Functionally Significant Mobile Regions of *Escherichia coli* SecA ATPase Identified by NMR*

Received for publication, September 9, 2002, and in revised form, October 10, 2002
Published, JBC Papers in Press, October 22, 2002, DOI 10.1074/jbc.M209237200

Yi-Te Chou‡, Joanna F. Swain§, and Lila M. Gierasch‡§¶

From the ‡Departments of Chemistry and §Biochemistry and Molecular Biology, University of Massachusetts, Amherst, Massachusetts 01003-4510

**SecA**, a 204-kDa homodimeric protein, is a major component of the cellular machinery that mediates the translocation of proteins across the *Escherichia coli* plasma membrane. SecA promotes translocation by nucleotide-modulated insertion and deinsertion into the cytoplasmic membrane once bound to both the signal sequence and portions of the mature domain of the preprotein. SecA is proposed to undergo major conformational changes during translocation. These conformational changes are accompanied by major rearrangements of SecA structural domains. To understand the interdomain rearrangements, we have examined SecA by NMR and identified regions that display narrow resonances indicating high mobility. The mobile regions of SecA have been assigned to a sequence from the second of two domains with nucleotide-binding folds (NBF-II; residues 564–579) and to the extreme C-terminal segment of SecA (residues 864–901), both of which are essential for preprotein translocation activity. Interactions with ligands suggest that the mobile regions are involved in functionally critical regulatory steps in SecA.

Preprotein translocation in bacterial cells is driven by SecA, a dissociable peripheral membrane ATPase that recognizes the protein precursor and assists its translocation across the inner membrane (1–5). Profound conformational changes are necessary for SecA to alternately bind to the preprotein/SecB complex, associate with the membrane/SecYEG translocase channel, and drive the stepwise translocation of the preprotein across the membrane. These steps are coordinated by ATP binding and hydrolysis (6–9).

SecA conformational changes appear to be achieved by as-yet poorly understood rearrangement of multiple independently folded domains of SecA. The domain structure of SecA was originally suggested by limited proteolysis, which yields reproducible patterns of resistant fragments (10–20). Moreover, several of these fragments have been expressed as individual domains (18–20). The recent crystal structure of the cytoplasmic form of *Bacillus subtilis* SecA (21) provides structural insights into the domain folds and interdomain packing and confirmed that this large protein is poised to undergo major domain rearrangements as it performs its multiple functions. Within the N-terminal domain are two nucleotide-binding folds, termed NBF-I and NBF-II, which exhibit high homology to RNA and DNA helicases from superfamilies 1 and 2 (22). Furthermore, as predicted from sequence data (23), all of the expected DEAD-box helicase motifs (I through VI) are found in structurally homologous locations in SecA NBF-I and -II. As in the helicases, the nucleotide-binding site is at the NBF-I/NBF-II interdomain junction, and the conserved motifs juxtapose the binding site. A novel insertion in NBF-I (residues 220–380, *Escherichia coli* numbering), not present in helicases, was found in an early study of *E. coli* SecA to cross-link a precursor protein (24) and therefore is termed the preprotein cross-linking domain. The last 39 residues at the C terminus of SecA are disordered and not resolved in the crystal structure.

Structures of DEAD-box helicases suggest that their mechanisms rely on regulated rigid body rearrangements of the two nucleotide-binding domains that require interdomain flexibility (22). Consistent with the structural similarity between SecA and the helicases, numerous published data support the importance of interdomain interactions in the SecA mechanism. Schmidt et al. (25) reported that several suppressors of protein secretion defects and azide-resistant mutants of SecA perturbed the SecA endothermic transition by weakening interdomain contacts. Several laboratories, including our own, have shown that any disruption of the C terminus of SecA, through truncation or partial unfolding, leads to enhanced ATPase activity (10, 13, 14). Deletion of a specific region of the C-terminal sequence (residues 783 to 795) recapitulates the ATPase activation observed in C-terminal truncated versions of SecA; this region has been termed by Economou and coworkers (15) the intramolecular regulator of ATP hydrolysis 1 (IRA1). These same researchers presented biochemical evidence that a region nearly coincident with NBF-II (amino acids 462 to 610, dubbed IRA2) regulates the nucleotide binding and release function of NBF-I (16). Recent cross-linking and biochemical experiments by Economou and coworkers (17) suggest that binding of signal peptides occurs to a segment from residues 219 to 244 and that IRA1 regulates access to this binding site. Binding of SecB to the C-terminal region of SecA enhances its ATPase activity and promotes exchange with membrane-bound SecA (26). In addition to the domain reorganization within a SecA monomer, binding to phospholipids or signal peptides modulates SecA oligomerization with lipids favoring monomeric states and signal peptides shifting the equilibrium toward dimers (27).

