcium is complexed in the bones. Of the small portion circulating in the blood, roughly one-half is “free” and metabolically active, while the rest is protein-bound (e.g., to albumin).</p> <p>A total calcium test is most frequently used to measure serum calcium levels because it is simpler to perform; it measures both free and bound forms. Results should be corrected for low albumin levels (see BC Cancer Supportive Care Protocol <a href="#">SCHYPCAL</a>). The ionized calcium test measures the free form of serum calcium only.</p> <ul style="list-style-type: none"> <li>- Hypercalcemia of malignancy is common in patients with advanced cancers and can be symptomatic, requiring treatment. It may be caused by hormonal mechanisms, or cancers affecting the bones.</li> </ul>
<p><b>Magnesium (Mg)</b> 0.64 – 0.98 mmol/L</p>	<p>Magnesium cations are important enzyme cofactors and are involved in energy production, protein synthesis, gene maintenance, nerve function, muscle contraction, and bone formation.</p> <ul style="list-style-type: none"> <li>- Hypomagnesemia can be induced by certain cancer medications such as CISplatin and PANitumumab, and associated treatment protocols may include magnesium supplementation guidelines.</li> </ul>
<p><b>Phosphate (PO<sub>4</sub>)</b> 0.8 – 1.5 mmol/L</p>	<p>Phosphate anions are vital for cell energy production, muscle and nerve function, blood coagulation and bone growth.</p> <p>A serum phosphate test measures inorganic phosphate levels, which are generally inversely regulated in proportion to serum calcium levels.</p> <ul style="list-style-type: none"> <li>- Hypophosphatemia may be caused by some tumours, but hyperphosphatemia from decreased renal function or TLS is more commonly seen in cancer patients.</li> </ul>
<b>Endocrine Tests</b>	
<p><b>Endocrine Tests</b></p>	<p>Endocrine tests measure various endocrine hormone levels and are used to assess if the endocrine glands</p>



Normal Range**	Interpretation Tips
	<p>are functioning correctly.</p> <ul style="list-style-type: none"> <li>- Endocrine gland tumours, such as some adrenal gland tumours, can cause increases in hormone production.</li> <li>- Checkpoint-inhibitor immunotherapies can induce immune-mediated adverse reactions that damage the endocrine glands. This could result in endocrine hormone deficits which may require supplementation (see Endocrine section in BC Cancer Supportive Care Protocol <a href="#">SCIMMUNE</a>).</li> </ul>
<p><b>Thyroid Stimulating Hormone (TSH)</b> 0.32 – 5.04 mU/L</p> <p><b>Thyroxine (T4 free)</b> 10.6 – 19.7 pmol/L</p> <p><b>Triiodothyronine (T3 free)</b> 2.6 – 5.8 pmol/L</p>	<p>TSH is produced by the pituitary gland as part of the body's feedback system to maintain stable amounts of the thyroid hormones, T3 and T4, in the body. T3 and T4 can be bound (inactive) or free (active); free levels are usually ordered.</p> <ul style="list-style-type: none"> <li>- Certain immunotherapy protocols require TSH monitoring. Since TSH level changes can be transient, they are usually repeated with T3 and/or T4 tests added to confirm the diagnosis.</li> <li>- Hypothyroidism may require thyroid supplementation. T4 is a more sensitive test than T3 for hypothyroid patients, while T3 is often useful to diagnose hyperthyroidism. <ul style="list-style-type: none"> <li>o Primary hypothyroidism reflected by an elevated TSH, along with low T4, is most common.</li> <li>o Secondary (central) hypothyroidism is reflected by low-to-normal TSH, along with low T4.</li> <li>o Subclinical hypothyroidism is defined as elevated TSH but normal T4.</li> </ul> </li> <li>- Hyperthyroidism is reflected by low TSH and elevated T3 and/or T4, and may require treatment.</li> </ul>
<p><b>AM Cortisol</b> 125 – 536 nmol/L</p> <p><b>Adrenocorticotrophic Hormone (ACTH)</b> 1.6 – 13.9 pmol/L</p>	<p>Cortisol is a hormone secreted by the adrenal glands. It is involved in protein, lipid, and carbohydrate metabolism and helps the body manage stress. Cortisol levels are generally highest early in the morning.</p> <p>ACTH is a hormone that stimulates the production of cortisol. ACTH is produced by the pituitary gland.</p> <ul style="list-style-type: none"> <li>- Immunotherapy-induced adrenal insufficiency or hypophysitis may require steroid supplementation.</li> </ul>
<p><b>Follicle-Stimulating Hormone (FSH)</b> Females: varies by menstrual phase</p>	<ul style="list-style-type: none"> <li>- Hormone levels can be used for screening and determining treatment response in patients receiving hormone treatment, such as estrogen levels in breast cancer or testosterone levels in</li> </ul>

