Subunit Organization in a Soluble Complex of Tar, CheW, and CheA by Electron Microscopy*

The Salmonella and Escherichia coli aspartate receptor, Tar, is representative of a large class of membrane receptors that generate chemotaxis responses by regulating the activity of an associated histidine protein kinase, CheA. Tar is composed of an NH2-terminal periplasmic ligand-binding domain linked through a transmembrane sequence to a COOH-terminal coiled-coil signaling domain in the cytoplasm. The isolated cytoplasmic domain of Tar fused to a leucine zipper sequence forms a soluble complex with CheA and the Src homology 3-like kinase activator, CheW. Activity of the CheA kinase in the soluble complex is essentially the same as in fully active complexes with the intact receptor in the membrane. The soluble complex is composed of ~28 receptor cytoplasmic domain chains, 6 CheW chains, and 4 CheA chains. It has a molecular weight of 1,400,000 (Liu, L., Levit, M., Lurz, R., Surette, M.G., and Stock, J.B. (1997) EMBO J. 16, 7231–7240). Electron microscopy reveals an elongated barrel-like structure with a largely hollow center. Immunoelectron microscopy has provided a general picture of the subunit and domain organization of the complex. CheA and CheW appear to be in the middle of the complex with the leucine zippers of the receptor construct at the ends. These findings show that the receptor signaling complex forms higher ordered structures with defined geometric architectures. Coupled with atomic models of the subunits, our results provide insights into the functional architecture by which the receptor regulates CheA kinase activity during bacterial chemotaxis.

Motile prokaryotes sense environmental signals via receptors such as those responsible for chemotaxis to serine (Tsr) or aspartate (Tar) in Salmonella and Escherichia coli (for recent reviews on bacterial chemotaxis, see Refs. 1–5). Tar and Tar are transmembrane proteins with periplasmic serine- or aspartate-binding domains connected by transmembrane sequences to coiled-coil structures in the cytoplasm that serve as docking sites for a histidine protein kinase, CheA, and an Src homology 3-like kinase activator, CheW. Complexes formed between these transmembrane receptors, CheW, and CheA exhibit a several hundredfold enhancement of CheA activity (6). The cytoplasmic chemoreceptor domain that binds CheA and CheW is a highly conserved antiparallel coiled coil (7), which binds to CheW with a 1:1 stoichiometry (8, 9). Genetically engineered receptor constructs have been used to express soluble fragments of the cytoplasmic domain that bind CheW and CheA to form soluble complexes with the same high kinase activities obtained with complexes formed between CheA, CheW, and intact receptors in membranes (9–11). One soluble receptor construct that worked particularly well in activating CheA was generated by fusing a leucine zipper dimerization domain to the NH2 terminus of the cytoplasmic coiled-coil domain of Tar (termed lzTarC). Mixtures of lzTarC with CheW and CheA resulted in the efficient production of large, stable, roughly uniform structures with a composition of ~28 lzTarC, 6 CheW, and 4 CheA chains (12).

Here we report an investigation of the molecular architecture of the highly active assemblies formed between lzTarC, CheA, and CheW. Results from electron microscopy, image processing, and immunogold labeling indicate a bipolar, barrel-like structure of staves formed from the coiled-coils of lzTarC. The leucine zipper portions of lzTarC extend from the ends of the barrel and CheA and CheW appear to be bound to the surface of the lzTarC barrel near the center of the longitudinal axis. The dimensions of the complex together with the known structures of its subunits have allowed the formulation of a low-resolution model of the complex. The results indicate that a higher order assembly involving multimeric receptor interactions may be required for receptor-mediated kinase CheA regulation during bacterial chemotaxis.

