C-Peptide of Insulin

**Analyte:** C-Peptide of Insulin

**Specimen Type:** Serum, EDTA Plasma, P-800 Plasma (or equivalent)

**Optimum Volume:** 0.5 mL

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Storage</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-8°C</td>
<td>2d ser; 6d pl</td>
<td>2-8°C</td>
</tr>
<tr>
<td>-20°C</td>
<td>6 months</td>
<td>-20°C</td>
</tr>
<tr>
<td>-70°C</td>
<td>1 year</td>
<td>-70°C</td>
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**Reporting units:** nd/mL

**Method:** Electrochemiluminescence

**Biological or Clinical Significance:**

C-peptide of insulin is a metabolic by-product produced during the activation of insulin. Insulin is a polypeptide hormone originating in the beta cells of the pancreas and serving as a principal regulator for the storage and production of carbohydrates. It is synthesized as part of a larger inactive protein, proinsulin. A rise in plasma glucose concentration induces an enzyme cleavage of proinsulin into the active hormone, insulin (MW 6000), and a fragment called C-peptide (MW 3000). Upon glucose stimulation, the beta cells release equimolar amounts of insulin and C-peptide, and small amounts of proinsulin and other intermediates (less than 5% of normal total insulin secretion). However, insulin is rapidly cleared both by liver uptake, tissue utilization and renal clearance (t1/2 ~4 min), and circulating insulin levels are very low during fasting. In contrast, C-peptide of insulin does not undergo significant liver or extra-renal metabolism and, therefore, has a much longer circulating half-life (about 35 min) than insulin; 5 to 10 times higher concentration of C-peptide persists in the peripheral circulation, and these levels fluctuate less than insulin. The liver does not extract C-peptide, which is removed from the circulation by the kidneys and degraded, with a fraction excreted unchanged in the urine. The concentration in urine is about 20-50 fold higher than in serum. C-peptide concentrations are therefore elevated in renal disease. This SOP covers only the assay of serum or plasma C-peptide.

In the past, C-peptide has been considered biologically inactive. However, recent studies have demonstrated that it is capable of eliciting molecular and physiological effects suggesting that C-peptide is itself a bioactive peptide. There is evidence that C-peptide replacement, together with insulin administration, may prevent the development or retard the progression of long-term complications in type 1 diabetes. Measurements of C-peptide, insulin, and glucose are used as an aid in the differential diagnosis of hypoglycemia (factitious hypoglycemia and hypoglycemia caused by hyperinsulinism) to ensure appropriate management and therapy of the patients. To quantify the endogenous insulin secretion, C-peptide is measured basally, after
fasting and after stimulation and suppression tests. Due to high prevalence of endogenous anti-insulin antibodies C-peptide concentrations reflect the endogenous pancreatic insulin secretion more reliably in insulin-treated diabetics than the levels of insulin itself. Measurements of C-peptide may therefore be an aid in the assessment of a residual β-cell function in the early stages of type 1 diabetes mellitus (T1D) and for the differential diagnosis of latent autoimmune diabetes of adults (LADA) and type 2 diabetes (T2D). C-peptide measurements are also used to assess the success of islet transplantation and for monitoring after pancreatectomy. Correlation was also found between higher C-peptide levels and increasing hyperlipoproteinaemia and hypertension. Decreased C-peptide levels are observed in: Starvation, factitious hypoglycemia, hypoinsulinism (T2D, T1D), Addison’s disease and after radical pancreatectomy.

**Principle of Test Method:**

The C-Peptide assay is a sandwich immunoassay on an automated platform employing chemiluminescent detection.

**References:**