Interaction between the τ subunit of the DNA polymerase III holoenzyme and the DnaB helicase is critical for coupling the replicase and the primosomal apparatus at the replication fork (Kim, S., Dallmann, H. G., McHenry, C. S., and Marians, K. J. (1996) Cell 84, 643–650). In the preceding manuscript, we reported the identification of five putative structural domains within the τ subunit (Gao, D., and McHenry, C. (2000) J. Biol. Chem. 275, 4433–4440). As part of our systematic effort to assign functions to each of these domains, we expressed a series of truncated, biotin-tagged τ fusion proteins and determined their ability to bind DnaB by surface plasmon resonance on streptavidin-coated surfaces. Only τ fusion proteins containing domain IV bound DnaB. The DnaB-binding region was further limited to a highly basic 66-amino acid residue stretch within domain IV. Unlike the binding of immobilized τ to the DnaB hexamer, the binding of monomeric domain IV to DnaB was dependent upon the density of immobilized domain IV, indicating that DnaB is bound by more than one τ protomer. This observation implies that both the leading and lagging strand polymerases are tethered to the DnaB helicase via dimeric τ. These double tethers of the leading and lagging strand polymerases proceeding through the τ–τ link and an additional τ–DnaB link are likely important for the dynamic activities of the replication fork.

The DNA polymerase III holoenzyme is responsible for the replication of the *Escherichia coli* chromosome. Like other replicases from eukaryotes and prokaryotes, the holoenzyme contains three functional subassemblies (for reviews see Refs. 1–3): the DNA polymerase III (αεθ) core, the β sliding clamp processivity factor, and the DnaX complex, a clamp assembly apparatus. The DNA polymerase III core contains the α, ε, and θ subunits and provides the polymerase function. The DnaX complex (τγδεχψ) is a multiprotein ATPase that recognizes the primer terminus and loads the β processivity factor onto DNA.

The τ and γ subunits are different translation products of the *dnaX* gene (4–7). The τ subunit plays central roles in the structure and function of the holoenzyme. It interacts with the core polymerase to coordinate leading and lagging strand synthesis (8, 9). τ also interacts with DnaB helicase to couple the replicase with the primosome and mediate rapid replication fork movement (10, 11). These two important functions of τ reside in C-τ, a proteolytic fragment consisting of its unique C-terminal 213 amino acid residues. τ binds tightly to the α subunit; the shorter translation product γ does not. C-τ is a monomer and binds α with a 1:1 stoichiometry as determined by sedimentation equilibrium analyses (12). Results from a recent study indicated that C-τ binds DnaB, can partially replace full-length τ in reconstituted rolling circle replication reactions, and effectively couples the leading strand polymerase with DnaB helicase at the replication fork (12). DnaB helicase is composed of six identical subunits and is a stable hexamer over a wide range of concentration in the presence of magnesium ions (13, 14).

In the preceding manuscript, we reported that τ comprises five potential structural domains (15). Domains I, II, and III are common to both γ and τ. Domain IV includes 66 amino acid residues of the C-τ sequence and the C-terminal 17 residues of γ. Domain V corresponds to the 147 C-terminal residues of the τ subunit. Based on these assignments, biotin-hexahistidine-tagged τ proteins lacking specific domains were produced. Results from binding studies employing these truncated fusion proteins indicated that the binding site of τ for α subunit lies within its C-terminal 147 amino acid residues (domain V).

The objective of this study was to determine the domain(s) of the τ subunit involved in binding DnaB. Biotin-hexahistidine-tagged τ proteins lacking specific domains were expressed and purified. Analysis of DnaB binding to these truncated τ proteins by surface plasmon resonance permitted the assignment of the DnaB-binding domain of τ.

**EXPERIMENTAL PROCEDURES**

**Strains**—*E. coli* DH5α and HB101 were used for initial molecular cloning procedures and plasmid propagation. *E. coli* BL21(A DE3) was used for protein expression.

**Buffers**—Buffer L, Buffer W and HKGM Buffer were prepared as previously described (15).

**Chemicals and Reagents**—SDS-polyacrylamide gel electrophoresis protein standards were obtained from Amersham Pharmacia Biotech, and prestandardized molecular mass markers were from Bio-Rad or Life Technologies, Inc. N,N′-dicyclaurylcarbodiimide, and ethanolamine hydrochloride were obtained from Pierce. CM5 sensor chips (research grade), P-20 surfactant, N-hydroxysuccinimide, [1-ethyl-3-(3-dimethylaminopropyl) carbodiimide, and ethylenediamine hydrochloride were obtained from BIAcore Inc.

