G-protein Palmitoyltransferase Activity Is Enriched in Plasma Membranes*

Jullianne T. Dunphy†, Wendy K. Greentree, Carol L. Manahan§, and Maurine E. Linder¶

From the Department of Cell Biology and Physiology, Washington University School of Medicine, St. Louis, Missouri 63110

Heterotrimeric G proteins are covalently modified by lipids. Myristoylation of G-protein α subunits and prenylation of γ subunits are stable modifications. In contrast, palmitoylation of α subunits is dynamic and thus has the potential for regulating protein function. Indeed, receptor activation of Gs increases palmitate turnover on the α subunit, presumably by stimulating deacylation. The enzymes that catalyze reversible palmitoylation of G-protein α subunits have not been characterized. Here we report the identification of a palmitoyl-CoA:protein S-palmitoyltransferase activity that acylates G-protein α subunits in vitro. Palmitoyltransferase activity is membrane-associated and requires detergent for solubilization. The preferred G-protein substrate for the enzyme activity is the α subunit in the context of the heterotrimer. Both myristoylated and nonmyristoylated G-protein α subunits are recognized as substrates. The palmitoyltransferase activity demonstrates a modest preference for palmitoyl-CoA over other fatty acyl-CoA substrates. Palmitoyltransferase activity is high in plasma membrane and present at low or undetectable levels in Golgi, endoplasmic reticulum, and mitochondria of rat liver. The subcellular localization of this enzyme activity is consistent with a role for regulated cycles of acylation and deacylation accompanying activation of G-protein signal transduction pathways.

Signal-transducing G proteins are located on the cytoplasmic surface of the plasma membrane where they couple receptors to intracellular effectors. Membrane association of heterotrimeric G proteins is facilitated by the covalent addition of lipids to G-protein subunit polypeptides (reviewed in Refs. 1 and 2). The carboxyl-terminal cysteine residue is prenylated and methylated on G-protein γ subunits. Prenylation is not required for βγ complex formation, but facilitates subunit and effector interactions. G-protein α subunits are fatty-acylated. Members of the mammalian Gα family (Gαs, Gαo, and Gαz), and transducin α contain amide-linked myristate at the amino terminus. Transducible α (Tα)3 is modified heterogeneously at this site by C14:0, C14:1, C14:2, or C12:0 fatty acids. Myristoylation also facilitates subunit and effector interactions. Thioester-linked palmitate is found on most mammalian G-protein α subunits. Gαs, Gαo, and Gαz, which are not N-myristoylated, are palmitoylated at one or more cysteine residues near the amino terminus. Gαi, Gαo, and Gαz are palmitoylated at a cysteine residue (Cys-3) adjacent to the amino-terminus myristoylated glycine.

Reversible post-translational modification is a well-characterized mechanism for regulating protein activity. Regulatory cycles of acylation and deacylation of G-protein α subunits may fit this paradigm. Dynamic acylation of G proteins has been characterized best for Gαs. Studies of agonist-induced turnover of palmitate on Gαs are consistent with a model where Gαs is deacylated upon activation and dissociation from βγ subunits (3–5). Deacylation may be accompanied by release of Gαs from membranes, suggesting a potential role for this process in desensitization of Gα-coupled pathways (5).

The enzymes responsible for addition and removal of palmitate from G-protein α subunits have not been identified. G-protein α subunits can be deacylated in vitro by a protein palmitoyl thioesterase that has recently been purified (6). However, subsequent cloning of the cDNA encoding protein palmitoyl thioesterase and characterization of the gene product revealed that protein palmitoyl thioesterase is a secreted enzyme (7), and thus is not likely to be a physiological regulator of G-protein palmitoylation. Palmitoyltransferase (PAT) activities have been identified using a number of proteins known to be palmitoylated as substrates, including viral glycoproteins (8), p21ras(9), and p59fyn(10), but it is not known whether the substrate specificity of these activities extends to G-protein α subunits. Purification to homogeneity and molecular cloning of palmitoyltransferase activities have not been achieved to date. Here we report the initial characterization of a palmitoyl-CoA:protein S-palmitoyltransferase activity highly enriched in plasma membranes that acylates both myristoylated and nonmyristoylated G-protein α subunits in vitro.

