The membrane insertion of the Sec-independent M13 Procoat protein in bacteria requires the membrane electrochemical potential and the integral membrane protein YidC. We show here that YidC is involved in the translocation but not in the targeting of the Procoat protein, because we found the protein was partitioned into the membrane in the absence of YidC. YidC can function also to promote membrane insertion of Procoat mutants that insert independently of the membrane potential, proving that the effect of YidC depletion is not due to a dissipation of the membrane potential. We also found that YidC is absolutely required for Sec-dependent translocation of a long periplasmic loop of a mutant Procoat in which the periplasmic region has been extended from 20 to 194 residues. Furthermore, when Sec-dependent membrane proteins with large periplasmic domains were overproduced under YidC-limited conditions, we found that the exported proteins pro-OmpA and pre-peptidoglycan-associated lipoprotein accumulated in the cytoplasm. This suggests for Sec-dependent proteins that YidC functions at a late stage in membrane insertion, after the Sec translocase interacts with the translocating membrane protein. These studies are consistent with the understanding that YidC cooperates with the Sec translocase for membrane translocation and that YidC is required for clearing the protein-conducting channel. 

In bacteria, secreted and outer membrane proteins are exported across the inner membrane using a Sec translocase composed of the proteins SecA, SecY, SecE, SecG, SecD, SecF, and YajC (reviewed in Ref. 1). The minimal components for translocation are SecA, which uses the energy of ATP hydrolysis to push the preprotein across the membrane (2), and SecYE (3), two multispanning membrane proteins comprising the protein-conducting channel (4). SecG, SecD, SecF, and YajC enhance translocation efficiency, although they are not absolutely required for the translocation process (5).

In addition, the majority of membrane proteins use the Sec translocase for membrane insertion. The membrane insertion of leader peptidase (6, 7), MalF (8), mannitol permease (9), ProW (10), SecY (11), FtsQ (12), and AcrB (13) is affected in conditional-lethal sec mutants. Cross-linking studies implicated that membrane proteins such as FtsQ and leader peptidase come into contact with SecA and SecY during the insertion process (12, 14).

Several membrane proteins have been shown to insert into the membrane directly by a Sec-independent mechanism. These include the M13 Procoat (6, 7, 20) and the Pf3 coat protein (21) as well as the polytopic membrane protein melibiose permease (22). Interestingly, some membrane proteins, such as the M13 Procoat (7) do not require the Ffh and FtsY targeting system for insertion.

Recently, a new protein component called YidC has been identified to play a key role in the membrane insertion of bacterial proteins (23). YidC is the bacterial homologue of mitochondrial Oxa1p, shown to mediate insertion of proteins from the mitochondrial matrix into the inner membrane (24, 25). Using a YidC depletion strain, YidC was shown to be absolutely essential for the insertion of the M13 Procoat protein into the Escherichia coli inner membrane (23). YidC appeared less important for insertion of proteins that require the Sec translocase, although it was needed for efficient transfer. During membrane insertion YidC makes contact with membrane-spanning regions of nascent chains of FtsQ (12) and leader peptidase (14, 23). This implies that YidC is closely associated with the Sec translocase. Indeed, it was found that some YidC copurifies with the Sec translocase (12).

Here, we have examined in more detail the role of YidC in the membrane biogenesis of the M13 Procoat protein. In cells where YidC was depleted, the localization of Procoat was analyzed by fractionation and protease mapping techniques. We tested whether a Procoat mutant that inserts in the absence of an electrochemical membrane potential (26) requires YidC. Procoat mutants that utilize a Sec-dependent insertion pathway were also investigated. We show that Procoat can partition into the membrane even in the absence of YidC, demonstrating that YidC is not a membrane receptor. We also show that Procoat mutants require YidC even if they insert in a potential independent or in a Sec-dependent manner. Interestingly, we...
observed an interfering effect on OmpA and PAL, export when YidC is depleted and when a Sec-dependent Procoat protein with a long periplasmic loop is overexpressed, consistent with the idea that the absence of YidC causes jamming within the Sec translocone.

**EXPERIMENTAL PROCEDURES**

**Strains, Plasmids, and Materials**—The *E. coli* JS7131 YidC depletion strain is from our laboratory collection (23). Construction of the Procoat-Lep fusion (27) and several derivatives thereof have been described (26, 28, 29). Procoat and Procoat-Lep proteins were expressed from the plasmid pMS119, which contains the IPTG-inducible tac promoter and the lacIq gene. Amino acids, lysosome, and azide were from Sigma. Tran35S-label, a mixture of 85% [35S]methionine and 15% [35S]cysteine, 1000 Ci/mmol, was from ICN.

