Many eukaryotic cell surface proteins are anchored to the membrane via glycosylphosphatidylinositol (GPI). GPI is synthesized from phosphatidylinositol by step-wise reactions and attached en bloc to nascent proteins. In mammalian cells, the major GPI species transferred to proteins is termed H7. By attachment of an additional ethanolamine phosphate (EtNP) to the second mannose, H7 can be converted to H8, which acts as a minor type of protein-linked GPI and also exists as a free GPI on the cell surface. Yeast GPI7 is involved in the transfer of EtNP to the second mannose, but the corresponding mammalian enzyme has not yet been clarified. Here, we report that the human homolog of Gpi7p (hGPI7) forms a protein complex with PIG-F and is involved in the H7-to-H8 conversion. We knocked down hGPI7 by RNA interference and found that H7 accumulated with little production of H8. Immunoprecipitation experiments revealed that hGPI7 was associated with and stabilized by PIG-F, which is known to bind to and stabilize PIG-O, a protein homologous to hGPI7. PIG-O is a transferase that adds EtNP to the third mannose, rendering GPI capable of attaching to proteins. We further found that the overexpression of hGPI7 decreased the level of EtNP and, therefore, decreased the level of EtNP transferred to the third mannose. Finally, we propose a mechanism for the regulation of GPI biosynthesis through competition between the two independent enzymes, PIG-O and hGPI7, for the common stabilizer, PIG-F.
Experimental Procedures

Cell Lines—HeLa and K562 class K cells (19) were cultured in Dulbecco’s modified Eagle’s medium supplemented with 10% fetal calf serum. CHO hamster ovarian (CHO) cells were cultured in Ham’s F-12 supplemented with 10% fetal calf serum.

Cloning of Human GPI7 cDNA—We searched the GenBank database using the BLAST software (20) for homologous sequences to S. cerevisiae Gpi7p (YJL062w) and found a human cDNA containing a 3′ region and a genomic sequence (accession numbers AK000272 and AC015776, respectively). We designed two primers, 5′-H11032-3′-TGCCTGTGGGACGCT-TCCAGCCTAGCGTGTCCG (forward) and 5′-AC015776, respectively. We expected that hGPI7 would be in-}

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...analyzed by SDS-PAGE/Western blotting with anti-GST or anti-FLAG antibodies (18). CHO cells (4 × 10^6) were also co-transfected with 4 μg of pMEEB-FLAG-Pig and 2 μg of pMEEB-FLAG-PIG-F, 2 μg of pMEEB-FLAG-mPig-o, and 2 μg of pMEEB-FLAG-mPig-n, with 26 and 20% identity, respectively. hGPI7 shows homology to two other EtNP trans-

RESULTS

Cloning of hGPI7—We cloned the human homolog of yeast GPI7, which is involved in transferring EtNP to Man2 (9), and termed the gene hGPI7 (DDBJ/GenBankTM/EMBL accession number NM_017733). The hGPI7 gene encodes 853 amino acids, consists of 13 exons, and is located on chromosome 4p16.3 (30). The hGPI7 protein shows 26 and 28% amino acid identity to S. cerevisiae and Schizosaccharomyces pombe Gpi7p, respectively. The hGPI7 protein has conserved type I phosphodiesterase/nucleotide pyrophosphatase regions in its N-terminal hydrophilic domains (Fig. 2A). The first 27 amino acids of hGPI7 appear to constitute a signal sequence for translocation across the ER membrane. hGPI7 has a hydrophilic domain of ~400 amino acids in the N terminus followed by multiple putative transmembrane domains in the C-terminal region (Fig. 2B). hGPI7 shows homology to two other EtNP trans-ferases, PIG-O and PIG-N, with 26 and 20% identity, respectively (15, 16). Together, these data suggest that hGPI7 is one of the EtNP transferases in the GPI biosynthetic pathway.

hGPI7 RNAi Causes HeLa Cells to Accumulate GPI Biosynthetic Intermediates—We expected that hGPI7 would be in-

FIG. 1. Later steps in GPI biosynthesis in mammalian cells. G, glucosamine; M, mannose; E, ethanolamine phosphate; H6-H8, GPI intermediates according to Hirose et al. (31).