EXPERIMENTAL PROCEDURES

Synthesis of [3H]Palmitoyl-CoA—[3H]Palmitoyl-CoA was prepared as described (11) using [3H]palmitate (60 Ci/mmol) from American

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¶To whom correspondence should be addressed: Dept. of Cell Biology and Physiology, Washington University School of Medicine, 660 South Euclid Ave., Box 8228, St. Louis, MO 63110. Tel.: 314-362-6040; Fax: 314-362-7463.

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The abbreviations used are: Tα, transducin α subunit; Tγ, transducin βγ subunits; Tγγ, recombinant Gγ; N-myristoyltransferase, myristoyl-CoA-protein N-myristoyltransferase; PAT, palmitoyl-CoA:protein S-palmitoyltransferase; protease inhibitors, 0.1 mM phenylmethylsulfonyl fluoride, 21 μg/ml TPCK (N-tosyl-L-phenylalanine chloromethyl ketone), 21 μg/ml TLCK (N-p-tosyl-L-lysine chloromethyl ketone), 1.8 μg/ml aprotinin, 3.2 μg/ml leupeptin, 3.2 μg/ml benzamidine inhibitor; BCA, bicinchoninic acid; Mes, 2-(4-morpholinol)ethanesulfonic acid; DTT, dithiothreitol; PAGE, polyacrylamide gel electrophoresis; INT, 2-(p-iodophenyl)-1-(p-nitrophenyl)-5-pentanehexoside chloride; GTPγS, guanosine 5′-(α-thio)triphosphate; HPLC, high pressure liquid chromatography; ER, endoplasmic reticulum.
Radiolabeled Chemicals, Inc. or DuPont NEN. [3H]Palmitoyl-CoA was purified by applying the reaction mixture to a C8 reversed phase cartridge (Analytichem) that had been equilibrated in 50 mM ammonium acetate buffer (pH 5.3). The column was washed sequentially with 50 mM ammonium acetate buffer and 20% acetonitrile in 50 mM ammonium acetate buffer. [3H]Palmitoyl-CoA was eluted with 40% acetonitrile in 50 mM ammonium acetate buffer. [3H]Palmitoyl-CoA was resuspended in 50 mM sodium acetate buffer (pH 5.3), adjusted to −50 μM with cold palmitoyl-CoA (50,000–70,000 dpm/μM), and stored in aliquots at −80°C. Recoveries ranged from 50–90% of starting radioactivity.

G-protein Substrates—Myristoylated recombinant G-protein α subunits were purified as described after expression in a Sulfolobus solfataricus N-myristoyltransferase (12). The C3A mutant of Gα1 was constructed using site-directed mutagenesis of the rat Gα1 coding sequence in plasmid pQE60 (Qiagen). The amino-terminal 387 nucleotides of Gα1 were amplified in a polymerase chain reaction using a mutagenic oligonucleotide (5′-GAAATTAACCATGGGCGCCACACTGAGC-3′) as the forward primer and an oligonucleotide carbamoyl-terminal to an internal SadI site as the reverse primer (5′-CGAGCTCCGGGCGTCT-3′). The resulting PCR product was digested with Ncol and SadI and subcloned into the pGEX-2T vector (5′-GGGCCGCGGCGGTCAT-3′) to create a Gα1 expression plasmid. The expression plasmids were transformed into JM109 bacteria. Activity of recombinant G-protein α subunit preparations was assessed by GTP·S binding (14).

Identification of an Enzymatic Activity That Acylates G-protein α Subunits—To develop an assay for palmitoyltransferase (PfAT) activity, a suitable protein substrate was required. Because most G-protein α subunits can be purified easily after expression in E. coli, we chose to use the recombinant α subunit as a substrate in the assay. Gαi, Gαs, and Gαω are palmitoylated at Cys-3, adjacent to the myristoylated glycine (4, 25, 26). Although myristoylation is not required for palmitoylation (27), it appears to increase palmitoylation of Gαi family members in vivo (4, 25). Accordingly, we purified recombinant myristoylated α subunits from E. coli that co-express N-myristoyltransferase with its G-protein substrate (12, 28). This system provided an abundant source of substrate with an appropriately modified amino terminus to use for in vitro acylation.

