Lipid Profile
Triglyceride Test

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Triglyceride

H\(\text{-C-OH}\) + HO\(-C\text{-R'}\) \rightarrow \ H\text{-C-O-C-R'}
glycerol \quad 3 \text{ fatty acids} \quad \text{triglyceride (triester of glycerol)}
Preparation

Fasting

• Blood should be collected after a 12-hour fasting (no food or drink, except water).
• TG remains high for several hours after meal
Preparation

- Do not Drink Tea and Coffee

  - The intake of coffee especially unfiltered coffee is contributed significantly to the increase in TC, LDL-C and TG. And the changes were related to the level of intake.

  Drinking coffee for 45 days was associated with an increase of 8.1 mg/dl for total cholesterol (TC), 5.4 mg/dl for low-density lipoprotein cholesterol (LDL-C) and 12.6 mg/dl for triglyceride (TG) (Cai et al., 2012).
Relation between Hyperglycemia and Hyperlipidemia

Glucose $\rightarrow$ F1,6 P $\rightarrow$ DHAP + G6P $\rightarrow$ pyruvate

Glycolysis

Glycerol synthesis

Triglyceride $\rightarrow$ Glycerol $\rightarrow$ Fatty Acid

Acetyl CoA
Reagents

R1: BUFFER
- Magnesium Chloride
- Preservative

R2: ENZYMES
- Lipase
- Peroxidase (POD)
- Glycerol 3 phosphate oxidase (GPO)
- Glycerol Kinase (GK)

R3: STANDARD
- Glycerol (200 mg/dL)
<table>
<thead>
<tr>
<th>Triglycerides</th>
<th>Mg/dl</th>
<th>mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reference Range</td>
<td>35-160</td>
<td>0.40 – 1.82</td>
</tr>
</tbody>
</table>
**Procedure**

<table>
<thead>
<tr>
<th></th>
<th>Blank</th>
<th>Standard</th>
<th>Assay</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reagent</td>
<td>1 mL</td>
<td>1 mL</td>
<td>1 mL</td>
</tr>
<tr>
<td>Demineralized Water</td>
<td>10µL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Standard</td>
<td></td>
<td>10µL</td>
<td></td>
</tr>
<tr>
<td>Specimen</td>
<td></td>
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<td>10µL</td>
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Mix. Le stand for 5 minutes at 37C or 10 minutes at room temperature. Record absorbance at 500 nm (480-520) against reagent blank.

*Reaction stable for 1 hour*
Fossati Principle method.

Reaction scheme is as follows:

- **Triglycerides**$\rightarrow$ lipase $\rightarrow$ **glycerol + Free Fatty Acids**

- **Glyceraldehyde 3-phosphate (GK)**
  - **Glycerol + ATP**$\rightarrow$**Glycerol 3-phosphate + ADP**$\leftarrow$ **Glycerol + ATP**

- **Glycerol 3-phosphate** + $\text{O}_2$ $\rightarrow$ **Dihydroxyacetone Phosphate** + H$_2$O$_2$

- **H$_2$O$_2$ + 4-Chlorophenol + PAP** $\rightarrow$ **Quinoneimine (Pink)** + H$_2$O
Result = \frac{\text{Abs (assay)}}{\text{Abs (standard)}} \times \text{standard concentration}
Indications

TG identify the risk of developing coronary heart disease (CHD). This test is part of a lipid profile that includes the measurement of cholesterol and lipoproteins. This test is also performed on patients with suspected fat metabolism disorders.
Interfering Factors

Ingestion of fatty meals may cause elevated TG levels.

Ingestion of alcohol may cause elevated levels of TG by increasing the production of VLDL.

Pregnancy may cause increased levels.

Drugs that may cause increased TG levels include cholestyramine, estrogens, and oral contraceptives.

Drugs that may cause decreased levels include ascorbic acid, asparaginase, clofibrate, colestipol, fibrates, and statins.
Increased Levels

**Hypothyroidism**: Catabolism of TG is diminished.

**High-carbohydrate diet**: Excess carbohydrates are converted into TG

**Poorly controlled diabetes**: Diabetics have an increased synthesis of TG-carrying VLDL and a decreased catabolism of the same.

**Chronic renal failure**: Patients have a deficiency in lipoprotein lipase that clears the blood of TG.
Decreased Levels

Malabsorption syndrome

Malnutrition: These patients have diminished fat in the diet. As TG is the major component of dietary fat, TG levels can be expected to fall.

Hyperthyroidism: The catabolism of VLDL, the main TG-carrying lipoprotein, is increased. Therefore, TG blood levels diminish.