**Assay for Membrane Insertion by Signal Peptide Processing**—JS7131, the YidC depletion strain, bearing plasmids encoding Procoat or Procoat-Lep derivatives, was grown in LB medium supplemented with 0.2% glucose or 0.2% arabinose. To deplete cells of YidC, cells were grown for 3 h in glucose. Prior to labeling, the cells were pelleted and resuspended in prewarmed M9 medium containing glucose or arabinose. After 30 min, Procoat and Procoat-Lep derivatives were induced with 1 mM IPTG for 5 min. Cells were labeled with trans-35S]methionine for 20 s and chased for various times. Samples were immunoprecipitated with antiserum to Procoat, leader peptidase (which recognizes Procoat-Lep), OmpA, or PAL and analyzed by SDS-PAGE and fluorography (30).

**Cell Fractionation**—Cell fractionation experiments were performed with sodium carbonate to determine whether Procoat H5 or the Procoat-Lep protein can partition into the membrane when YidC is absent. Cultures (1 ml) of JS7131 bearing Procoat or Procoat-Lep were grown in LB media supplemented with glucose or arabinose and then switched to M9 media and prepared for labeling as described in the signal peptide processing assay. IPTG (1 mM) was added for 5 min prior to labeling to induce Procoat or Procoat-Lep. Unless otherwise stated, cells were pulse-labeled for 30 s with 100 μCi of [35S]methionine and chased with nonradioactive methionine for 20 s and chased for various times. Samples were chilled on ice and centrifuged, and the pellet was resuspended in 300 μl of spheroplast buffer (33 mM Tris, pH 8.0, 40% sucrose). Lysozyme (5 μg/ml, final concentration) and EDTA (1 mM, final concentration) were added for 15 min. Then 400 μl of water and 700 μl of sodium carbonate (pH 11.5, 0.2 M final concentration) were added, and the samples were vortexed vigorously. After lysis, the sample was centrifuged (110,000 × g) for 30 min at 4 °C to pellet the membrane fraction. The supernatant and membrane fractions were then precipitated with trichloroacetic acid, analyzed by immunoprecipitation using antisera against Procoat, leader peptidase (which precipitates Procoat-Lep), and GroEL/Band X (a cytosolic control), and analyzed by SDS-PAGE and phosphorimaging.

**RESULTS**

**Procoat Partitions into the Membrane in the Absence of YidC**—Previously, we have shown that the M13 Procoat protein cannot insert across the membrane to expose the central loop into the periplasmic space when YidC is depleted from the cell (23). This suggests that M13 Procoat protein is blocked either for partitioning or for the translocation step in the membrane insertion pathway. To investigate in which step yidC is involved, we examined whether the Procoat mutant H5, which spans the membrane twice (see Fig. 1; H5 has the same topology as uncleaved Procoat), can integrate into the membrane of JS7131 cells when YidC is depleted. The mutant H5 has an amino acid substitution in the leader sequence at position −3 from Ser to Phe, which prevents cleavage by leader peptidase (31). Therefore, the Procoat protein remains in the uncleaved form independently of the presence or absence of YidC. To analyze whether Procoat H5 is in the membrane or cytoplasmic fraction, JS7131 cells were pulse-labeled for 1 min with [35S]methionine and chased with nonradioactive methionine (Fig. 2A). Samples were treated with sodium carbonate, and the membrane was pelleted. When cells were grown with arabinose to express YidC, nearly all of the Procoat was found in the pellet fraction after carbonate extraction. When the cells were grown under YidC deficient conditions (−glucose medium), the majority of the Procoat protein was also in the pellet. This is in contrast to procoat mutants that do not bind to the membrane surface which accumulate in the soluble fraction (32). As a control, we show that the cytoplasmic protein GroEL was found mainly in the supernatant. Similarly, the wild-type Procoat protein that accumulated under YidC-depleted conditions fractionated in the membrane pellet (data not shown). In addition, a Procoat mutant protein, Procoat-lep (PCLep, Fig. 1), in which the cytoplasmic region is extended by 103 amino acids of the P2 domain of leader peptidase (27), was analyzed for partitioning into the membrane (Fig. 2B). Under YidC-depleted conditions the precursor of Procoat-lep accumulated and was found in about equal amounts in the supernatant and pellet. This shows that Procoat-lep can partition into the membrane, although the P2 domain increases the solubility of the protein. In addition, Band X, a cytosolic protein, was found in the supernatant fraction. Taken together, this clearly indicates that the targeting of Procoat to the membrane is not affected substantially by the absence of YidC.