PAT activity was detected in membranes and detergent extracts. Bovine brain membranes were incubated with myristoylated Gαiα1 (Fig. 1A, lane 1). Gαiα1 was solubilized by detergent (Fig. 1, lane 3). The C3A

To confirm that the radioactivity incorporated into myristoylated Gαiα1 in vitro was palmitate, the protein was excised from the gel and treated with base to cleave thioester bonds. Hydrolysates were resolved by reversed phase HPLC, and fatty acids were identified by co-elution with fatty acid standards. The radioactivity released from myristoylated Gαiα1 was identified as palmitic acid (data not shown). These data also demonstrate that in vitro palmitoylation of the protein is alkanesensitive, consistent with a thioester linkage.
mutant of myristoylated $r_{G_{i1}}$ was not a substrate in the assay (Fig. 1A, lane 4), indicating that in vitro acylation is occurring at the appropriate cysteine residue in the protein.

Experiments were performed to determine if in vitro acylation of $G$-protein substrates was indeed enzymatic. Uncatalyzed acylation of substrate proteins incubated with palmitoyl-CoA in the absence of a source of enzyme was first reported for rhodopsin (29) and myelin proteolipid protein (30). Low levels of palmitate were incorporated into myristoylated $r_{G_{i1}}$ in the absence of enzyme (Fig. 1A, lane 6). Uncatalyzed acylation was more pronounced if detergent was omitted from the assay or when the protein was incubated with higher concentrations of palmitoyl-CoA (data not shown). To confirm that the activity observed in a detergent extract of membranes required a protein component, we treated the extract with trypsin. Protease treatment reduced PAT activity to background levels (Fig. 1B). Soybean trypsin inhibitor abolished the effect of trypsin. PAT was also inactivated by boiling or after treatment with SDS, a denaturing detergent (Fig. 1B). PAT activity is time-dependent; in the presence of membranes, the assay is linear to 10 min (data not shown). The activity demonstrated strict concentration dependence on the source of enzyme, $G$ protein, and palmitoyl-CoA. At high concentrations of palmitoyl-CoA or $G$ protein, the activity was saturable (data not shown). Taken together, these data demonstrate that in vitro acylation of myristoylated $r_{G_{i1}}$ is an enzymatic process.

G-protein Substrate Specificity—To analyze G-protein substrate specificity of PAT, a detergent extract of bovine brain membranes was incubated with various purified preparations of G-protein $\alpha$ subunits in the presence or absence of $\beta Y$ subunits (Fig. 2). Incorporation of $[^3H]$palmitate was analyzed by SDS-PAGE and fluorography (A) or by liquid scintillation counting of protein-bound radioactivity (B). Comparison of panels A and B shows that the results obtained with both methods were essentially the same. The $\alpha$ subunit in the context of the heterotrimer appeared to be the preferred substrate for PAT. The addition of stoichiometric amounts of $\beta Y$ subunits significantly increased the palmitate incorporated into myristoylated $r_{G_{i1}}$ (lanes 3 and 4). PAT activity toward $r_{G_{i1}}$ (lanes 5 and 6) and $r_{G_{i2}}$ (lanes 7 and 8) was barely detectable in the absence of $\beta Y$ subunits. As expected, $T_\alpha$ was not acylated in vitro (lanes 9 and 10). Although $T_\alpha$ contains N-linked fatty acids at its amino terminus, it does not have cysteine residues near the amino terminus and is not palmitoylated in vivo (31, 32).

Myristoylated $r_{G_{i1}}$ was a better substrate than nonmyris-
The effect of myristoylation on PAT activity was also observed when rG_{\alpha2} and myristoylated rG_{\alpha2} were assayed (data not shown). PAT activity is not limited to myristoylated substrates. G_{\alpha1} is not a myristoylated protein, but is palmitoylated at Cys-3 (4). rG_{\alpha1} in the presence of \( \beta\gamma \) subunits was palmitoylated in vitro, although not as efficiently as myristoylated rG_{\alpha2} (Fig. 2B, lanes 7 and 8). rG_{\alpha1} (C3A) was not a substrate for PAT activity in vitro (data not shown).