**Proteins**—Three biotin-tagged τ proteins C(0)τ, C(Δ147)τ, and

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1 The abbreviations used are: NTA, nitrotriacetic acid; PCR, polymerase chain reaction; RU, resonance unit.
**RESULTS**

**Expression and Purification of the Truncated τ Fusion Proteins**—The τ subunit binds to DnaB helicase and is the only subunit within the holoenzyme shown to interact with DnaB (10). The unique C terminus of τ (C-τ) bound DnaB in a coupled immunoblotsing method (12). To confirm this observation and more precisely map the DnaB binding region of τ, a series of truncated τ proteins lacking specific domains were produced, and their interactions with DnaB helicase were quantified using BLAcore methodology. The τ fusion proteins employed in this study included C(0)-τ (domains I-V), C(Δ417)-τ (domains I-IV), C(Δ213)-τ, which was equivalent to γ (domains I-III plus 17 amino acids of domain IV), N-Δ413τ (domains IV and V), and N-Δ430τ, which was equivalent to C-τ (the C-terminal 66 residues of domain IV plus domain V in its entirety). The truncated terminus of each fusion protein was tagged with a peptide containing both a hexahistidine sequence to aid in purification as well as a short biotinylation sequence. The biotinylation sequence enabled oriented immobilization of the fusion proteins onto BLAcore sensor chips via biotin-streptavidin binding. C(0)-τ, C(Δ417)-τ, and N-Δ413τ were expressed and purified as previously described (15). C(Δ213)-τ and N-Δ430τ were expressed in the BL21 (DE3) strain by induction with isopropyl-β-D-thio-galactoside and reached similar expression levels (2–5% of total cell proteins). Both C(Δ213)-τ and N-Δ430τ were purified by Ni²⁺-NTA affinity chromatography. After Ni²⁺-NTA purification, C(Δ213)-τ was obtained at 80% purity, and N-Δ430τ at 90% purity as determined by SDS-polyacrylamide gel electrophoresis analysis (Fig. 1). The activities of the fusion proteins were ascertained by their ability to replace γ or τ in DNA polymerase III reconstitution assays (15). The specific activity of C(Δ213)-τ was 5.5 × 10⁶ units/mg, similar to that of full-length C(0)-τ (5.7 × 10⁶ units/mg). As expected, no holoenzyme reconstitution activity was detected for N-Δ430τ, which lacks the γ sequence required for assembly of the β processivity factor on DNA.

**DnaB Binding to τ Proteins Containing Domain IV**—The interaction between DnaB and C(0)τ was first characterized via use of BLAcore technology. C(0)τ (2025 RU) was immobilized onto a streptavidin sensor chip. DnaB solutions of varying concentrations were passed over the immobilized C(0)τ, and binding activity was monitored (Fig. 2A). Attempts to fit the dissociation phase to a single first-order dissociation equation were unsuccessful, suggesting that a more complex mechanism was operative. To simplify the kinetic analysis, a limited interval (35–125 s following the starting point of dissociation) was analyzed from each binding curve and fit to a model in which two simultaneous independent dissociation processes occur. The two apparent dissociation rate constants kₐₘₐₗ and kₐₘᵢₙᵢₜ (Table II) corresponded to 70–80% and 20–30% of the dissociating species, respectively. kₐₘₐₗ was used to calculate the apparent association rate (kₐ). The apparent kₐ was calculated from kₐₘₐₗ and kₐₘᵢₙᵢₜ. The interaction between DnaB and C(0)τ had an apparent Kₐ of 4 nM (Table II). Under the conditions employed in these studies, DnaB is known to exist as a

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**Table I**

| Oligonucleotide number | Use | Sequence* |
|------------------------|-----|-----------|
| C-213p1                | C-Δ213τ | TACACATTTCCGCGCTGAGATGG |
| C-231p2                | C-Δ213τ | GGGACATGACCTTTTTCGTCG |
| N-430p1                | N-Δ430τ | AACTGCGAGAAGTGACGCGG |
| N-430p2                | N-Δ430τ | CTGGCGATGGGAGACCCAC |

* The underlined regions are complementary to the DnaX gene sequence on the template.