Clearly, a deliberate balance of interdomain interactions poises SecA to undergo profound conformational change in a regulated way, depending upon binding to its many ligands. The

---

* This work was supported by National Institutes of Health Grant GM34962 (to L. M. G.). The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

‡ To whom correspondence should be addressed. Tel.: 413-545-6094; Fax: 413-545-1289; E-mail: gierasch@biochem.umass.edu.

¶ The abbreviations used are: NBF, nucleotide-binding fold; HSQC, heteronuclear single quantum correlation; IRA, intramolecular regulator of ATP hydrolysis; NOE, nuclear Overhauser effect; NOESY, NOE spectroscopy; TMSp, 3-(trimethylsilyl)propionate-2,2,3,3-d4; TOCSY, total correlation spectroscopy; wt, wild-type.

---

This paper is available on line at http://www.jbc.org
ligand-modulated changes in interdomain packing in SecA necessarily require regions of sequence that are able to adopt alternate conformations in order to allow rigid body rearrangements. It is reasonable to anticipate that some regions of SecA will show enhanced mobility, enabling them to perform regulatory or linker roles in its mechanistic cycle. Static pictures of the SecA structure do not reveal the locations and dynamics of these functionally important regions. Therefore, we embarked on an effort using NMR to ask whether there are mobile regions in SecA and to understand their roles in its domain rearrangements. This approach had been productive in our previous work on GroES, where the GroEL-interactive regions emerged as ‘mobile loops’ that displayed narrow resonances in both one- and two-dimensional NMR spectra (28). In examination of SecA, we indeed found several narrow resonances in NMR spectra, and we have been able to carry out sequence-specific assignment of these resonances. Strikingly, these regions have been assigned to residues within NBF-II (mobile region 1, residues 564 to 579) and the C terminus (mobile region 2, residues 864 to 901), both of which play critical roles in SecA function and both of which are structurally related to the proposed IRA regions.

EXPERIMENTAL PROCEDURES

Reagents—Unless specifically mentioned, standard laboratory reagents were purchased from Sigma or VWR. All deuterated or 15N-labeled compounds were purchased from Cambridge Isotope Laboratories (Andover, MA) or Isotec (Miamisburg, OH).

SecA Preparation—SecA and variants were purified from overexpressing E. coli strains BL21 (p77-SecA2, p77-SecA95) kindly provided by D. Oliver (Wesleyan University) (29, 30). All proteins were purified as described previously (10) with minor modifications. Uniform 15N-labeled SecA protein was obtained by growing cells in M9 minimal medium containing 15NH4Cl. The sample with reverse selective labeling of arginine residues was prepared by providing cells with 250 mg/liter culture of unlabeled arginine in addition to 15NH4Cl. Purity of SecA was assessed by SDS-PAGE; samples of lower than 98% purity were found to aggregate during concentration. The principal impurities observed were proteolytic breakdown products (molecular masses 95, 64, 43 kDa), most of which are less soluble than the intact protein. Observed were proteolytic breakdown products (molecular masses 95, 64, 43 kDa), most of which are less soluble than the intact protein. ATPase activity of purified SecA was verified to be comparable with that of 15N-labeled SecA protein was obtained by growing cells in M9 minimal media containing 15NH4Cl. The sample with reverse selective labeling of arginine residues was prepared by providing cells with 250 mg/liter culture of unlabeled arginine in addition to 15NH4Cl. Purity of SecA was assessed by SDS-PAGE; samples of lower than 98% purity were found to aggregate during concentration. The principal impurities observed were proteolytic breakdown products (molecular masses 95, 64, 43 kDa), most of which are less soluble than the intact protein.

NMR Studies—At least three individual preparations of fresh protein samples were used to confirm the reproducibility of the data. Samples for NMR spectrometry consisted of 0.8–1.2 mM SecA (monomer) in 25 mM KC1, 25 mM potassium phosphate buffer (pH 7.0) with 10% D2O with or without 0.02% azide. Under these conditions, SecA is overwhelmingly in the dimer state (31). Spectra were obtained using Bruker AMX 500 or Avance 600 NMR spectrometers at 25 °C. 1H chemical shifts were referenced with respect to the methyl protons of 3-(trimethylsilyl) propionate (TMS) (0 ppm). Data were processed within the FELIX 97 program (Biovax Technologies, San Diego) running on a Silicon Graphics Indigo work station (Mountain View, CA).