The substrate specificity of PAT activity in vitro correlates well with our understanding of G-protein palmitoylation in vivo. G_{\alpha1} and G_{\beta\gamma} are substrates for PAT; the site of palmitoylation in vitro and in vivo appears to be Cys-3. Myristoylated G_{\alpha1} is the optimal substrate for palmitoylation in vivo. In mammalian cells, expression of a mutant G_{\alpha1} lacking the myristoylation site (G2A) results in a protein that is almost entirely cytosolic and has undetectable levels of \(^{3}H\)palmitate incorporation. If \( \beta\gamma \) subunits are co-expressed with G_{\alpha1} (G2A), a small fraction of G_{\alpha1}(G2A) is found associated with membranes and a low level of palmitoylation is observed (27). In the in vitro assay, myristoylated \( \alpha \) subunits were better substrates than those lacking myristate. However, a moderate level of acylation of nonmyristoylated G_{\alpha1} was observed in the presence of \( \beta\gamma \) subunits. Thus, these characteristics are similar to what is observed in vivo.

The role of G-protein \( \beta\gamma \) subunits in substrate affinity for PAT may be to provide a mechanism for substrate presentation to PAT. \( \beta\gamma \) may facilitate targeting of the \( \alpha \) subunit to the membrane, allowing it to become acylated by PAT. Although prenylation of G protein \( \gamma \) subunits is required for membrane association of the \( \beta\gamma \) complex (1, 2), it is not required to support in vitro acylation of myristoylated rG_{\alpha1}. Mutation of the prenylated cysteine residue to serine (C68S) in the G_{\alpha1} subunit yields a nonacylated \( \gamma \) that heterodimerizes with the \( \beta1 \) subunit (16). The mutant \( \beta\gamma \) (\( \beta\gamma\)C68S) binds to myristoylated rG_{\alpha2}, forming a heterotrimer (33) that is acylated in vitro with efficiency similar to that of the wild type heterotrimer (data not shown). These data suggest that \( \beta\gamma \) provides more than a hydrophobic anchor to bind to a membrane (or detergent microenvironment) containing PAT. The palmitoylation site is contained within the amino-terminal region of the \( \alpha \) subunit, which is known to directly interact with \( \beta\gamma \) subunits (33–35). Indeed, \( \beta\gamma \) binding changes G_{\alpha1} amino-terminal structure (33), perhaps making it a better substrate for PAT.

Myristoylation may also facilitate access to PAT. Studies with acylated peptides and model membranes have demonstrated that a myristoyl moiety is not sufficient for high affinity interaction of the acylated peptide with phospholipid vesicles (36). However, even a transient interaction of a myristoylated protein with the membrane may allow the protein to be recognized by PAT and become palmitoylated. The dually acylated protein will then have a high affinity for membranes. Most members of the Src family of protein-tyrosine kinases have an amino-terminal motif of a myristoylated glycine followed by a palmitoylated cysteine (37–39). Palmitoylated Src family kinases require prior myristoylation to be palmitoylated both in vivo (39) and in vitro (10). Although we have shown that myristoylation is not an absolute requirement for recognition of the \( \alpha \) subunit by the enzyme, the presence of myristate increases in vitro acylation of G-protein subunits. Further purification and characterization of PAT activity is required to resolve whether the same PAT activity acylates G-protein \( \alpha \) subunits and Src family kinases.

Fatty Acyl-CoA Substrate Specificity—To determine the fatty acyl chain length that PAT prefers as substrate, unlabeled acyl-CoAs of varying chain length and saturation were tested for inhibition of a partially purified preparation of PAT. Unlabeled palmitoyl-CoA competed the best for PAT activity, suggesting that palmitate is the biologically relevant fatty acid for the enzyme (Fig. 3A). C14 and C18 chain length acyl-CoAs competed almost as well as C16. Unsaturated acyl-CoAs were as effective as saturated forms of similar chain length. Inhibition of PAT activity by competing acyl-CoAs was concentration-dependent (Fig. 3B). Although not as potent an inhibitor as C14 and C16 fatty acyl-CoAs, arachidonoyl-CoA does compete with palmitoyl-CoA. Hallak et al. (40) have reported that arachidonate is incorporated into G-protein \( \alpha \) subunits in platelets. Consistent with the competition experiments, \(^{3}H\)myristate and \(^{3}H\)stearate were incorporated into myristoylated rG_{\alpha1} when the corresponding \(^{3}H\)acyl-CoAs were used as fatty acid donors in assays of PAT activity (data not shown). The extent of acylation with myristoyl and stearoyl fatty acids was reduced compared to palmitate. The fatty acyl-CoA substrate specificity...
of the enzyme is similar to that reported by Berthiaume and Resh (10).