EXPERIMENTAL PROCEDURES

Proteins—CheA (13), CheY (14), CheW (15), and lzTarC (constructed using tar gene from Salmonella Q-LZ-MH1-S.M.-MH2 in (9)) were purified as described previously. Ternary complexes of lzTarC-CheW-CheA were formed, purified, and characterized according to Liu et al. (12) except that high performance liquid chromatography gel filtration of the complex on a TSK-Gel G5000PWXL column was performed using an elution buffer lacking glycerol (25 mM Tris-HCl, 50 mM potassium glutamate, 25 mM NaCl, 5% Me2SO, pH 7.5) because of the interference of glycerol with the electron microscopy experiments. After removal of glycerol, the activity of the complex (measured as described in Ref. 12) decreased exponentially with a t1/2 = 60 min; therefore aliquots of the complex peak fraction were immediately placed on microscope grids or incubated with antibodies for immunoelectron microscopy.

Affinity-purified rabbit polyclonal antibodies raised against a recombinant protein corresponding to amino acids 199–345 mapping in the carboxyl terminal domain of the transcription factor C/EBP-β were purchased from Santa Cruz Biotechnology. Western blot analysis showed that these antibodies bind to lzTarC, possessing the leucine zipper sequence from C/EBP-β, but do not bind to the corresponding protein, Tar054, lacking the leucine zipper sequence (9). Antibodies against CheA, CheW, CheY, and CheB were polyclonal; antibodies

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Organization of Chemoreceptor-Kinase Complex

**TABLE I**

| Type of complex          | Specific activity$^a$ | Activation factor$^b$ |
|--------------------------|-----------------------|-----------------------|
| lzTar-CheW-CheA          | 40                    | 100                   |
| Tsr-CheW-CheA            | 38                    | 95                    |
| Tsr-CheW-CheA$^c$        | 42                    | 105                   |
| Tsr-CheW-CheA$^c$        | 42                    | 105                   |

$^a$ Specific steady-state activity (turnover number) of the receptor-bound CheA dimers was measured using the spectroscopic assay as described previously (28) in a buffer containing 25 mM Tris-HCl, 50 mM potassium glutamate, 25 mM NaCl, 5% Me$_2$SO, pH 7.5 (12). Specific activities of CheA ($s^{-1}$) were calculated as a ratio of the rates of ATP hydrolysis ($\mu$M s$^{-1}$) and concentration of the receptor-bound CheA dimers ($\mu$M).

$^b$ Compared with the activity of CheA dimers alone under these conditions, 0.40 s$^{-1}$.

$^c$ Complexes were obtained as described previously (28) by incubation of 6.4 $\mu$M Tar in membranes with the following concentrations of CheW and CheA: 20 $\mu$M CheW and 1.0 $\mu$M CheA (Footnote c); 5.0 $\mu$M CheW and 10 $\mu$M CheA (Footnote c); 20 $\mu$M CheW and 10 $\mu$M CheA (Footnote c). In these experiments Tar membrane complexes were used instead of Tar. Under similar conditions, Tar and Tsr gave virtually the same CheA activation, but because levels of Tar expression in membranes were considerably higher than Tar (28), the Tsr complexes were much better defined in terms of subunit stoichiometry and kinase molecular activity.

Kinase Activity of lzTar$_c$-CheW-CheA Complexes—Complexes between lzTar$_c$ CheA, and CheW were produced and purified as described previously (12). The histidine kinase activity per mol of CheA in these complexes was the same as the kinase activity per mol of CheA in receptor signaling complexes assayed under similar conditions (Table I). This corresponds to 100-fold activation over the activity exhibited by the purified CheA protein in the absence of receptors or CheW.

