CheY, the response regulator of bacterial chemotaxis, plays a pivotal role in signal transduction in bacterial chemotaxis and interacts with at least three proteins: CheA, FliM, and CheZ. CheA receives signals from chemoreceptors and then transfers the signal to CheY by a phosphotransfer reaction. Phosphorylated CheY binds to FliM, one of the switch proteins, resulting in a change in flagellar rotation from counterclockwise to clockwise. Phosphorylated CheY is dephosphorylated by its intrinsic autophosphatase activity and by CheZ. The CheA- and FliM-binding surfaces of CheY have been well studied, but characterization of the CheZ-binding surface of CheY is incomplete. We have analyzed the effect of CheZ on the dephosphorylation rates of 14 mutants of CheY. Nine mutant CheY proteins showed more resistance to CheZ phosphatase activity than did wild-type CheY. These nine mutant CheY proteins could be divided into two groups: one with altered CheZ binding and the other with normal CheZ binding. The mutations causing reduced CheZ binding altered residues on the same surface of CheY, a region consisting of the β2-α5 loop, the α4-helix, and part of the α5-helix. Mutations rendering CheY resistant to CheZ, isolated by Sanna et al. (Sanna, M. G., Swanson, R. V., Bourret, R. B., and Simon, M. I. (1995) Mol. Microbiol. 15, 1069–1079), were also found to affect this surface. The mutations in the CheY protein that affect CheZ activity but not CheZ binding are located in the β2-α5 loop, which appears to be involved in the catalytic activity of CheZ. Finally, our results indicate that the CheY surfaces that bind CheA, FliM, and CheZ overlap, but are not completely identical.

The concentration of CheY-P determines whether cells tumble or swim smoothly. The level of CheY-P in vivo is controlled by the autophosphorylation and phosphotransfer activities of the CheA kinase (15, 16) and by the dephosphorylation rate of CheY-P. The dephosphorylation rate is controlled by the intrinsic autophosphatase activity of CheY (15, 16) and by CheZ activity (15, 17). Although CheY-P is capable of autodephosphorylation, this reaction is relatively slow compared with the response time of the chemotaxis system (18). The dephosphorylation reaction is markedly accelerated by CheZ (15, 17, 19). The importance of CheZ activity is indicated by the extremely tumbly motility and loss of normal chemotactic ability of cheZ null mutants (20–23).

The interaction between CheZ and CheY has been investigated by several research groups (19, 24–30). The binding of CheZ to CheY-P is greater than its binding to apo-CheY (24, 30). The carboxyl-terminal domain in CheZ has been identified as the CheY-binding domain (27). The CheZ-binding region of CheY has not been completely elucidated, although two mutant CheY proteins with reduced ability to bind CheZ have been reported (29, 30). In this study, we characterize a number of mutant CheY proteins as substrates for CheZ. Nine mutant CheY proteins were more resistant to dephosphorylation by CheZ than was wild-type CheY. These nine mutant CheY proteins fell into two categories based on their affinity for CheZ: 1) mutant CheY proteins with reduced sensitivity but normal binding activity and 2) mutant CheY proteins with both reduced sensitivity and altered activity for CheZ.
CheY-CheZ Interaction

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TABLE I

Bacterial strains and plasmids

| Strain/plasmid | Relevant genotype and description | Source/Ref. |
|---------------|-------------------------------|------------|
| RP437         | Wild type (Che+)               | J. S. Parkinson |
| RP4079        | cheY216 recA                   | J. S. Parkinson |
| RP5135        | Δstar-CheZ                     | J. S. Parkinson |
| SG1           | trpR(amy)suDPts cheY::kan'     | Lab collection |
| XY77          | recD1903, cheY7771             | Lab collection |
| XY79          | recD1903, cheY7771/1Y106W      | Lab collection |

Plasmids

| pT7–7M       | penR, FliM expression          | Lab collection |
| pRL22Z       | CheY, CheZ expression          | Lab collection |
| pRL22AZ      | CheZ deletion, wild-type       | Lab collection |
| pRL22AZ.CheY |                  | Lab collection |
| pYM3         | cheY93K in pRL22AZ            | Lab collection |
| pYM10        | cheYA88T in pRL22AZ           | This study |
| pXY220       | cheY7871 in pRL22AZ           | Lab collection |
| pXY220.Z     | cheY106W in pRL22AZ           | Lab collection |
| pXY201       | cheY7871/1Y106W in pRL22AZ    | Lab collection |
| pXY201.Z     | cheY11M7 in pRL22AZ           | Lab collection |
| pXY202       | cheY686M in pRL22AZ           | This study |
| pRB404.Z876  | cheY8765 in pRBB404.Z876      | J. Appleby and R. B. Bourret |