Normal Range**	Interpretation Tips
<p>Males: &lt;9.5 U/L</p> <p><b>Luteinizing Hormone (LH)</b></p> <p>Females: varies by menstrual phase</p> <p>Males: 1.1 – 8.8 U/L</p> <p><b>Testosterone</b></p> <p>Females: &lt;1.8 nmol/L</p> <p>Males: 8.4 – 28.8 nmol/L</p> <p><b>Estradiol (E2)</b></p> <p>Females: varies by menstrual phase</p> <p>Males: &lt;157 pmol/L</p>	<p>prostate cancer.</p> <ul style="list-style-type: none"> <li>- Immunotherapy-induced endocrine gland damage and its impact on associated feedback mechanisms can increase or decrease hormone levels and may require hormone supplementation.</li> </ul> <p>FSH and LH are produced by the pituitary gland. In women, FSH is associated with ovulation and egg development. FSH, LH and estradiol are often ordered together to assess a woman’s menopausal status. In men, LH is associated with testosterone and sperm development.</p> <p>Testosterone is an androgen produced by leydig cells in men, adrenal glands in both men and women, and in small amounts by the ovaries in women. Testosterone production is stimulated and controlled by LH.</p> <p>Estradiol is an estrogen produced by the ovaries in pre-menopausal women, testicles in men, and converted from estrone (E1) to estradiol (E2) in post-menopausal women. It is a good marker of ovarian function, and it plays a role in bone metabolism and growth.</p>
Other Tests	
<p><b>Lipase</b></p> <p>&lt;75 U/L</p> <p><b>Amylase</b></p> <p>0 – 100 U/L</p>	<p>Lipase is an enzyme produced by the pancreas to digest dietary fats. Amylase is an enzyme produced by the pancreas to digest carbohydrates.</p> <ul style="list-style-type: none"> <li>- The levels of these enzymes can increase in the blood when the pancreas is injured (pancreatitis), or when the pancreatic duct is blocked by gall stones or pancreatic tumours.</li> <li>- Immunotherapy-induced pancreatic injury can also elevate serum lipase or amylase levels</li> </ul>
<p><b>Serum Glucose</b></p> <p>Fasting: 3.3 – 5.5 mmol/L</p> <p>Random: 3.3 – 11 mmol/L</p>	<p>Glucose is the body’s main energy source and is regulated by insulin and glucagon hormones produced in the pancreas.</p> <ul style="list-style-type: none"> <li>- Diabetic patients may require dose modifications of their medications and glucose levels should be closely monitored.</li> <li>- There is an increased risk of hyperglycemia in patients receiving corticosteroids or certain cancer medications such as immunotherapy.</li> </ul>
<p><b>C-Reactive Protein (CRP)</b></p> <p>&lt; 4.8 mg/L</p>	<p>C-reactive protein (CRP) is produced by the liver and released into the blood during injury, infection, or inflammation.</p> <ul style="list-style-type: none"> <li>- CRP is a sensitive and non-specific indicator of acute inflammation, which can be increased in patients with cancer.</li> </ul>

Normal Range**	Interpretation Tips
	<ul style="list-style-type: none"> <li>CRP may also be used as a prognostic indicator (in combination with other prognostic markers) in solid tumors, with elevated CRP suggestive of a more negative prognosis.</li> </ul>
Cardiac Tests	
<p><b>Left Ventricular Ejection Fraction (LVEF)</b></p> <p>≥ 50 % normal</p>	<p>LVEF is a measurement of the percentage of blood leaving the heart's main pumping chamber, the left ventricle, each time it contracts. It is used as an assessment of heart function.</p> <p>An echocardiogram (ECHO) or multigated acquisition (MUGA) scan can be used to measure LVEF.</p> <ul style="list-style-type: none"> <li>A reduced LVEF can be caused by various heart conditions, or by certain cardiotoxic cancer medications.</li> </ul>
<p><b>Electrocardiogram (ECG or EKG)</b></p> <p><b>QT interval (QT)</b></p> <p><b>Corrected QT interval (QTc)</b></p> <p>Females: QTc ≤ 470 milliseconds (ms)</p> <p>Males: QTc ≤ 450 milliseconds (ms)</p>	<p>An ECG graphically assesses heart rhythm by recording the electrical activity of the heart. The QT interval on the ECG represents the time it takes for the ventricles of the heart to depolarize (contract) and repolarize (relax).</p> <p>Heart rate affects the QT interval as it lengthens with bradycardia and shortens with tachycardia. Therefore, a corrected QT interval (QTc) accounting for heart rate is frequently calculated and used. Patients who experience a prolonged QTc interval are at risk for developing torsade de pointes (TdP), a ventricular tachycardia which can be life-threatening.</p> <ul style="list-style-type: none"> <li>Certain cancer medications, such as oxaliplatin, are directly associated with an increased risk of TdP.</li> <li>Other medications can increase risk via drug interactions or by contributing to bradycardia or electrolyte abnormalities.</li> </ul>

\*Antigen, tumour marker and diagnostic tests are not included in this guide.

\*\*NOTE: Reference ranges can vary depending on the assays, equipment, and techniques used. Use the reference range supplied by the laboratory that performed the test. Due to inter- and intra-patient variability, interpret laboratory findings with consideration of normal physiological fluctuations (e.g. assess trends rather than an isolated laboratory value) and the patient's clinical presentation.

Revision date: Jan 2020