Electron Microscopy of lzTar$_c$-CheW-CheA Complexes—Purified lzTar$_c$-CheW-CheA complexes were examined by electron microscopy. Fig. 1A shows a typical field of images derived from these preparations. A set of 201 individual images of the complex in negative stain were aligned and averaged (Fig. 1, B and C). The appearance of the average (Fig. 1C) and indeed the individual images indicate that the complex is bipolar. The complex is 45 nm long by 15 nm wide. At the center of the complex is an 11 $\times$ 15-nm bulge. Extending axially from the bulge are 11-nm-long rods ending in a sharp disk of density. A 4 $\times$ 8-nm knob caps each end. Although the particles all sit on the grid with their long axis essentially in the plane of the grid, they present different views about the long axis. The average therefore combines views at different angles and approximately represents a projection of the cylindrically symmetric structure. From this average, we generated a three-dimensional reconstruction of the cylindrically averaged structure (16). Fig. 1D shows a central slice through the reconstruction. The long axis of the complex appears darker than the surroundings, indicating a hollow, barrel-like assembly. Given that the lzTar$_c$ protein that was used to form the complexes is a rod-like tetramer structure (12), and there are $\sim$28 lzTar$_c$ chains in each complex, the most likely rotational symmetry for the long...
The end of each bar are the actual numbers of particles counted for each label for the three classes (end, middle, and undecided). The numbers at

\[ D \]

is a schematic of the complex with the position of a gold label indicated. The axis of the complex is 7-fold, although 6- or 8-fold symmetries cannot be excluded.

**Subunit Organization within \( \text{IzTar}_{r-C} \text{-CheW-CheA} \) Complexes Determined by Immunolabeling**—We have used antibodies against CheA, CheW, and the leucine zipper domain to localize these components. Additionally, in some experiments we added CheY, which binds to CheA, in combination with anti-CheY antibodies. Even though the fraction of decorated complexes was low, we were able to score from 98 to 212 antibody-decorated complexes for each of the decoration experiments. Fig. 2, A–C show representative images from typical experiments. Antibodies were decorated with protein A that has a 5-nm gold tag on a long (10–15 nm) tether. All antibodies were polyclonal and hence could potentially bind at multiple sites over the surface of a given subunit. Even with the variations in the positions of the gold tags, however, we could distinguish whether the leucine zippers were in the middle and CheA or CheW at the ends or vice versa. Given the range of possible distances from the gold tag to the epitope, we divided the structure into just the middle region and the end regions and scored each gold tag as to whether it lay at the ends (Fig. 2A) or in the middle (Fig. 2B). About 15% of the tags lay near the boundary between the middle and the ends (Fig. 2C). We classified these as undecided. Fig. 2D is a histogram of our results given in percentages with the actual numbers of images counted in each category indicated at the ends of the bars. (Note that immunolabeling with the affinity purified antibodies against leucine zipper and CheY produced more clear-cut localization than labeling with antibodies against CheA or CheW.) It is clear from these results that, contrary to what was first surmised (12), CheA and CheW are positioned at the middle of the complex, and the leucine zippers of the receptor construct are at the ends.

**DISCUSSION**

Electron microscopy and image analysis have provided a low resolution picture of the structure of complexes formed between \( \text{IzTar}_{r-C} \) CheA, and CheW. The major building block of these complexes, \( \text{IzTar}_{r-C} \), forms tetramers in solution (11, 12), appearing as long thin rods in electron micrographs (12). Since previous studies had indicated that receptors were dimeric (17), we assumed, prior to the results reported here, that tetramer formation resulted from an association of the leucine zipper portion of the molecule (12). This led Liu et al. (12) to propose a model wherein rod-like \( \text{IzTar}_{r-C} \) coiled-coil dimers extended out from a central assembly of leucine zippers to form a barrel with CheW and CheA bound to the ends. The immunolabeling experiments described above have clearly established that this is not the case. Instead, the \( \text{IzTar}_{r-C} \) subunits are oriented with the opposite polarity so that the region that binds CheW and CheA is located near the center of the barrel and the leucine zippers are at the ends. Cytoplasmic domain constructs of the receptor form tetramers when fused to completely different leucine zipper dimerization domains, from GCN4 (11) and from C/EBP (12). This indicates that tetramer formation is a property of the receptor’s cytoplasmic domain rather than the leucine zipper, and it is likely that these structural arrangements also occur in vivo.