1H, 15N HSQC experiments were recorded using the States-TPPI method (32) with 32–64 scans; the spectral widths and number of points were 7000–8000 Hz × 1650–4000 Hz and 768 × 128 points in the 1H and 15N dimensions, respectively. The spectral widths and number of complex points in the F3, F2, and F1 dimensions, the number of scans per free induction decay, and the total measurement time of three-dimensional 1H, 15N TOCSY-HSQC and 1H, 15N NOE-HSQC (32) were 7000 × 1650 × 7000 Hz, 512 × 32 × 64, and 16 scans for ~5 days. The mixing times were 29 ms for the TOCSY-HSQC and 70 ms for the NOE-HSQC experiments.

RESULTS

Assignment of Two Highly Mobile Regions of SecA by NMR Spectroscopy—As a 204-kDa homodimer, SecA is beyond the size of proteins routinely assignable via NMR methods. A roughly spherical protein of M, 200 would typically be expected to have broad signals (>100 Hz), consistent with an overall correlation time (τc) of ~100 ns (32). This protein, however, contains a set of relatively narrow peaks superimposed on an envelope of broad, overlapping resonances in the one-dimensional 1H and two-dimensional 1H, 15N HSQC NMR spectra (Fig. 2). The narrow resonances indicate that this protein contains some significantly mobile residues. We localized the amino acid sequences of sequential assignment to these resonances of SecA using methods of sequential assignment (32, 33), relying on 1H, 15N TOCSY-HSQC, and 1H, 15N NOE-HSQC data obtained in 90% H2O/10% D2O on uniformly 15N-labeled samples. The assignment was initiated by identifying unique spin systems (e.g. serine, threonine, alanine, glycine, and others) in the three-dimensional TOCSY data. Sequential connections between those specific residues or other spin systems (e.g. S-AMX, AMX-G, S-T-G, and S-G) were identified in the heteronuclear NOESY spectrum. Further sequential assignments of backbone amide resonances were tentatively made by additional NOEs extracted from the three-dimensional NOE-HSQC data. We confirmed and extended our assignments using “reverse selective labeling of arginine” (34), in which all residues but arginine are 15N-labeled (Fig. 1D, locations of assigned arginines indicated by open circles).

Based on all NMR data, we were able to determine that the mobile resonances of E. coli SecA correspond to amino acids 564 to 579 (mobile region 1) and 864 to 901 (mobile region 2) (Fig. 1A). The following example illustrates the approach used: The assignment of mobile region 1 was initiated by unique assignment of L759QRLG757. Sequential Hα–Hα, Hα–Hβ, and Hβ–Hβ NOEs were used to define a Glu/Gln/Met-Leu-Ile-Arg sequence. In this case, the third residue was shown to be arginine by reverse labeling; other residue identifications were obtained from 1H, 15N TOCSY-HSQC data. Search of the SecA sequence for a region consistent with these data yields two possible assignments (418MIR420 and 570QLR572). A strong Hα–Hα NOE between the arginine and an i+1 glycine residue uniquely identifies these resonances as the L759QRLG757 sequence. Another set of resonances correlated by Hα–Hα, Hα–Hβ, and Hβ–Hβ sequential NOEs consists of an arginine followed by a Lys/Glu/Gln/Met spin system, which itself has an Hα–Hβ NOE to an i+1 glycine residue. Only two regions of the SecA sequence fit these criteria (757RQQ759 and 768RQG786). Here and throughout this assignment procedure, we have made the plausible assumption that short segments of mobile residues separated by a short unassignable sequence in the context of the 901-amino acid dimer arise from a single, larger mobile region.

For example, we found no other mobile residues in the vicinity of 786RQQ788 and hence assigned this RQQ sequence to residues 577–579. Similar logic allowed us to tentatively assign a Ser-Arg pair and an Arg-Ile-Leu pair to the 584SSR587 sequence. Thus we conclude that one mobile region comprises residues Ser564 to Glu579 (mobile region 1).