RESULTS

In Vitro Phosphorylation and Dephosphorylation of Mutant CheY Proteins—The phosphorylation/dephosphorylation of CheY consists of a phosphotransfer from phospho-CheA and either the intrinsic autophosphatase activity of CheY or CheZ-enhanced dephosphorylation. To understand the effect of CheZ on the CheY dephosphorylation reaction, we screened a number of mutant CheY proteins for their ability to be dephosphorylated by CheZ. The mutant CheY proteins in this study were selected by one or more of four criteria. 1) They contain residue substitutions close to the phosphorylation site of Asp57 (V86M, T87I, T87S, T87I/Y106W, and A87T). 2) They had previously been shown to have altered CheA binding (D93K, A90V, Y106W, V108M, F111V, and T112I) (35). 3) They were previously described CheZ-binding mutants (29, 30). 3) They had previously been shown to have altered CheA binding (D93K, A90V, Y106W, V108M, F111V, and T112I) (35). 4) They had been identified as suppressors of mutations affecting the flagellar switch (A90V, V108M, F111V, T112I, E117K, and E27K) (9). To test the dephosphorylation rates of mutant CheY proteins, we first needed to know whether the mutant CheY proteins could be phosphorylated by CheA. Fourteen mutant CheY proteins were characterized to be phosphorylated by CheA in vitro. Thirteen mutant CheY proteins had CheY-P levels similar to that of wild-type CheY under our conditions. The A87T mutant CheY protein had 40% phosphorylation activity relative to wild-type CheY (data not shown).

The dephosphorylation rates of these mutant CheY proteins in the presence of CheZ were assayed using purified CheY-P (36, 40). Different amounts of CheZ were added, and the dephosphorylation rates were measured. Examples of the results obtained are presented in Fig. 1, and Table II summarizes the results for all of the CheY proteins assayed. Nine of the phosphorylated mutant CheY proteins were 5- to >1000-fold more resistant to CheZ activity than was wild-type CheY-P. The others had sensitivity to CheZ similar to that of wild-type CheY. The autodephosphorylation rates of the nine mutant CheY proteins exhibiting decreased rates were also assayed using purified CheY-P in the absence of CheZ. Two mutant CheY proteins (T87I and T87S/Y106W) had 5-fold lower autodephosphorylation rates than did wild-type CheY. The other seven mutant CheY proteins had normal dephosphorylation.

CheY-CheZ Binding—One explanation for the phenotypes of these CheY mutations is that they disrupt CheY-CheZ interaction. To explore this possibility, the binding activity of wild-type and mutant CheY proteins with CheZ was measured. CheZ binding to the phosphorylated form of CheY with higher affinity than it does to the dephosphorylated form of CheY (24, 26, 30). Therefore, binding of apo-CheY and CheY-P to CheZ proteins was measured. Most mutant CheY proteins were phosphorylated to the same extent as wild-type CheY (data not shown); however, proteins with the T87I and T87S/Y106W substitutions were not phosphorylated by acetyl

Previously (38, 39), briefly, the autophosphorylating CheA kinase was coupled to Sepharose beads and phosphorylated with [γ-32P]ATP. CheY was then added to the CheA beads to allow the phosphoryl group transfer reaction to occur. The phosphorylated CheA reaction mixture contained 3 μl of 10-fold phosphoryl buffer, 3 μl of [γ-32P]ATP, and 0.5 μl of unlabeled ATP (15 mM) in a total volume of 30 μl, with -1 μg of CheA/μl of bead. The reaction was carried out at room temperature by rotating the beads for 30 min and was stopped by washing the beads with excess phosphoryl buffer. The phosphorylated CheA beads were stored on ice. 100 μl of purified CheY (1 μmol in 50 mM Tris, pH 7.5) was added to the beads attached to phosphorylated CheA, and phosphate transfer was allowed to progress for 30 s while rotating at 10-12 °C. Phosphorylated CheY was removed with a Hamilton syringe and immediately transferred at 10-12 °C into a buffer containing 0.2 mM MgCl₂, 50 mM Tris, pH 7.5, and different amounts of CheZ. Samples of phosphorylated CheY were removed at various times, and the autodephosphorylation reaction or the CheZ-enhanced dephosphorylation reaction was quenched with 2-XTDS-PAGE sample buffer. Samples were loaded directly onto gels for SDS-PAGE (15% polyacrylamide). Radiolabeled proteins were visualized by autoradiography, and the radioactivity of the protein bands was determined with an AMBIS β-scanning system.