Subcellular Localization of hGPI7—CHO cells (6 × 10^6) transfected with 45 μg of pMEEB-hGPI7-GST and 20 μg of pMEEB-FLAG-PIG-F were used for subcellular fractionation (24). Membranes fractionated by sucrose density gradient centrifugation were lysed in 1% Nonidet P-40, 50 mM Tris-HCl (pH 7.4), 150 mM NaCl, and proteinase inhibitor mixture (Roche Applied Science). Insoluble material was removed by centrifugation for 1 h, and the GST-tagged proteins were precipitated from the supernatants with glutathione-Sepharose 4B. The GST-tagged and co-precipitated proteins were analyzed by SDS-PAGE/Western blotting and anti-GST or anti-FLAG antibodies (18). CHO cells (4 × 10^6) were also co-transfected with 4 μg of pMEEB-FLAG-PIG-F, 20 μg of pMEEB-hGPI7-GST, 2 μg of pMEEB-FLAG-mPig-o, and 2 μg of pMEEB-FLAG-mPig-n, with 26 and 20% identity, respectively. hGPI7 shows homology to two other EtNP trans-

Analysis of Protein Complexes—CHO cells (4 × 10^6) were co-transfected with 1 μg of pMEEB-FLAG-PIG-F, 3 μg of pMEEB-HA-mPig-o, 2 μg of pMEEB-HA-mPig-n, and 0, 10, or 20 μg of pMEEB-hGPI7. The total amounts of the plasmids were kept constant by adding the pMEEB vector. Two days later, the transfectants were lysed in 1% digitonin and divided into two aliquots. FLAG-PIG-F was precipitated with anti-FLAG M2-conjugated beads (Sigma) from one aliquot, and HA-tagged proteins were precipitated with anti-HA HA7 (Sigma) plus protein G-Sepharose (Amersham Biosciences) from the other aliquot. The precipitates were analyzed by Western blotting.

Stability of hGPI7 in PIG-F-defective Cells—CHO30.5 cells defective in the PIG-F gene were co-transfected with 8 μg of pMEEB-FLAG-PIG-F, 2 μg of pMEEB-FLAG-PIG-F, 2 μg of pMEEB-HA-Pig-o, and 10 μg of either pMEEB-FLAG-PIG-F or pMEEB vector. Two days later, the transfectants were lysed in buffer containing 1% Nonidet P-40, 50 mM Tris-HCl (pH 7.4), 150 mM NaCl, and proteinase inhibitor mixture. The GST-tagged proteins were precipitated with anti-GST antibodies and protein G beads (16, 24). The precipitates were analyzed by Western blotting and the Fuji Image Analyzer software (Fuji Film Co.).
olved in transferring EtNP to Man2. To test this possibility, we created knockdown (KD) HeLa cells in which hGPI7 expression was repressed by RNAi and analyzed their GPI. If our expectation was correct, hGPI7 KD cells would be deficient in generating H8 from H7. As shown in Fig. 1, H7 and H8 were characterized as GPIs containing EtNPs on Man1 and Man3 or on all three mannoses, respectively (31, 32). We stably transfected HeLa cells with pH1-sihGPI7 to generate hGPI7 KD cells.
cells and with pH1-siEGFP to generate control cells. The level of hGPI7 mRNA in hGPI7 KD cells was 27% that in control cells, indicating that the expression of hGPI7 was knocked down (Fig. 3A). As shown in Fig. 3B, the control cells showed a similar glycolipid pattern to that of HeLa cells (lanes 1 and 3). In contrast, hGPI7 KD cells had a different pattern, showing more than 5-fold accumulation of H7 and a decrease in H8 (Fig. 3B, lane 2 and line in the histogram). Because H7 and probably also H8 are used for GPI-anchoring to proteins (5, 33, 34), and as this may have an effect on the GPI profile, we knocked down hGPI7 expression in class K mutant cells, in which GPI is not transferred to proteins because of defective GPI transamidase (19). Similar to the case with HeLa hGPI7 KD cells, hGPI7 caused strong accumulation of H7 in the class K mutant cells (Fig. 3C). These results suggest that hGPI7 is indeed involved in transferring EtNP to Man2, thereby converting H7 to H8.