In an analogous fashion, we assigned mobile region 2 to the extreme C terminus of the SecA protein (residues 864 to 901). Assigned sequences in this region included 864SA65, 857LAAGTGER877, 879VGRN882, 888GSGKKYKQCHGRLQ901. We argue that this is one consecutive mobile region based on similar assumptions to those used for mobile region 1.

Ligand-binding Effects on the SecA Mobile Regions Suggest Functional Roles—As shown in Fig. 2, the HSQC spectrum of SecA upon addition of either Mg2+ alone or MgATP-γS reveals significant chemical shift perturbation (both proton and nitrogen dimensions) to selected resonances, notably Arg566 and Arg572 in mobile region 1. Mg2+ is reported to bind to SecA directly, with its proposed site of binding involving Asp209 and Asp217 (E. coli numbering) (35). The former residue is part of the DEAD-box sequence of SecA (helicase motif II), 209DEVD212, the last residue of which interacts directly with...
Gln570 of helicase motif VI (and mobile region I) in the structures of both SecA (21) and UvrB (36, 37). Asp217 directly interacts with Arg566 of mobile region 1. The involvement of helicase motif VI in forming the nucleotide-binding site is consistent with the sensitivity of Gln570, Arg572, and Arg577 to addition of ATP-γS. Interestingly, in addition, significant chemical shift changes are observed for Arg566 in mobile region 1 when ATP-γS binds, even though this residue does not directly contact the nucleotide, and for Lys894 and Lys891 in mobile region 2, the latter upon binding of ATP-γS but not Mg2⁺.

Further insight into the spatial arrangement of the mobile regions was obtained through the use of Mn2⁺ ion as a paramagnetic broadening agent. Nuclei within 20 Å of the site of Mn2⁺ binding may be perturbed, and the effect is distance-dependent by an inverse third power (38–41). Mn2⁺ is nearly isosteric to Mg2⁺, thus its addition to SecA should broaden the signals of residues that are close in space to the Mg2⁺-binding site. Strikingly, addition of 2.0 mM Mn2⁺ causes significant line broadening and attenuation of cross peaks arising from both mobile regions of SecA (Fig. 3). In mobile region 2, the broadening was most pronounced for central residues (874–881), whereas residues throughout mobile region 1 were affected. The observation that Mn2⁺ addition differentially broadens signals from the SecA mobile regions argues that its influence arises from specific binding and not a nonspecific process (such as aggregation). Additionally, the signal from an internal standard (TMSP) was not broadened (data not shown), supporting the interpretation that the observed changes arise from specific binding.

Because Mn2⁺ functionally substitutes for Mg2⁺ in NTP binding to most ATPases and GTPases (42, 43), we could explore nucleotide-induced domain rearrangements of SecA with the added Mn2⁺ reporting on the location of mobile regions 1 and 2 with respect to the nucleotide-binding site. Surprisingly, many (but not all) 1H-15N cross peaks in the HSQC spectrum arising from the mobile regions were restored to nearly their original intensity when AMP-PNP was added (Fig. 3C), arguing that AMP-PNP reversed the influence of Mn2⁺. As indicated above, the observation that AMP-PNP restored the intensities of some, but not all, broadened signals points to a specific effect of this ligand.

**DISCUSSION**

Our results reveal that there are two well-defined regions of high mobility in SecA, such that narrow NMR resonances are observable despite the large size of this protein. Moreover, the locations of these regions are functionally provocative. Thus, SecA joins several multidomain proteins that have been found to have high mobility in functionally important regions (28, 44–46).
Mobile region 1 of SecA spans amino acids 564 to 579 and contains the conserved helicase motif VI (570–577) (23). This region has direct contact with bound nucleotide in several helicases (Ref. 22 and references within), and the recent crystal structure of \textit{B. subtilis} SecA confirms that the same role is played in SecA (21). Alignment of 30 different SecA sequences shows it to be one of the most conserved sequences in SecA (near 100\%) (21). Several mutations that are within or near mobile region 1 cause altered SecA ATPase activity (16, 20).

The second mobile segment (mobile region 2) consists of the extreme 43 C-terminal residues of SecA, a region that has been shown to be important for several functions: translocation activity \textit{in vitro}, lipid binding, SecB binding, and Zn\(^{2+}\) binding (8, 30, 47–51). Woodbury et al. (26) have suggested that this C-terminal region is not only a SecC-binding site but that the interaction between SecB and the C terminus of SecA is also crucial to trigger the conversion from the inactive to the active form of the SecA/SecB complex, promoting exchange with membrane-bound SecA. In addition, Kendall and coworkers (52) have recently shown that SecB binding induces a conformational change in SecA, directly enhancing the ATPase activity of SecA in a concentration-dependent and saturable manner.