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**Other Procedures**—DNA polymerization assays, protein determinations, and SDS-polyacrylamide gel electrophoresis were performed as described in the preceding paper (15).
DnaB-binding Domain of τ

**FIG. 1. Purified truncated τ fusion proteins.** The upper panel shows the truncated fusion proteins of τ used in this study: C(0)τ (domain I-V); C-Δ147τ (domains I-IV); C-Δ213τ (domains I-III + 17 amino acids of domain IV), which is equivalent to the γ protein plus a C-terminal tag; N-Δ413τ (domains IV and V); and N-Δ430τ contains the intact domain V and the majority of domain IV lacking its N-terminal 17-amino acid sequence. The rectangular box represents the fusion peptide. The lower panel is the Coomassie Blue-stained 12% SDS-polyacrylamide gel of 1.5 μg of each purified protein after Ni²⁺-NTA chromatography (C-Δ147τ and N-Δ413τ were shown in the preceding paper).

hexamer (14), and C(0)τ is a tetramer (18). The binding ratio of the DnaB hexamer (DnaB₆) to the C(0)τ tetramer [C(0)τ]₄ was 0.72, indicating that these multimers likely interact with a 1[DnaB₆]: 1[C(0)τ]₄ stoichiometry.

C-Δ213τ, equivalent to C-terminally tagged γ, was captured onto the streptavidin-derivatized sensor chip (3400 RU), followed by injection of DnaB (1 μM). No interaction between C-Δ213τ and DnaB was detected (Fig. 2B), consistent with the previous finding that γ does not interact with DnaB helicase (10).

Next, DnaB samples (0.05–0.5 μM) were injected over immobilized C-Δ147τ (2860 RU) (Fig. 2C). An apparent $K_d$ of 5 nM was obtained, which is similar to that of the C(0)τ-DnaB interaction (Table II). This suggests that C-Δ147τ contains elements sufficient for binding to DnaB at the same level observed for the intact τ subunit. C-Δ147τ (domains I-IV) bound DnaB, but C-Δ213τ (domains I-III) did not, localizing the region required for DnaB binding to somewhere within domain IV.

**DnaB Recognizes a 66-Amino Acid Sequence within Domain IV**—To confirm that domain IV was the DnaB-binding domain, N-Δ430τ (1200 RU) was captured onto a BIAcore sensor chip, and its interaction with DnaB was assessed (Fig. 3A). The dissociation phase did not fit to a single first-order dissociation equation, so the binding data were fit to the model that assumes two parallel dissociation processes. The apparent $K_d$ was about 8 μM, which is similar to that of the interaction between DnaB and C(0)τ (Table II). The sum of these results indicates that the DnaB binding site is located within the unique C-terminal 66 residues of the τ subunit.

The C-terminal 17 amino acid residues of domain IV are lacking in N-Δ430τ. To investigate whether these 17 residues provide additional binding energy for the τ-DnaB interaction, DnaB binding studies using fusion protein N-Δ413τ were per-per-
DnaB-binding Domain of τ

**TABLE II**

| τ derivative | Apparent $k_{off}$ | Apparent $k_{on}$ | Apparent $K_d$ |
|--------------|-------------------|-------------------|---------------|
| C(0)τ        | Major             | Minor             |               |
|              | 10^{-3} s^{-1}    | 10^{-3} s^{-1}    |               |
| C−Δ147τ      | 10^{-3} s^{-1}    | 10^{-3} s^{-1}    |               |
| N−Δ413τ      | 10^{-3} s^{-1}    | 10^{-3} s^{-1}    |               |

* Results are the averages of two separate experiments.

**TABLE III**

| Immobilized N−Δ430τ | Density | DnaB Bound | Binding ratio (DnaB$_6$/N−Δ430τ)$_2$ |
|---------------------|---------|------------|-------------------------------------|
| RU                  | Density |
| 146 56 0 0          | 1437 0.04 | 0.05 (28%) | 0.48 |
| 720 261 315 0.08   | 419 0.15 | 0.03 (22%) | 0.24 |
| 1156 419 937 0.23  | 724 0.23 | 0.02 (16%) | 0.18 |
| 2700 978 3774 0.24 | 261 0.24 | 0.01 (8%)  | 0.09 |

* The concentrations of the immobilized N−Δ430τ and C(0)τ were calculated using the following equation (19): concentration (M) = RU/100 × molecular weight of the immobilized species (M). The binding ratio of the DnaB hexamer to the N−Δ430τ or C(0)τ dimer was determined by the following equation: binding ratio = (RU of DnaB bound)/(RU of dimer immobilized) × (dimer $M_r$/DnaB$_6$).