CheY Phosphorylation by Acetyl Phosphate—Acetyl [32P]phosphate was synthesized according to Welch et al. (10). CheY phosphorylation by acetyl [32P]phosphate was performed as previously described (38). Identical amounts of wild-type or mutant CheY proteins were mixed with 20 mM acetyl [32P]phosphate in a buffer containing 5 mM MgCl₂, 2 mM dithiothreitol, and 50 mM Tris-HCl, pH 7.9, in a total volume of 20 μl. The reactions were allowed to stand at room temperature (22 °C) for various times (10-10 min) before being quenched by the addition of 2× SDS-PAGE loading buffer. 10 μl of reaction products was analyzed on 15% SDS-polyacrylamide gels. Labeled proteins were visualized by autoradiography.

CheY-CheZ Binding Assay—The assays for binding of CheY to CheZ were carried out as described previously (26), with some modifications. CheY beads were suspended to homogeneity in 50 mM Tris-HCl, pH 7.9, and dispensed in aliquots of 50 μl in microcentrifuge tubes (-3 nmol of CheY in each aliquot). 100 μl of reaction buffer (50 mM Tris and 5 mM MgCl₂) and 35 μl of stabilizer buffer (3.4% glycerol and 12.5 mM MgCl₂) were added. Acetyl phosphate was added from a 1 M stock solution to 20 mM, as needed, and the phosphorylation reactions proceeded for 2 min at room temperature. Then, 3 nmol of purified CheZ was added to each tube. The final volume of each reaction mixture was adjusted to 200 μl with 50 mM Tris-HCl. The reactions were incubated at room temperature for 10 min. The beads were washed twice with 1 ml of 50 mM cold Tris buffer containing 5 mM MgCl₂ with acetyl phosphate added to the appropriate wash buffer. 60 μl of 2× SDS-PAGE loading buffer was added, and the suspension was mixed at room temperature for 5 min. The beads were boiled for 2 min to remove the bound CheZ from the CheY beads. 10 μl of supernatant was loaded onto gels for 15% SDS-PAGE. Gels were stained with Coomassie Brilliant Blue. CheZ bands were scanned by Sigma Gel Gel Analysis software. Bovine serum albumin beads served as controls.
phosphate, and the A88T protein could be phosphorylated to only 40% of the wild type.

As shown in Fig. 2, among seven CheZ-resistant mutant CheY proteins assayed for CheZ binding, four (E27K, F111V, T112I, and E117K) showed at least 50% reduction in CheZ binding, whereas three other mutant CheY proteins (A88T, A90V, and E93K) had a similar affinity for CheZ compared with wild-type CheY. The combined results from the dephosphorylation and CheY-CheZ binding assays indicate that there are two classes of CheY mutants that affect CheZ activity. One class is resistant to CheZ activity, but does not affect CheZ binding. The other decreases CheZ activity by reducing CheZ binding. We were not able to test CheZ binding with the phosphorylated T87I and T87I/Y106W proteins since they could not be phosphorylated by acetyl phosphate.

**Mapping Residue Changes Causing CheZ Resistance onto the CheY Structure**—The black residues in Fig. 3 depict the positions on CheY where CheY residue substitutions reduce CheZ binding. Sites at which substitutions conferring CheZ resistance allow wild-type levels of CheZ binding are shown in gray. These latter residues are located near the active site of CheY, which is identified by the label for Asp57 (D57). The residues

![% of CheY-P](image)