Despite considerable effort by several groups, there have been only two types of soluble receptor constructs that have led to the formation of complexes with CheA and CheW with high levels of histidine kinase activity. The first type, exemplified by \( \text{IzTar}_{r-C} \) has a leucine zipper dimerization domain fused to the NH\(_2\) terminus of the cytoplasmic coiled-coil domain (9, 11). The second type is comprised solely of sequences from the cytoplasmic domain. In the latter case, however, a portion of the receptor corresponding to about half of the \( \alpha \)-helix that forms the second antiparallel strand of the coiled-coil (o2) is deleted (9, 10). This deletion leaves the NH\(_2\)-terminal half of the first \( \alpha \)-helix (\( \alpha1 \)) without a partner. In the absence of its normal partner, this NH\(_2\)-terminal region would be expected to form a homodimeric parallel \( \alpha1-\alpha1 \) coiled-coil similar to the NH\(_2\)-terminal homodimeric coiled coil provided by the leucine zipper adducts. All of these receptor constructs produced active complexes that appeared to be very large structures comprised of numerous receptor subunits (10, 12). These findings suggest that formation of a homodimeric parallel \( \alpha1-\alpha1 \) coiled-coil greatly facilitates formation of active receptor-kinase complexes.

The crystal structure of the cytoplasmic domain of Tsr indicates a dimer formed by supercoiling of monomeric antiparallel \( \alpha1-\alpha2 \) coiled coils (7). Alternatively, dimerization resulting from formation of the \( \alpha1-\alpha1 \) coiled-coil would produce a supercoiled structure with the antiparallel \( \alpha2 \)-helices folding back to lie on opposing hydrophobic surfaces between the parallel \( \alpha1-\alpha1 \) coiled-coils, as has been concluded from the cysteine and disulfide scrambling studies (18). Presumably, the function of the leucine zipper is to favor this type of coiled-coil interaction. The aspartate receptor cytoplasmic domain is highly dynamic (19). Unlike a structure originating from dimeric interaction between antiparallel coils, the antiparallel strands in the leucine zipper-induced dimer could easily dissociate and participate in interactions with other dimers (20). Such helix swapping inter-

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**FIG. 2** A gallery of examples of electron micrographic images of the complex labeled with primary antibodies and gold-labeled protein A. We classified the positions of the labels as end (A), middle (B), or in between these two extremes (undecided) (C). The inset is a schematic of the complex with the position of a gold label indicated. **D** is a histogram of our results as described above with three classes (end, middle, and undecided). The numbers at the end of each bar are the actual numbers of particles counted for each group.
action could account for the rod-like tetramers formed by lzTarC alone (Fig. 3). Helix swapping between receptors in the membrane would provide a mechanism whereby each receptor dimer could have interactions with up to four neighbor dimers. These types of interactions would contribute to the formation of the extensive receptor clusters that have been observed in intact cells (21–23).

Given our low resolution picture of the lzTarC-CheW-CheA complex and the general organization of the components, we ask how the component parts fit into the structure. A bipolar barrel formed from the four-helix bundles like those depicted in Fig. 3 would generate a structure with an outer diameter of 8 nm, which fits well with the observed width of the rod portion of the complex (Fig. 4). The expected length of these four-helix bundles is 19 nm; the length of the leucine zipper is ~6.5 nm (43 amino acid residues); the length of the 7-amino acid flexible linker that connects the zipper to the cytoplasmic domain is 1–2 nm. The COOH-terminal ends have an additional 35 residues not present in the atomic model of the receptor structure. Fig. 4 shows a model that can account for images of the receptor constructs. In it, the COOH-terminal ends form the sharp disk of density, and the leucine zippers form the knobs at the ends of the complex. The long four-helix bundles make up the rod-like features seen in the images. The four-helix bundles, which are 19 nm in length, are about 1.5 nm longer than the corresponding dimension measured in the image. This discrepancy may be accounted for if there is a slight shrinkage in negative stain. Shrinkage in negative stain is well known and can exceed the 10% suggested here (24–26). The long dimension of the CheA dimer is ~12 nm (27). This distance fits well with the dimensions of the central part in the soluble complex (Fig. 4). CheW is much smaller than CheA and can easily fit into this region since it is likely to bind to the same sites as CheA (28).