The ability to monitor the two mobile regions of SecA provides windows into ligand modulation of SecA conformational equilibria. The chemical shift change of Arg\(^{566}\) upon Mg\(^{2+}\) binding (Fig. 2) most likely reports a remodeling of the NBF-I/NBF-II interface due to the direct complexation of the cation to Asp\(^{569}\) and Asp\(^{572}\), the latter of which interacts with Arg\(^{566}\). This is a novel interaction not present in other helicases (22), and it will be of interest to examine further its relationship to the function of SecA. The more widespread perturbation from binding of ATP-\(\gamma\)S likely arises from conformational rearrangements, whereas the more localized changes upon Mg binding may be because of direct effects on groups that bind the ion.

Substitution of Mn\(^{2+}\) for Mg\(^{2+}\) added new insights into the spatial arrangement of the mobile regions. Specifically, the broadening of resonances arising from both mobile segments upon addition of Mn\(^{2+}\) indicates that both mobile regions 1 and 2 are close to the Mg\(^{2+}\)-binding site, which is proposed to involve residues Asp\(^{569}\) and Asp\(^{572}\) (35). The proximity of mobile region 1 to these residues is not surprising, given the observed direct interactions between these segments in the structure (21), but these data provide the first direct evidence that the extreme C-terminal region of SecA (unresolvable in the crystal structure) approaches closely to the interface between NBF-I and -II. The observation that addition of AMP-PNP led to restoration of intensity of several resonances from both mobile regions 1 and 2 was striking (Fig. 3) and points to movements of major portions of both mobile regions away from the nucleotide-binding pocket of NBF-I due to domain rearrangements upon binding to AMP-PNP. AMP-PNP was earlier reported to favor the more open conformation of SecA, in contrast to ATP-\(\gamma\)S or ADP, which in turn stabilize its soluble, compact conformation (53). Consistent with this proposal, neither ATP-\(\gamma\)S nor ADP led to the restoration of signal intensities. We conclude that AMP-PNP influences the conformational equilibrium of SecA in such a way that the distance between mobile regions and the nucleotide-binding pocket is increased; most strikingly, the C-terminal region encompassing mobile region 2 is close to the interface of NBF-I and -II in the more compact conformation of SecA and moves away from this interface in the presence of AMP-PNP, which favors its more open conformation.

Several independent lines of evidence suggest that the two mobile regions identified by NMR are in dynamic conformational equilibrium. Whereas helicase motif VI is a well structured α-helix and a loop in the crystal structure of \textit{B. subtilis} SecA (21), the same region is disordered in the crystal structure of yeast initiation factor 4A, a DEAD-box RNA helicase whose NBF-I domain is a close structural homologue to that of SecA (54). This comparison suggests that mobile region 1 has the capacity to adopt alternate conformations. Additionally, IRA1 was proposed to be flexible and to undergo changes in its α-helical content with increases in temperature (16). The behavior of IRA1 and its proximity in sequence to mobile region 2 suggests a dynamic equilibrium for the C-terminal segment of SecA. The final 40 residues at the extreme C terminus of \textit{B. subtilis} SecA, corresponding to mobile region 2 in our studies of \textit{E. coli} SecA, are unresolved, consistent with high mobility (21). Limited proteolysis of SecA favors clip sites within the two mobile regions identified by NMR (10, 49, 55, 56). Previous biochemical data from several laboratories, including our own, have shown that any disruption of the C terminus of SecA, through truncation or partial unfolding, leads to enhanced ATPase activity (10, 13, 14). As noted above, deletion of the IRA1 region of the C-terminal sequence (residues 783 to 795) recapitulates the ATPase activation observed in C-terminally truncated versions of SecA (15). It is noteworthy that both mobile regions display narrow NMR resonances, even in the presence of nucleotides. One might expect a reduction in mobility and consequent line broadening, particularly for residues in mobile region 1 that are involved in nucleotide binding. The most likely explanation is the existence of a dynamic equilibrium between domain-dissociated, more extended SecA confor-
mations and more compact forms with tighter domain interac-
tions, as previously proposed (53). So long as there is a
population of molecules with high mobility in localized regions
and these interconvert with molecules in which these regions
are less mobile, we would expect to see some narrow reso-
nances. All of these results are consistent with a model in
which the C-terminal segment of SecA visits alternate con-
formations, either directly associated with the interdomain in-
terface between NBF-I and -II, where it modulates nucleotide
binding/release/hydrolysis, or freely accessible to external part-
ners such as SecB.