**TABLE II**

| Mutant | Half-life$^a$ of CheY-P normalized to WT$^b$ | CheZ required to reduce half-life$^a$ of CheY-P by one-half |
|--------|---------------------------------------------|----------------------------------------------------------|
| WT CheY | 1                                           | 1                                                        |
| M17W   | 2                                           | 2                                                        |
| E27K   | 1                                           | 50                                                       |
| V86M   | 1.5–2                                       | 5                                                        |
| T87S   | 1.5–2                                       | 0.5–1                                                    |
| T87I   | 5                                           | >1000                                                    |
| T87I/Y106W | 5                                           | >1000                                                    |
| A88T   | 1                                           | >50                                                     |
| A90V   | 1                                           | 5                                                        |
| E93K   | 1                                           | 5                                                        |
| Y106W  | 2                                           | 1–2                                                     |
| V108M  | 1                                           | 2                                                        |
| F111V  | 1                                           | 100                                                      |
| T112I  | 1                                           | >50                                                     |
| E117K  | 1                                           | 50                                                       |

$^a$ The half-lives of CheY-P calculated as ln 2/slope.

$^b$ WT, wild type.

![Relative CheZ binding](image)
were analyzed in vivo using motility plates, the cell tethering assay, and direct microscopic observation of the liquid bacterial culture. Mutant cheY alleles, except those encoding the A88T and E93K proteins, were either subcloned into a low-copy-number plasmid (9) or introduced into the chromosome DNA (40) to ensure single-copy cheY expression. As shown in Table III, all these mutants, except A90V, were incapable of swarming on motility plates.

The A90V mutant could swarm somewhat. All of the cheY mutant strains exhibited smooth-swimming behavior when observed under the microscope, and all showed counterclockwise-biased flagellar rotation in the cell tethering assay. These results were surprising because one would expect that the CheZ-resistant cheY mutants will cause the accumulation of CheY-P in vivo, resulting in a tumbling phenotype. Under “Discussion,” we review the data for the interactions of CheY with CheA (35) and of CheY with FliM (9) that enable us to speculate why these mutant cheY proteins confer a smooth-swimming phenotype rather than a tumbling phenotype like the CheZ-resistant N23D and E26K CheY mutations described by Sanna et al. (29).

**DISCUSSION**

**CheZ Binding, CheZ Catalysis, and the Mechanism of CheY Dephosphorylation**—Nine CheZ-resistant cheY mutants were obtained in this study (Table II). Seven of these mutant cheY proteins were tested for their CheZ binding ability. They showed two different CheZ binding properties. Four mutant cheY proteins had reduced binding of CheZ (Fig. 2). The residues, altered in these proteins, cluster in the three-dimensional structure of CheY on the same face of CheY as the N23D and K26E CheY mutations that alter CheZ binding (29, 30). Fig. 3 indicates the positions of CheY altered residues that reduce CheZ binding. This surface consists of the α1-helix (N23D, K26E, and E27K), part of the α5-helix (E117K), and the β2–α2 loop (F111V and T112I). These residues are located on the surface of CheY. The gray residues are in the β1–α1 loop (A88T and A90V) and at the top of the α2-helix (E93K), suggesting that this region is involved in CheZ catalytic activity.

**A Structural Shift in the β2-α2 (90’s) Loop Affects CheY Autophosphatase Activity and Sensitivity to CheZ**—Of these 14 mutant CheY proteins, four structures have been solved: T87I (39), T87I/Y106W (40), Y106W (40), and T87S. The overall structure of all four mutant CheY proteins is the same as that of wild-type CheY. Both the T87I (39) and T87I/Y106W (40) proteins showed distinct backbone conformational changes in the 90’s loop (41). This shift was directly attributable to the substitution of isoleucine for threonine at position 87 since no such backbone changes were found in the Y106W (40) or T87S2 protein (Fig. 4). The 90’s loop consists of residues 88–92 and is near (7–14 Å) the Asp57 phosphorylation site of CheY. The T87I and T87I/Y106W mutant proteins were completely resistant to CheZ activity and had five times lower autodephosphorylation rates than wild-type CheY (Fig. 1 and Table II). Furthermore, they were not able to be phosphorylated by acetyl phosphate, although they can be phosphorylated by CheA. On the other hand, the Y106W and T87S proteins, which lack the shift in the 90’s loop, are not resistant to CheZ and have only slightly altered autodephosphorylation rates compared with wild-type CheY. Both of these proteins could be phosphorylated by either CheA or acetyl phosphate. These results indicate that the shift in the 90’s loop of CheY is highly correlated with changes in CheY autodephosphorylation and enhanced dephosphorylation by CheZ.