Most of the previous literature assumes that the functional receptor unit is a dimer. This conclusion derives almost entirely from x-ray crystallographic studies of the periplasmic sensing domain. The isolated aspartate-binding domain of Tar is a symmetric dimer of two four-helix bundles with aspartate-binding sites bridging the dimer interface (29). These results with the periplasmic domain cannot be used to infer the structure of the cytoplasmic portions of the receptor. Indeed, in vivo studies of this domain have indicated a variety of quaternary states, ranging from loosely folded monomers to large aggregates (30). The dimeric models of the intact receptors that have been advanced previously are composites formed by joining the dimeric unit from the x-ray crystal structure of a fragment of the cytoplasmic domain of Tar, with the crystal structure of the Tar aspartate-binding domain dimer (7). In this type of model the cytoplasmic domain is generally represented as a well-defined dimeric structure, but in the crystal of the Tsr cytoplasmic domain fragment there are numerous higher order interactions as well, and the unit cell is essentially a trimer of dimers (7). Another assumption of the model is that the transmembrane and linker regions are directly connected by a continuous α-helical domain extending from the periplasmic dimer at the outside surface of the membrane to the coiled-coil dimer in the cytoplasm. There is evidence, however, that the linker region is a distinct domain in its own right and is not completely α-helical (5, 31, 32).

It is now well established that most of the several thousand chemotaxis receptor subunits in E. coli are localized together with CheA and CheW in one or two tight clusters, generally at the poles of the cell (21, 22, 33). Although CheA and CheW are essential for optimal cluster formation, the receptors are capable of extensive higher order interactions that function to initiate cluster assembly (34). The structure of our soluble receptor signaling complexes provides clues as to how receptor clusters may assemble. The process could be initiated by interactions between monomeric antiparallel coiled-coil chains coming together to form dimers and higher order structures like the trimers of dimers that are seen in the x-ray crystal structure of the isolated Tsr coiled-coil domain (7). At some stage the intrasubunit antiparallel coiled-coil interactions could be converted to intersubunit parallel α1-α1 coiled-coils with antiparallel interactions between subunits like those that appear to underlie the tetrameric structure as depicted in Fig. 3. CheA and CheW binding might facilitate this transition to help organize the receptor structures into higher order assemblies. The two receptor-binding domains of CheA are separated by 8 nm (19), which is sufficient to bridge between two tetramers. The linker domain at the membrane-cytoplasm interface would mediate sensory interactions between the transmembrane sensory portions of the receptor and the underlying coiled-coil scaffold.
How does this relate to the mechanism of transmembrane signaling? Models have been advanced suggesting that higher order receptor interactions could account for the extremely high sensitivity of *E. coli* to very small changes in attractant concentrations (23, 35–37). It has also been shown that different classes of receptors interact with one another to effect adaptive responses to long term changes in background stimulation (38, 39). A highly ordered array of numerous different receptors provides a much greater potential for information processing than would a corresponding number of independently functioning receptors scattered over the cell surface. The cytoplasmic signaling domains of chemotaxis receptors are highly conserved in virtually all motile prokaryotes, but the periplasmic sensing domains are highly variable (5). There are, for instance, no known homologues of the Tar sensing domain in any bacterium aside from species closely related to *E. coli*. Even homologous periplasmic sensing domains in *E. coli* have little sequence identity. Given this diversity, it is unlikely that a specific change in sensing domain conformation provides a general mechanism to regulate the receptor-signaling network inside the cell. Instead, one would expect that any number of subtle stimulus-induced changes could cooperate to significantly perturb the dynamic architecture of the signaling array to produce a response. Transmembrane signaling could be accomplished by a variety of different conformational changes in sensory domain structure that lead to altered packing interactions within a compact sensory array at the periplasmic surface of the membrane.

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