**Subcellular Localization of PAT**—Although palmitoylation of proteins occurs both in intracellular compartments and at the plasma membrane (41), dynamic acylation of G-protein \(\alpha\) subunits is likely to occur at the plasma membrane. To determine if PAT activity is present in plasma membranes, subcellular fractions of rat liver were prepared and analyzed. Specific activity was highest in plasma membranes and present at significantly lower levels in Golgi and mitochondria (Fig. 4A). PAT activity in plasma membranes was sensitive to palmitoyl-CoA, assayed by TLC (43), and was not significantly reduced in Golgi, ER, or mitochondrial fractions during the time course of the assay (data not shown).

Studies of receptor-stimulated turnover of palmitate on \(G_{\alpha}\) are consistent with the following model (3–5). In the basal state, the \(\alpha\) subunit is palmitoylated and associated with \(\beta\gamma\) subunits. Upon ligand binding to the receptor, the \(\alpha\) subunit becomes activated and dissociates from \(\beta\gamma\) subunits. The \(\alpha\) subunit in its GTP-bound form is a substrate for a protein palmitoyl thioesterase and becomes deacylated. Deactivation of \(\alpha\) by GTP hydrolysis results in its reassociation with \(\beta\gamma\) subunits and coincides with reacylation of the \(\alpha\) subunit. These studies do not discriminate between palmitoylation occurring before or after inactive \(\alpha\) binds to \(\beta\gamma\) subunits. Because PAT activity prefers the \(\alpha\) subunit in the context of the heterotrimer as a substrate, we suggest that palmitoylation occurs after reassociation of the subunits. Enrichment of PAT activity in plasma membranes suggests that the subcellular localization of this enzyme allows for rapid reacylation of the G protein at the plasma membrane and does not require the G protein to cycle to an intracellular compartment for reacylation.