**YidC Functions to Promote Membrane Insertion of Both Membrane Electrochemical Potential-dependent and -independent Procoat Proteins**—In mitochondria, most of the proteins that are inserted into the inner membrane in an Oxa1p-dependent manner require the membrane electrochemical potential (33). Therefore, we investigated whether YidC, the bacterial Oxa1p homologue, can promote insertion of Procoat proteins that insert independently of the membrane potential. Mutants of Procoat-Lep have been studied extensively to examine the importance of the membrane electrochemical potential in membrane insertion (26). The extension of the Procoat
YidC Is Absolutely Required for Procoat Proteins, Which Are Sec-dependent—We investigated the YidC requirement for a Sec-dependent Procoat mutant, termed Procoat-828 (Fig. 1), in which the periplasmic loop was lengthened by a 174-amino acid OmpA fragment (20). Previously we found that YidC is absolutely required only for Sec-independent membrane proteins, whereas Sec-dependent proteins like leader peptidase or ProW are much less affected by YidC depletion (23). To our surprise, Procoat-828 was substantially affected for the translocation of the large central loop across the membrane into the periplasm. Fig. 4A shows that Procoat-828 membrane insertion is severely inhibited under YidC-depleted conditions, but Procoat-828 is translocated normally when ample YidC is present. To determine whether Procoat-828 requires YidC because of the presence of the membrane anchor region, we constructed Procoat-828ΔH2 with the membrane anchor deleted. Fig. 4B shows that this mutant was equally affected by YidC depletion. For both mutants Procoat-828 and Procoat-828ΔH2, we observed that the export of pro-OmpA across the membrane was hindered when YidC was depleted.

Previous studies showed that the signal peptide processing of several Procoat mutants (for example, -3MPCLep and -5PCLep) was slightly inhibited by sodium azide (26). Azide has been shown to be an inhibitor of the SecA activity (34). The results suggested that these mutants require SecA function for optimal insertion. Cells bearing -3MPCLep were pulse-labeled...
for 20 s and chased for the indicated times in media containing glucose or arabinose (Fig. 4C). The mutant protein -H110023MPCLep was rapidly inserted across the membrane and processed by leader peptidase when expressed in the presence of ample YidC. However, the membrane insertion of -H110023MPCLep was completely blocked when expressed in cells with deficient levels of YidC. Similar results were found with the -H110025PCLep mutant (Fig. 4D). In contrast to the results with overexpression of Procoat-828, there was no significant effect on OmpA export when the -H110023MPCLep and -H110025PCLep mutants were overexpressed in YidC-depleted cells.

Jamming of the Translocase by Overproduction of a Sec-dependent Membrane Protein under YidC-depleted Conditions—A delayed processing of pro-OmpA was observed after overexpression of Procoat-828 (Fig. 4) and was also observed in a YidC-depleted strain when the Sec-dependent ProW-Lep or leader peptidase was overexpressed (23). This indirect effect on OmpA export is probably due to jamming of the Sec translocase caused by increased traffic of a Sec-dependent membrane protein that becomes stalled in the absence of YidC. To explore this jamming phenomenon further, we investigated whether it was linked to the rate of expression. Two cultures of JS7131 carrying pMS119-lep were grown under conditions to deplete YidC. Only the culture induced with IPTG for 10 min to overexpress leader peptidase displayed an effect on OmpA export (Fig. 5A). Interestingly, when we used another lot of OmpA antisera, we observed that the overexpression of leader peptidase (+IPTG) also caused delayed processing of pre-peptidoglycan-associated lipoprotein (PAL) (Fig. 5B). The identity of PAL was verified by immunoprecipitation using antibodies specific to PAL (Fig. 5C). PAL, an outer membrane protein (35), translocates across the inner membrane in a Sec-dependent fashion, because its export is strongly inhibited by 2 mM azide, an inhibitor of SecA (Fig. 5C).

To show that the jamming effect within the Sec translocase is specific to the overexpression of a membrane protein that depends on YidC, we tested whether expression of maltose binding protein (MBP) affects export of OmpA. Two cultures of JS7131 bearing the plasmid encoding preMBP were grown under conditions to deplete YidC. When IPTG was added to overexpress the YidC-independent periplasmic protein MBP, no accumulation of pro-OmpA was observed as compared with uninduced cells (Fig. 5D). Accumulation of preproteins was observed only when Procoat-828 or Lep was overexpressed. Therefore, the block in OmpA export is specific to overexpression of a Sec-dependent membrane protein with a large
periplasmic domain. The absence of YidC thus causes a secondary effect on Sec-mediated protein export.