with sensor chips bearing differing amounts of N−Δ430τ (146 RU-2700 RU). The corresponding densities of the six different levels of N−Δ430τ tested are shown in Table III. Binding of DnaB to immobilized N−Δ430τ at 146 RU was not observed. Increased binding ratios of DnaB to N−Δ430τ were observed for surfaces bearing greater densities of N−Δ430τ (Fig. 4). If we assume that each DnaB$_6$ binds two (N−Δ430τ)$_2$ molecules, then the binding ratio of DnaB to N−Δ430τ is increased from 0.04 to 0.24 within the range of the amount of immobilized N−Δ430τ tested (Table III). The same apparent dissociation and association rate constants for the DnaB and N−Δ430τ interaction were obtained at different N−Δ430τ density as reported in Table II. These results are consistent with the multivalent binding of DnaB and N−Δ430τ. The observed $K_d$ is the product of the individual $K_d$ values for single site binding interactions. No binding was observed at low N−Δ430τ density, suggesting that the monomeric τ-DnaB$_6$ interaction is too weak to be observed with the BIAcore methodology. The apparent $K_d$ values of the DnaB-N−Δ430τ interaction and DnaB-C(0)τ interaction were the same, suggesting that the interaction between DnaB and C(0)τ is also multivalent; more than one C(0)τ monomer binds each DnaB$_6$. The binding ratio between DnaB and N−Δ431τ was also N−Δ431τ density-dependent and increased with increased immobilized N−Δ431τ density (data not shown).

To ensure that the observed binding ratio of the hexameric DnaB to the tetrameric C(0)τ was not density-dependent, the binding ratio of DnaB$_6$ to C(0)τ was examined at an increased density (4262 RU) of C(0)τ on a sensor chip. In a previous experiment, 2025 RU of C(0)τ was used (Fig. 2A), and a binding ratio of 0.72 DnaB$_6$ to C(0)τ was observed. The C(0)τ concentrations in these two different experiments corresponded to 568

**FIG. 3.** N−Δ430τ and N−Δ413τ bind DnaB. Streptavidin was chemically coupled onto the BIAcore sensor chips as described under “Experimental Procedures.” 1200 RU of N−Δ430τ (A) and 1293 RU of N−Δ413τ (B) were captured onto streptavidin-derivatized sensor chips, and DnaB$_6$ diluted in HKGM buffer at the indicated concentrations was injected over the chips bearing immobilized N−Δ430τ and N−Δ413τ, respectively. Control injections over a streptavidin derivatized sensor chip were performed and subtracted from the data shown.

formed. However, the DnaB/N−Δ413τ interaction was characterized by an apparent $K_d$ of 5 nM, which is similar to that observed for the interaction of DnaB with N−Δ430τ (Fig. 3B and Table II). Thus, it is unlikely that the C-terminal 17 residues of domain IV contribute significantly to DnaB binding interactions.

More than One τ Protomer Binds a DnaB Hexamer—In the preceding experiment, the binding ratio of DnaB$_6$ to the monomeric N−Δ430τ was less than 0.1. This value is significantly different from 0.72, the observed ratio for the interaction between DnaB$_6$ and C(0)τ. One potential underlying cause of the low binding ratio for the former interaction is the binding of DnaB$_6$ to more than one immobilized N−Δ430τ molecule. To test this hypothesis, we examined the interactions of DnaB (1 μM) with sensor chips bearing differing amounts of N−Δ430τ (146 RU-2700 RU). The corresponding densities of the six different levels of N−Δ430τ tested are shown in Table III. Binding of DnaB to immobilized N−Δ430τ at 146 RU was not observed. Increased binding ratios of DnaB to N−Δ430τ were observed for surfaces bearing greater densities of N−Δ430τ (Fig. 4). If we assume that each DnaB$_6$ binds two (N−Δ430τ)$_2$ molecules, then the binding ratio of DnaB to N−Δ430τ is increased from 0.04 to 0.24 within the range of the amount of immobilized N−Δ430τ tested (Table III). The same apparent dissociation and association rate constants for the DnaB and N−Δ430τ interaction were obtained at different N−Δ430τ density as reported in Table II. These results are consistent with the multivalent binding of DnaB and N−Δ430τ. The observed $K_d$ is the product of the individual $K_d$ values for single site binding interactions. No binding was observed at low N−Δ430τ density, suggesting that the monomeric τ-DnaB$_6$ interaction is too weak to be observed with the BIAcore methodology. The apparent $K_d$ values of the DnaB-N−Δ430τ interaction and DnaB-C(0)τ interaction were the same, suggesting that the interaction between DnaB and C(0)τ is also multivalent; more than one C(0)τ monomer binds each DnaB$_6$. The binding ratio between DnaB and N−Δ413τ was also N−Δ413τ density-dependent and increased with increased immobilized N−Δ413τ density (data not shown).