hGPI7 Is Localized in the Endoplasmic Reticulum—The enzymes involved in GPI biosynthesis are localized in the ER (1). We therefore investigated the subcellular localization of hGPI7, CHO cells expressing GST-tagged hGPI7 (hGPI7-GST) were homogenized and separated into five fractions by sucrose density gradient centrifugation. hGPI7-GST was mostly present in fractions 3–5, which contained the ER, but not in other fractions containing the plasma membrane and Golgi (Fig. 4). Although it has been reported that S. cerevisiae Gpi7p is mainly localized in the plasma membrane (9), the current results indicate that hGPI7 is localized in the ER rather than the plasma membrane of mammalian cells.

PIG-F Binds to and Stabilizes hGPI7 and PIG-O Independently—As shown in Fig. 3B, the catalytic activity of PIG-O was apparently enhanced as evidenced by the decrease in H6 and concomitant increase in H7 in hGPI7 KD cells. We therefore investigated the possibility that hGPI7 is somehow involved in regulating the PIG-O catalytic activity. hGPI7 is highly homologous to PIG-O and PIG-N. We previously reported that PIG-F binds to and stabilizes PIG-O, but not PIG-N (16). From these observations, we examined the association between PIG-F and hGPI7. We transfected CHO cells with FLAG-tagged PIG-F and GST-tagged mPig-n, mPig-o, or hGPI7. From their digitonin extracts, we affinity-precipitated GST-tagged proteins and analyzed the co-precipitated FLAG-PIG-F by Western blotting (Fig. 5A). FLAG-tagged PIG-F was co-precipitated with GST-tagged hGPI7 and mPig-o but not with mPig-n. These results indicate that PIG-F associates not only with PIG-O but also with hGPI7. We next examined whether PIG-F, PIG-O, and hGPI7 form a triple complex. We transfected CHO cells with FLAG-tagged PIG-F and GST-tagged mPig-n, mPig-o, or hGPI7. From their digitonin extracts, we affinity-precipitated GST-tagged proteins and analyzed the co-precipitated FLAG-PIG-F by Western blotting (Fig. 5A). FLAG-tagged PIG-F was co-precipitated with GST-tagged hGPI7 and mPig-o but not with mPig-n. These results indicate that PIG-F associates not only with PIG-O but also with hGPI7. We therefore investigated the subcellular localization of hGPI7, CHO cells expressing GST-tagged hGPI7 (hGPI7-GST) were homogenized and separated into five fractions by sucrose density gradient centrifugation. hGPI7-GST was mostly present in fractions 3–5, which contained the ER, but not in other fractions containing the plasma membrane and Golgi (Fig. 4). Although it has been reported that S. cerevisiae Gpi7p is mainly localized in the plasma membrane (9), the current results indicate that hGPI7 is localized in the ER rather than the plasma membrane of mammalian cells.

Fig. 3. Effects of hGPI7 RNAi on GPI biosynthesis. A, Northern blot analysis showing a decreased level of hGPI7 mRNA in RNAi cells. Samples of 40 μg of total RNA from HeLa cells (lane 1), control cells (lane 2), or hGPI7 KD cells (lane 3) were analyzed by Northern blotting with probes for hGPI7 or β-actin. B, in vivo mannose labeling. hGPI7 KD, control, and wild-type HeLa cells were radiolabeled with [3H]mannose for 45 min, and extracted lipids were subjected to TLC. Lane 1, control cells; lane 2, hGPI7 KD; lane 3, wild-type HeLa cells. Right panel shows the overlaid histograms. Intensities of radioactivity were corrected for dolichol-phosphate-mannose (DPM) intensities. Filled area, control cells; line, hGPI7 KD. C, in vivo mannose labeling in hGPI7 KD class K cells. Lane 1, control class K cells; lane 2, hGPI7 KD class K cells.
pared the expression levels of the GST-tagged proteins in the presence or absence of PIG-F expression (Fig. 5C). The expression level of hGPI7-GST in the absence of PIG-F was one-half that in the presence of PIG-F, whereas the expression levels of hPIG-L-GST and GST-msALDH remained unchanged. These results indicate that hGPI7, like PIG-O, is bound and stabilized by PIG-F.