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**REFERENCES**

1. Wedegaertner, P. B., Wilson, P. T., and Bourne, H. R. (1995) J. Biol. Chem. 270, 503–506
2. Casey, P. (1995) Science 268, 221–225
3. Degtyarev, M. Y., Spiegel, A. M., and Jones, T. L. Z. (1993) J. Biol. Chem. 268, 23769–23772
4. Mumby, S. M., Kleuss, C., and Gilman, A. G. (1994) Proc. Natl. Acad. Sci. U. S. A. 91, 2800–2804
5. Wedegaertner, P. B., and Bourne, H. R. (1994) Cell 77, 1063–1070
6. Camp, L. A., and Hofmann, S. L. (1993) J. Biol. Chem. 268, 22566–22574
7. Camp, L. A., Verkruysse, L. A., Afendis, S. J., Slaughter, C. A., and Hofmann, S. L. (1994) J. Biol. Chem. 269, 23212–23219
8. Schmidt, M. F. G., and Burns, G. R. (1989) Biochem. Soc. Trans. 17, 625–626
9. Gutierrez, L., and Magee, A. I. (1991) Biochim. Biophys. Acta 1078, 147–154
10. Berthiaume, L., and Resh, M. (1995) J. Biol. Chem. 270, 22399–22405
11. Taylor, D., Weber, N., Hogge, L., and Underhill, E. (1990) Anal. Biochem. 184, 311–316
12. Munby, S. M., and Linder, M. E. (1993) Methods Enzymol. 237, 254–268
13. Sanger, F., Nicklen, F., and Coulson, A. (1977) Proc. Natl. Acad. Sci. U. S. A. 74, 5463–5467
14. Linder, M. E., Ewald, D. A., Miller, R. J., and Gilman, A. G. (1990) J. Biol. Chem. 264, 8243–8251
15. Robishaw, J., Smigel, M., and Gilman, A. (1986) J. Biol. Chem. 261, 9587–9590
16. Iniguez-Lluhi, J., Simon, M. I., Robishaw, J. D., and Gilman, A. G. (1992) J. Biol. Chem. 267, 23409–23417
17. Sternweis, P. C. (1986) J. Biol. Chem. 254, 3333–3340
18. Sternweis, P. C., and Robishaw, J. D. (1984) J. Biol. Chem. 259, 13806–13813
19. Brown, R., Jarvis, K., and Hyland, K. (1989) Anal. Biochem. 180, 136–139
20. Fleischer, S., and Kervina, M. (1974) Methods Enzymol. 31, 6–41
21. Blanco, G., Sanchez, G., and Mercer, R. (1995) Biochemistry 34, 9897–9903
22. Aronson, N. Jr., and Touster, O. (1974) Methods Enzymol. 31, 90–102
23. Starr, B., and Madden, E. (1990) Methods Enzymol. 182, 203–235
24. Pennington, R. (1961) Biochem. J. 80, 649–654
25. Hallak, H., Brass, L. F., and Manning, D. R. (1994) J. Biol. Chem. 269, 4571–4576
26. Parenti, M., Viganò, M. A., Newman, C. M., Milligan, G., and Magee, A. I. (1993) Biochem. J. 291, 349–353
27. Degtyarev, M. Y., Spiegel, A. M., and Jones, T. L. Z. (1994) J. Biol. Chem. 269, 30898–30903
28. Linder, M. E., Pang, I.-H., Duronio, R. J., Gordon, J. I., Sternweis, P. C., and Gilman, A. G. (1992) J. Biol. Chem. 266, 4654–4659
29. O’Brien, P., St. Jules, R., Reedy, T., Bazan, N., and Zatz, M. (1987) J. Biol. Chem. 262, 5210–5215
30. Bizzozero, O., McGarry, J., and Lees, M. (1987) J. Biol. Chem. 262, 13550–13557
31. Neubert, T. A., Johnson, R. S., Hurley, J. B., and Walsh, K. A. (1992) J. Biol. Chem. 267, 18274–18277
32. Kokame, K., Fukada, Y., Yoshizawa, T., Takao, T., and Shimonishi, Y. (1992) Nature 359, 749–752
33. Wall, M., Coleman, D., Lee, E., Iniguez-Lluhi, J., Posner, B., Gilman, A., and Sprang, S. (1995) Cell 83, 1047–1058
34. Navon, S., and Fung, B.-K. (1987) J. Biol. Chem. 262, 15476–15751
35. Neer, E., Pulssifer, L., and Wolf, L. (1988) J. Biol. Chem. 263, 8996–9000
36. Peltzsch, R. M., and McLaughlin, S. (1993) Biochemistry 32, 10436–10443
37. Paige, L. A., Nadler, M. J. S., Harrison, M. L., Cassidy, J. M., and Geahlen, R. L. (1993) J. Biol. Chem. 268, 8669–8674
38. Shenoy-Scaria, A. M., Dietzen, D. J., Kwong, J., Link, D. C., and Lublin, D. M. (1994) J. Cell Biol. 126, 333–363
39. Berthiaume, L., Déchâtel, I., Pesek, S., and Resh, M. (1994) J. Biol. Chem. 269, 6498–6505
40. Hallak, H., Muszbek, L., Laposata, M., Belmonte, E., Brass, L. F., and Manning, D. R. (1994) J. Biol. Chem. 269, 4713–4718
41. Schlesinger, M. J., Veit, M., and Schmidt, M. F. G. (1993) in Lipid Modifications of Proteins (Schlesinger, M. J., ed) pp. 2–19, CRC Press, Boca Raton, FL
42. Munby, S. M., and Gilman, A. G. (1991) Methods Enzymol. 195, 215–223
43. O’Dowd, B. F., Hnatowich, M., Caron, M. G., Leffkowitz, R. J., and Bouvier, M. (1989) J. Biol. Chem. 264, 7564–7569
44. Ueda, N., Iniguez-Lluhi, J., Lee, E., Smrcka, A., Robishaw, J., and Gilman, A. (1994) J. Biol. Chem. 269, 4388–4395

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