Chapter 10 Opener part 1

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Figure 10.1

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Rate of $N$-carbamoylaspartate formation

[Diagram showing a curve with the y-axis labeled [CTP], mM]
Figure 10.3
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Cysteine

\[
\text{HO-} \text{Hg} - \text{COO}^-
\]

\[ p\text{-Hydroxymercuribenzoate} \]

\[
\text{HOH}
\]

Figure 10.4

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- **T state** (less active) favored by CTP binding
- **R state** (more active) favored by substrate binding
Figure 10.13
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The graph shows the rate of N-carbamoylaspartate formation as a function of [Aspartate], mM, with two curves: one without and one with +0.4 mM CTP.
Figure 10.14

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Figure 10.16a

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<table>
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<tr>
<th></th>
<th>Heart</th>
<th>Kidney</th>
<th>Red blood cell</th>
<th>Brain</th>
<th>Leukocyte</th>
<th>Muscle</th>
<th>Liver</th>
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<td>Modification</td>
<td>Donor molecule</td>
<td>Example of modified protein</td>
<td>Protein function</td>
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<td>Phosphorylation</td>
<td>ATP</td>
<td>Glycogen phosphorylase</td>
<td>Glucose homeostasis; energy transduction</td>
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<td>Acetylation</td>
<td>Acetyl CoA</td>
<td>Histones</td>
<td>DNA packing; transcription</td>
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<td>Src</td>
<td>Signal transduction</td>
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<td>ADP ribosylation</td>
<td>NAD⁺</td>
<td>RNA polymerase</td>
<td>Transcription</td>
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<td>Farnesylation</td>
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<td>Ras</td>
<td>Signal transduction</td>
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<td>γ-Carboxylation</td>
<td>HCO₃⁻</td>
<td>Thrombin</td>
<td>Blood clotting</td>
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<td>Sulfation</td>
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<td>Fibrinogen</td>
<td>Blood-clot formation</td>
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<td>Ubiquitination</td>
<td>Ubiquitin</td>
<td>Cyclin</td>
<td>Control of cell cycle</td>
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Acetylated lysine
Serine, threonine, or tyrosine residue

Protein kinase

ATP

Phosphorylated protein

ADP + H^+
<table>
<thead>
<tr>
<th>Signal</th>
<th>Enzyme</th>
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<tr>
<td>Cyclic nucleotides</td>
<td>Cyclic AMP-dependent protein kinase</td>
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<td>Cyclic GMP-dependent protein kinase</td>
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<tr>
<td>Ca(^{2+}) and calmodulin</td>
<td>Ca(^{2+})–calmodulin protein kinase</td>
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<tr>
<td></td>
<td>Phosphorylase kinase or glycogen synthase kinase 2</td>
</tr>
<tr>
<td>AMP</td>
<td>AMP-activated kinase</td>
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<tr>
<td>Diacylglycerol</td>
<td>Protein kinase C</td>
</tr>
<tr>
<td>Metabolic intermediates and other “local” effectors</td>
<td>Many target-specific enzymes, such as pyruvate dehydrogenase kinase and branched-chain ketoacid dehydrogenase kinase</td>
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</tbody>
</table>

Source: After D. Fell, *Understanding the Control of Metabolism* (Portland Press, 1997), Table 7.2.
Phosphorylated protein + H$_2$O $\rightleftharpoons$ Protein phosphatase $\rightarrow$ Orthophosphate (P$_i$)
Protein–OH + ATP → Protein–OPO₃²⁻ + ADP

1 → H₂O

Protein–OH + HOPO₃²⁻
Cyclic adenosine monophosphate (cAMP)
Figure 10.17

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<table>
<thead>
<tr>
<th>Site of synthesis</th>
<th>Zymogen</th>
<th>Active enzyme</th>
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<td>Stomach</td>
<td>Pepsinogen</td>
<td>Pepsin</td>
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<td>Pancreas</td>
<td>Chymotrypsinogen</td>
<td>Chymotrypsin</td>
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<tr>
<td>Pancreas</td>
<td>Trypsinogen</td>
<td>Trypsin</td>
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<tr>
<td>Pancreas</td>
<td>Procarboxypeptidase</td>
<td>Carboxypeptidase</td>
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</table>
Figure 10.20

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Chymotrypsinogen (inactive)

1

$\pi$-Chymotrypsin (active)

1 15

16

245

Trypsin

$\pi$-Chymotrypsin

$\alpha$-Chymotrypsin (active)

1 13

16

146

149 245

A chain

B chain

C chain

Two dipeptides

Figure 10.21

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Glutamine + Lysine $\xrightarrow{\text{Transglutaminase}}$ Cross-link
Figure 10.30

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γ-Carboxyglutamate residue
Antihemophilic factor (VIII)

Proteolysis
| Fibrin binding | Kringle | Kringle |          | Serine protease |

**Figure 10.35**
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Figure 10.36

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