**DISCUSSION**

In mitochondria, Oxa1p, the YidC homologue, plays a key role in the insertion of proteins from the matrix into the membrane (24, 25, 36). Most of the substrate proteins that use Oxa1p require the electrochemical membrane potential for insertion (33). Because mitochondria lack Sec homologues (37), the Oxa1 pathway may correspond to the Sec-independent pathway in bacteria (38), which is distinct from the bacterial prepore translocase pathway (39). Recently, the bacterial homologue of Oxa1p, YidC, has been discovered to be absolutely essential for the membrane insertion of the Sec-independent M13 Procoat protein. Similar to the mitochondrial membrane proteins, the membrane insertion of the Procoat protein requires the electrochemical membrane potential (23).

In this report, we have tested whether the YidC pathway functions only with substrates that require the membrane electrochemical potential for insertion. To carry out this test, we examined two Procoat mutants, which previously have been shown to insert independent of the electrochemical membrane potential (26, 29). We found that membrane insertion of both mutants is YidC-dependent (Fig. 3, A and B); this demonstrates that the effect of YidC depletion on the membrane insertion of Procoat is not because of dissipation of the membrane potential. A possible role of YidC for the membrane insertion of Procoat is that it may be necessary for hydrophobic partitioning of Procoat into the membrane or for the translocation event across the membrane. In the absence of YidC, we found that a significant amount of the Procoat protein and also Procoat-Lep fractionated with the membrane under alkaline conditions (Fig. 2), suggesting that YidC is not involved in the targeting and partitioning steps of the Procoat protein but rather in the translocation event across the membrane. This translocation step is known to depend on the electrochemical membrane potential (40). In carbonyl cyanide m-chlorophenyl hydrazone (CCCP)-treated cells, the Procoat protein partitions into the membrane but is not translocated (32). Procoat mutants that have a zero net charge within the periplasmic residues were found to insert independently of the membrane potential (26). These Procoat mutants still require YidC for the translocation step. Therefore, the molecular mechanism of how YidC supports membrane translocation is not linked to the action of the membrane potential.

We also show here that YidC is absolutely required for the insertion of the Sec-dependent mutant Procoat-828 (Fig. 4A). This protein contains a 194-amino acid residue periplasmic loop that is followed by the membrane anchor. These results confirm that YidC plays a key role not only for Sec-independent proteins but also for Sec-dependent proteins. It is interesting that YidC can play a more critical role for Procoat-828 than for the Sec-dependent leader peptidase or for ProW-Lep (23). We ruled out the possibility that this difference is because Procoat-828 has a C-terminal membrane anchor, whereas leader peptidase and ProW-Lep have a hydrophilic domain that is released to the periplasm. We found that Procoat-828, even missing the membrane anchor in the mature region of the protein, still inserts in a YidC-dependent manner. This finding was surprising because the protein should have behaved as a secretory protein; previously we have shown that secretory proteins are unaffected by YidC depletion (23). One possible explanation is that the Procoat-828 proteins with or without the membrane anchor is recognized by YidC through the leader sequence; this is being tested currently.

The results presented in this paper are consistent with YidC having two functions. First, YidC functions to insert Sec-independent proteins and in certain cases can also make insertion more efficient for Sec-dependent proteins (Fig. 4B). This may not be a general function, as YidC was not required for export of several preproteins (23). Second, YidC may function to facilitate the movement of a hydrophobic domain of a membrane protein from within the Sec channel into the lipid bilayer. Although proOmpA and pre-PAL are translocated normally by the Sec translocase in the absence of YidC, when a Sec-dependent membrane protein was overexpressed (23) these exported proteins accumulated in the cytoplasm. The inhibition of the Sec translocase may arise because the hydrophobic domains of a Sec-dependent membrane protein cannot efficiently leave the Sec translocase in the absence of YidC and therefore may cause jamming of the translocase resulting in an accumulation of the preprotein in the cytoplasm. This is consistent with recent work described by Luirink and co-workers (41), which demonstrates that there is a sequential interaction of a nascent membrane protein with SecY and YidC. Jamming of the translocase because of YidC depletion may also occur when a hydrophilic domain blocks the Sec channel, as is the case for Procoat-828/H2. Also, for the Sec-dependent Procoat mutants – 3MP-Cleap and – 5PChlep, we observed no jamming. This is possibly because the 20-residue hydrophilic domain is too short to block the Sec channel.