To ensure that the observed binding ratio of the hexameric DnaB to the tetrameric C(0)τ was not density-dependent, the binding ratio of DnaB$_6$ to C(0)τ was examined at an increased density (4262 RU) of C(0)τ on a sensor chip. In a previous experiment, 2025 RU of C(0)τ was used (Fig. 2A), and a binding ratio of 0.72 DnaB$_6$ to C(0)τ was observed. The C(0)τ concentrations in these two different experiments corresponded to 568
and 270 μM of C(0)τ as monomer, within the density range of N-Δ430τ used in the density dependence experiment (Table III). The observed binding ratio of DnaB6 to C(0)τ was 0.69, which was not significantly different from the ratio obtained when using with 2025 RU of C(0)τ (Table III).

**DISCUSSION**

In the preceding paper, we detailed our use of limited proteolysis studies to identify five putative structural domains of the τ protein (15). Domains I–III are common to both τ and γ. Domain IV is composed of 17 amino acid residues from the C-terminal end of γ plus 66 amino acids from the unique C terminus of τ. Domain V is located at the C-terminal end of τ. One function of τ is to bind DnaB, coupling the holoenzyme with the primosome at the replication fork. C-τ, the unique C terminus of τ, bound DnaB in a coupled immunoblotting method (12). In reconstituted rolling circle replication reactions, C-τ can partially replace full-length τ in coupling the leading strand polymerase with the DnaB helicase at the replication fork (12).

This study further defined the DnaB binding domain of τ by analyzing the interactions of DnaB with several truncated τ proteins. N-Δ413τ, N-Δ430τ, C-Δ147τ, and C(0)τ bound DnaB with similar apparent Kd values. Because complicated binding kinetics were operative, the apparent Kd values we obtained in this study were not the true constants. However, the resulting apparent Kd values presumably contain the same systematic errors and therefore permit a quantitative comparison of relative affinities. The relative binding affinities of the different τ fusion proteins for DnaB indicate that τ amino acid residues 431–496 are sufficient for DnaB binding. This 66-residue stretch corresponds to the C-terminal portion of τ domain IV.

Although similar apparent Kd values were obtained for the interactions of DnaBτC(0)τ and DnaBτ(N-Δ430τ), the binding ratios for the DnaBτ(N-Δ430τ), the binding ratios for the DnaBτ(N-Δ430τ) were density-dependent. We conclude that more than one N-Δ430τ monomer is required to bind DnaB and that the interactions between DnaB and τ involved multivalent binding. Thus, the true microscopic Kd for binding of DnaBτ to a single N-Δ430τ was too weak to observe using a BIAcore. At higher N-Δ430τ densities, binding was observed between DnaBτ to two or more N-Δ430τ molecules; the observed macroscopic Kd is roughly equal to the product of each of the constituent microscopic Kd values.

2 Assuming that the 8 nm apparent Kd resulted from (N-Δ430τ)2-DnaBτ interactions, the affinity between DnaB and each monomeric N-Δ430τ would be in the 900 μM range provided that there was no cooperativity involved (8 μM)2 = 900 μM. This low (900 μM) affinity range is consistent with the lack of detected interaction.
expected DNA binding sphere would be in the range from $4/3 \pi r^3(40 \text{ Å})^3$ to $4/3 \pi r^3(140 \text{ Å})^3$ (268–11480 nm$^3$, respectively).

If we assume that the interaction between $\tau$ and DnaB involves two N-430$\tau$ molecules, the calculated DNA binding sphere is 2500 nm$^3$, within the possible range for an interaction between two $\tau$ protomers and DnaB$_p$.

The notion that two $\tau$ protomers bind each DnaB hexamer is consistent with the presence of a $\tau$ dimer at the replication fork (Fig. 5). $\tau_2$ functions to dimerize the DNA polymerase III core to enable simultaneous synthesis of leading and lagging strands. We already know that the leading strand polymerase is tethered to DnaB (12). The findings presented in this report indicate that the same DnaB molecule couples both of the leading and lagging strand polymerases. Thus, a double tether exists between the leading and lagging strand polymerases, one through the $\tau$-$\tau$ link and the additional one through the $\tau$-DnaB link. This second tether might help keep the lagging strand associated with the replication fork (Fig. 5). Our mapping results demonstrate that the DnaB helicase binds $\tau$ domain IV and that the polymerase $\alpha$ subunit binds domain V (15). These findings indicate important roles that the C terminus $\tau$ plays in DNA synthesis.

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τ Binds and Organizes *Escherichia coli* Replication Proteins through Distinct Domains: DOMAIN IV, LOCATED WITHIN THE UNIQUE C TERMINUS OF τ, BINDS THE REPLICATION FORK HELICASE, DnaB
Dexiang Gao and Charles S. McHenry

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