**Behavioral Characterization of CheZ-resistant Mutants**—The effects of these CheZ-resistant mutants on chemotaxis

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2 X. Zhu, P. Matsumura, and K. Volz, unpublished data.
our CheY-CheZ binding assay, in which CheZ was coupled to Sepharose beads, CheY did not exhibit increased binding upon phosphorylation (data not shown). This result could be viewed as further evidence that CheZ must be oligomerized to increase its affinity for CheY-P since the beads could prevent CheZ from oligomerizing. CheZ activity on CheY-P might also be regulated as further evidence that CheZ must be oligomerized to increase phosphorylation (data not shown). This result could be viewed as showing that an area of CheY including the $\alpha_1$-$\alpha_2$ loop and part of the $\alpha_1$-helix might be involved in the interaction with the CheZ-binding surface of CheY (Table III). The data reported here suggest that the CheZ-binding surface of CheY consists of the $\beta_5$-$\alpha_5$ loop, the top of the $\alpha_5$-helix, and part of the $\alpha_1$-helix (Fig. 3 and Table III). Therefore, the three proteins bind to surfaces of CheY that overlap but are not completely identical.

All the residues shown by genetic studies to be involved in interactions with CheA, CheZ, and FliM were mapped on the CheY surface (Fig. 5). The red residues (E93K and Y106W) represent residues that are specifically required for CheA binding. The pink residues (A90V and V108M) are involved in both CheA and CheZ binding. The white residues (T112I and E117K) indicate positions at which substitutions affect binding of all three proteins. The green residue (E27K) is involved in both FliM and CheZ binding. The blue residues (N23D and K26E) represent positions at which substitutions reduce CheZ binding (29, 30) but presumably allow normal binding of CheA and FliM since they must bind both CheA and the motor to generate a tumbling phenotype (29, 30). As shown in Fig. 5, the CheA-binding surface extends from the $\alpha_1$-$\alpha_2$-helix of CheY; the CheZ-binding surface is mainly located on the $\alpha_5$-helix; and the FliM surface overlaps both the CheA- and CheZ-binding surfaces. The Venn diagram depicts this pattern.

CheA binds to apo-CheY and dissociates from CheY-P when it is phosphorylated (43). In contrast, CheZ and FliM bind to CheY-P with a higher affinity than to apo-CheY (10, 11, 24, 30). It is possible that the overlap region in the unphosphorylated state contributes to the CheA-binding surface (35) and that CheY phosphorylation alters the topology of this region, causing the release of CheY from CheA and increasing the affinity for CheZ binding.

**TABLE III**

| CheY mutant | Swarm rotation | CheA bindinga | FliM bindingb | CheZ binding |
|-------------|----------------|---------------|---------------|-------------|
| Wild type   | CCW-CW         | Normal        | Normal        | Normal      |
| In $\alpha_1$-helix | E93K           | CCW-CW       | Strong        | ND          |
| $\beta_5$-$\alpha_5$ loop | T87I           | CCW-CW       | ND            | Normal      |
| A90V        | CCW-CW         | Weak          | Weak          | Normal      |
| In $\beta_5$-sheet | V108M         | CCW-CW       | Weak          | Normal      |
| $\beta_5$-$\alpha_5$ loop | F111V         | CCW-CW       | Weak          | Weak        |
| T112I       | CCW-CW         | Weak          | Weak          | Weak        |
| In $\alpha_5$-helix | E117K         | CCW-CW       | Weak          | Weak        |
| $\beta_2$-N5 | E27K           | CCW-CW       | Normal        | Weak        |
| K26E $^c$   | CCW-CW         | ND            | ND            | Weak        |
| N23D $^d$   | CCW-CW         | ND            | ND            | Weak        |

$^a$ CheY-CheA binding data are from Shukla, et al. (D. Shukla, X. Zhu, and P. Matsumura, in revision).

$^b$ X. Zhu and P. Matsumura, unpublished data.

$^c$ CCW, counterclockwise; CW, clockwise; ND, not determined.