The proposed function that YidC supports the translocation of transmembrane segments into the bilayer is similar to the proposed function of translocating chain-associating membrane protein (TRAM) protein, an integral component of the endoplasmic reticulum membrane (42). TRAM, like YidC, was found to be associated with the transmembrane segment of a protein during its integration into the bilayer (43, 44). Like YidC, it is a multispansing membrane protein that may function to insert hydrophobic regions into the bilayer. The close cooperation of YidC and Sec in bacteria and TRAM and Sec61 in the endoplasmic reticulum would predict cooperation between the Sec components and the YidC homologue Albino3 in chloroplasts. However, recent data have show that antibodies to the stromal domain of Albino3 blocks translocation of LHCP but has no effect on the translocation of Sec-dependent luminal proteins of the thylakoid (45).

In conclusion, we have shown for the first time that YidC is involved directly in the translocation step of an inserting membrane protein and is not involved in targeting of the Sec-independent protein Procoat. Second, we have also ruled out that the observed accumulation of Procoat after YidC depletion is simply a result of the loss of the membrane electrochemical potential. For example, YidC is also required for the insertion of two Procoat mutants that do not require the membrane potential for insertion. Third, we find that YidC plays an essential role for the insertion of a Sec-dependent Procoat mutant in which the periplasmic domain is lengthened. Fourth, we find that overproduction of Sec-dependent membrane proteins in the absence of YidC leads to jamming of the Sec translocase, which has an inhibitory effect on protein export.