Our NMR results support a model in which the two mobile
regions spend time in proximity to one another (and to the site
of Mn\(^{2+}\) binding) and in which ligand binding (specifically
AMP-PNP, presumably mimicking ATP) shifts the SecA con-
formation such that the mobile regions are no longer proximal
to the nucleotide-binding site. This conclusion is consist-
Functionally Important Mobile Regions of SecA

42. Wittinghofer, A., and Pai, E. F. (1991) Trends Biochem. Sci. 16, 382–387
43. Schweins, T., Scheffzek, K., Assheuer, R., and Wittinghofer, A. (1997) J. Mol. Biol. 266, 847–856
44. Jasani, F., Park, S. J., and Wiley, D. C. (1995) Proc. Natl. Acad. Sci. U. S. A. 92, 9900–9904
45. Liang, H., Petros, A. M., Meadows, R. P., Yoon, H. S., Egan, D. A., Walter, K., Holzman, T. F., Robins, T., and Fesik, S. W. (1996) Biochemistry 35, 2095–2103
46. Volkert, T. L., Baleja, J. D., and Kumamoto, C. A. (1999) Biochem. Biophys. Res. Commun. 264, 949–954
47. Hartl, F. U., Lecker, S., Schiebel, E., Hendrick, J. P., and Wickner, W. (1990) Cell 63, 269–279
48. Breukink, E., Keller, R. C. A., and de Kruijff, B. (1993) FEBS Lett. 331, 19–24
49. Fekkes, P., de Wit, J. G., Boersma, A., Friesen, R. H., and Driessen, A. J. (1999) Biochemistry 38, 5111–5116
50. Fekkes, P., van der Does, C., and Driessen, A. J. (1997) EMBO J. 16, 6105–6113
51. Hirano, M., Matsuyama, S., and Tokuda, H. (1996) Biochem. Biophys. Res. Commun. 229, 90–95
52. Kim, J., Miller, A., Wang, L., Muller, J. P., and Kendall, D. A. (2001) Biochemistry 40, 3674–3680
53. den Blaauwen, T., Fekkes, P., de Wit, J. G., Kuiper, W., and Driessen, A. J. M. (1996) Biochemistry 35, 11994–12004
54. Caruthers, J. M., Johnson, E. R., and McKay, D. B. (2000) Proc. Natl. Acad. Sci. U. S. A. 97, 13080–13085
55. Chen, X., Brown, T., and Tai, P. C. (1998) J. Bacteriol. 180, 527–537
56. Shinkai, A., Mei, L. H., Tokuda, H., and Mizushima, S. (1991) J. Biol. Chem. 266, 5827–5833
57. von Heijne, G. (1990) J. Mol. Biol. 184, 99–105
58. Emr, S. D., and Silhavy, T. J. (1983) Proc. Natl. Acad. Sci. U. S. A. 80, 4599–4603
59. Briggs, M. S., and Gierasch, L. M. (1984) Biochemistry 23, 3111–3114
60. McKnight, C. J., Briggs, M. S., and Gierasch, L. M. (1989) J. Biol. Chem. 264, 17293–17297
61. Bruch, M. D., McKnight, C. J., and Gierasch, L. M. (1989) Biochemistry 28, 8554–8561
62. Bruch, M. D., and Gierasch, L. M. (1990) J. Biol. Chem. 265, 3851–3858
Functionally Significant Mobile Regions of *Escherichia coli* SecA ATPase Identified by NMR
Yi-Te Chou, Joanna F. Swain and Lila M. Gierasch

*J. Biol. Chem.* 2002, 277:50985-50990.  
doi: 10.1074/jbc.M209237200 originally published online October 22, 2002

Access the most updated version of this article at doi: 10.1074/jbc.M209237200

Alerts:
- When this article is cited
- When a correction for this article is posted

Click here to choose from all of JBC’s e-mail alerts

This article cites 58 references, 26 of which can be accessed free at http://www.jbc.org/content/277/52/50985.full.html#ref-list-1