$^d$ Data for these two mutants are from Sanna et al. (29, 30).

containing $\alpha_4$-$\beta_5$-$\alpha_6$ is involved in CheY-CheA recognition. Similar results were reported in a two-dimensional NMR study of CheY-CheA interaction (44). Studies of cheY suppressors of flagellar switch mutants indicated that the CheY mutants V111M, E27K, A90V, V108M, F111V, T112I, and E117K are fliG suppressors (9), whereas E27K, A90V, V108M, F111V, T112I, and E117K are fliM suppressors (35, 42, 43) (Table III). These data suggest that an area of CheY including the $\beta_5$-$\alpha_5$ loop and part of the $\alpha_5$-helix might be involved in the interaction with the CheZ-binding surface (Table III). The data reported here suggest that the CheZ-binding surface of CheY consists of the $\beta_5$-$\alpha_5$ loop, the top of the $\alpha_5$-helix, and part of the $\alpha_1$-helix (Fig. 3 and Table III). Therefore, the three proteins bind to surfaces of CheY that overlap but are not completely identical.

All the residues shown by genetic studies to be involved in interactions with CheA, CheZ, and FliM were mapped on the CheY surface (Fig. 5). The red residues (E93K and Y106W) represent residues that are specifically required for CheA binding. The pink residues (A90V and V108M) are involved in both CheA and CheZ binding. The white residues (T112I and E117K) indicate positions at which substitutions affect binding of all three proteins. The green residue (E27K) is involved in both FliM and CheZ binding. The blue residues (N23D and K26E) represent positions at which substitutions reduce CheZ binding (29, 30) but presumably allow normal binding of CheA and FliM since they must bind both CheA and the motor to generate a tumbling phenotype (29, 30). As shown in Fig. 5, the CheA-binding surface extends from the $\alpha_1$-$\alpha_2$-helix of CheY; the CheZ-binding surface is mainly located on the $\alpha_5$-helix; and the FliM surface overlaps both the CheA- and CheZ-binding surfaces. The Venn diagram depicts this pattern.
CheY's own autodephosphorylation reaction, but also that the activity of CheZ on CheY might be through enhancement of phosphorylated by CheA. These results suggest not only that CheY is phosphorylated by CheA through a phosphotransfer reaction. It has been suggested that this reaction is catalyzed by CheY rather than CheA (45, 46). The claim that CheY possesses kinase activity is supported by the observation that small molecule phosphodiesters such as acetyl phosphate can act in place of FliP to donate the phosphoryl group to CheY (47, 48). The T87I and T87I/Y106W mutant CheY proteins, which have a structural shift in their 90’s loop, cause severe defects in autodephosphorylation and then are completely resistant to CheZ activity (Table II). Furthermore, they cannot be phosphorylated by acetyl phosphate, although they can be phosphorylated by CheA. These results suggest not only that the activity of CheZ on CheY might be through enhancement of CheY's own autodephosphorylation reaction, but also that the mechanism of CheY phosphorylation by acetyl phosphate may involve the reverse dephosphorylation reaction rather than the forward phosphorylation by CheA. Alternatively, the mutant CheY proteins may alter binding to acetyl phosphate.

The Smooth-swimming Phenotype of the CheZ-resistant CheY Mutants—CheY is phosphorylated by CheA, and CheY-P binds to the motor switch, resulting in tumbles. A CheZ-resistant mutant CheY protein dephosphorylates more slowly, resulting in an elevated level of CheY-P. An increased level of CheY-P would affect signal transduction. An example is a mutation at Asp87, which cannot be phosphorylated by CheA and results in a smooth-swimming phenotype (49). One of our nine CheZ-resistant mutants (A87T) could be phosphorylated to only 40% of wild-type CheY levels. Any CheY mutation with a defect in binding to the switch will also block signal transfer. Five of our nine CheZ-resistant mutant CheY proteins (E27K, A90V, F111V, T112I, and E117K) show reduced binding to FliM. Furthermore, CheY phosphorylation and binding of CheY-P to the switch are necessary (but not sufficient) events in generating the tumble signal since a mutant CheY protein (Y106L) exhibits normal phosphorylation and dephosphorylation properties and normal binding to FliM, yet it fails to generate a tumble signal (38). It is possible that some of these CheZ-resistant mutant CheY proteins affect signal transduction at a step after CheY phosphorylation, like the Y106L mutant. The CheY mutants T87I and T87I/Y106W restrict the rotation of residue 106 (40), a limitation that may block signal propagation from CheY-P to the switch.

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