**Acknowledgments**—We thank Judith Hellman for providing antibody against PAL and a plasmid overproducing PAL.

**REFERENCES**

1. Driessen, A. J., Manting, E. H., and van der Does, C. (2001) Nat. Struct. Biol. 8, 492–498
2. Economou, A., and Wickner, W. (1994) Cell 78, 835–843
3. Brundage, L., Hendrick, J. P., Schiebel, E., Driessen, A. J., and Wickner, W. (1999) Cell 92, 649–657
4. Meyer, T. H., Menetret, J. F., Breiting, R., Miller, K. R., Akey, C. W., and Rapoport, T. A. (1999) J. Mol. Biol. 285, 1789–1800
5. Duong, F., and Wickner, W. (1997) EMBO J. 16, 4871–4879
6. Wolfe, P. B., Rice, M., and Wickner, W. (1985) J. Biol. Chem. 260, 1836–1841
7. de Gier, J. W., Scotti, P. A., Saaf, A., Valent, Q. A., Kuhn, A., Luirink, J., and Rapoport, T. A. (1999) J. Mol. Biol. 285, 1789–1800
8. Traxler, B., and Murphy, C. (1996) J. Biol. Chem. 271, 12094–12400
9. Beck, K., Wu, L. F., Brunner, J., and Muller, M. (2000) EMBO J. 19, 134–143
10. Cristobal, S., Scotti, P., Luirink, J., von Heijne, G., and de Gier, J. W. (1999) J. Biol. Chem. 274, 29068–29070
11. Koch, H. G., Hengelage, T., Neumann-Haefelin, C., MacFarlane, J., Hoffschlue, H. K., Schimz, K. L., Mechler, B., and Muller, M. (1999) Mol. Biol. Cell 10, 2163–2173
12. Scotti, P. A., Urbanus, M. L., Brunner, J., de Gier, J. W., von Heijne, G., van der Does, C., Driessen, A. J., Oudega, B., and Luirink, J. (2000) EMBO J. 19, 542–549
13. Qi, H. Y., and Bernstein, H. D. (1999) J. Biol. Chem. 274, 8993–8997
14. Houben, E. N., Scotti, P. A., Valent, Q. A., Brunner, J., de Gier, J. L., Oudega, B., and Luirink, J. (2000) FEBS Lett. 476, 229–233
15. Neumann-Haefelin, C., Schafer, U., Muller, M., and Koch, H. G. (2000) EMBO J. 19, 6419–6426
16. de Gier, J. W., Mansournia, P., Valent, Q. A., Phillips, G. J., Luirink, J., and von Heijne, G. (1996) FEBS Lett. 389, 307–309
17. Ulbrandt, N. D., Newitt, J. A., and Bernstein, H. D. (1997) Cell 88, 187–196
18. MacFarlane, J., and Muller, M. (1995) Eur. J. Biochem. 233, 766–771
19. Seluanov, A., and Bibi, E. (1997) J. Biol. Chem. 272, 2053–2055
20. Kuhn, A. (1988) Eur. J. Biochem. 177, 267–271
21. Rohrer, J., and Kuhn, A. (1990) Science 250, 1418–1421
22. Bassilana, M., and Gwizdek, C. (1996) EMBO J. 15, 5202–5208
23. Samuelson, J. C., Chen, M., Jiang, F., Muller, I., Wiedmann, M., Kuhn, A., Phillips, G. J., and Dalbey, R. E. (2000) Nature 406, 637–641
24. Hell, K., Herrmann, J., Pratje, E., Neupert, W., and Stuart, R. A. (1997) FEBS Lett. 418, 367–370
25. Hell, K., Herrmann, J. M., Pratje, E., Neupert, W., and Stuart, R. A. (1998) Proc. Natl. Acad. Sci. U. S. A. 95, 2250–2255
26. Cao, G., Kuhn, A., and Dalbey, R. E. (1995) EMBO J. 14, 866–875
27. Kuhn, A., Wickner, W., and Kreil, G. (1986) Nature 322, 335–339
28. Kuhn, A., Zhu, H. Y., and Dalbey, R. E. (1999) EMBO J. 9, 2385–2388, 2429; Correction (1990) EMBO J. 9, 3413
29. Schuenemann, T. A., Delgado-Nixon, V. M., and Dalbey, R. E. (1999) J. Biol. Chem. 274, 6855–6864
30. Ito, K., Date, T., and Wickner, W. (1980) J. Biol. Chem. 255, 2123–2130
31. Kuhn, A., and Wickner, W. (1985) J. Biol. Chem. 260, 15914–15918
32. Gallusser, A., and Kuhn, A. (1996) EMBO J. 19, 2723–2729
33. Herrmann, J. M., Neupert, W., and Stuart, R. A. (1997) EMBO J. 16, 2217–2226
34. Oliver, D. B., Cabelli, R. J., Dolan, K. M., and Jarosik, G. P. (1990) Proc. Natl. Acad. Sci. U. S. A. 87, 8227–8231
35. Hellman, J., Loiselle, P. M., Tehan, M. M., Allaire, J. E., Boyle, L. A., Kurnick, J. T., Andrews, D. M., Sik Kim, K., and Warren, H. S. (2000) Infect. Immun. 68, 2566–2572
36. Hell, K., Neupert, W., and Stuart, R. A. (2001) EMBO J. 20, 1281–1288
37. Glick, B. S., and Von Heijne, G. (1996) Protein Sci. 5, 2651–2652
38. Dalbey, R. E., and Kuhn, A. (2000) Annu. Rev. Cell Dev. Biol. 16, 51–87
39. Wickner, W., and Leonard, M. R. (1996) J. Biol. Chem. 271, 29514–29516
40. Date, T., Goodman, J. M., and Wickner, W. T. (1980) Proc. Natl. Acad. Sci. U. S. A. 77, 4669–4673
41. Urbanus, M. L., Scotti, P. A., Froderberg, L., Saaf, A., de Gier, J. W., Brunner, J., Samuelson, J. C., Dalbey, R. E., Oudega, B., and Luirink, J. (2001) EMBO Rep. 2, 524–529
42. Gorlich, D., Hartmann, E., Prehn, S., and Rapoport, T. A. (1992) Nature 357, 47–52
43. Do, H., Falcone, D., Lin, J., Andrews, D. W., and Johnson, A. E. (1996) Cell 83, 369–378
44. Hegde, R. S., and Lingappa, V. R. (1996) Cell 85, 217–228
45. Moore, M., Harrison, E. C., and Henry, R. (2000) J. Biol. Chem. 275, 1529–1532
Function of YidC for the Insertion of M13 Procoat Protein in Escherichia coli: TRANSLOCATION OF MUTANTS THAT SHOW DIFFERENCES IN THEIR MEMBRANE POTENTIAL DEPENDENCE AND Sec REQUIREMENT
James C. Samuelson, Fenglei Jiang, Liang Yi, Minyong Chen, Jan-Willem de Gier, Andreas Kuhn and Ross E. Dalbey

J. Biol. Chem. 2001, 276:34847-34852.
doi: 10.1074/jbc.M105793200 originally published online July 16, 2001

Access the most updated version of this article at doi: 10.1074/jbc.M105793200

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 45 references, 24 of which can be accessed free at http://www.jbc.org/content/276/37/34847.full.html#ref-list-1