hGPI7 Competes with PIG-O for Binding to PIG-F—Next, we considered the possibility that PIG-O and hGPI7 compete for binding to PIG-F. We transfected CHO cells with FLAG-PIG-F, HA-mPig-o, and HA-msALDH plasmids, and increasing amounts of hGPI7 plasmid. FLAG-PIG-F was immunoprecipitated, and the co-precipitates were analyzed by Western blotting. The levels of HA-mPig-o co-precipitated with FLAG-PIG-F decreased with increasing amounts of the hGPI7 plasmid (Fig. 6A, top panel, left), whereas the expression levels of FLAG-PIG-F and HA-msALDH remained unaffected (bottom panel and top panel, right). Similar to the level of co-precipitated HA-mPig-o, the total expression levels of HA-mPig-o decreased with increasing amount of hGPI7 (top panel, right), suggesting that HA-mPig-o detached from PIG-F was unstable and degraded rapidly. We obtained similar results by changing the amount of mPig-o plasmids (data not shown). These results demonstrate that hGPI7 competes with PIG-O for binding to PIG-F and imply that the expression levels of hGPI7 are capable of limiting the expression levels of PIG-O.

To determine the effect of the competition between hGPI7 and PIG-O on GPI biosynthesis, we analyzed the effect of hGPI7 overexpression in HeLa cells. The hGPI7-overexpressing cells accumulated more H6 than the mock transfectants (Fig. 6, B and C), implying inefficient conversion of H6 to H7. These results are consistent with the hypothesis that overexpression of hGPI7 decreases the biosynthetic activity of PIG-O by decreasing the available PIG-F, thereby destabilizing PIG-O.

**DISCUSSION**

In the late phase of GPI biosynthesis in mammalian cells, GPI species H7 bearing EtNP linked to Man3 is generated from H6 by the action of an enzyme complex consisting of PIG-O and PIG-F (Fig. 1). H7 can be converted to H8 by further addition of a side chain, most likely EtNP, to Man2 (32). In the yeast *S. cerevisiae*, Gpi7p is involved in the EtNP side-chain modification of Man2 (9). The major finding in the present study is that an enzyme complex consisting of hGPI7 and PIG-F is involved in the conversion of H7 to H8. H7 and the major protein-linked GPI-anchors have the same glycan structure (5, 33), suggesting that H7 is the major substrate of GPI transamidase, which transfers GPI to proteins. A glycan structure corresponding to that of H8 was found in a small fraction of GPI-anchored proteins (33). Non-protein-linked free H8 is expressed on the cell surface (35). The biological roles of the side chain on Man2 and free H8 remain to be determined. Our present finding clarified the enzyme that generates H8.

First, we cloned *hGPI7* and knocked it down by means of RNAi. When the *hGPI7* mRNA level was reduced to 27% in HeLa cells by RNAi, H7 increased 5-fold, whereas H8 decreased (Fig. 3). Increased accumulation of H7 was also seen when *hGPI7* knockdown was performed in class K K562 cells. Based on these results, we conclude that hGPI7 is involved in H7-to-H8 conversion.

Yeast Gpi7p, Gpi13p, and Mcd4p and their respective homologs hGPI7, PIG-O, and PIG-N form a family of EtNP-transferases. hGPI7 is closer to PIG-O (26% amino acid identity) than to PIG-N (20% identity), whereas PIG-O is closer to PIG-N than to PIG-N (20% identity) (15, 16). Similar relationships are true among the yeast members, because the amino acid identities between Gpi7p and Gpi13p, Gpi7p and Mcd4p, and Gpi13p and Mcd4p are 24, 21, and 21%, respectively (13, 14). These structural relationships are consistent with their functional characteristics, namely their substrate specificities; hGPI7/Gpi7p and PIG-O/Gpi13p are involved in EtNP additions to the 6-position in mannose, whereas PIG-N/Mcd4p transfers EtNP to the 2-position (5, 9, 13–16). We previously reported that PIG-O is associated with PIG-F (16). PIG-F is also involved in transferring EtNP to Man3 because PIG-F-
defective mutant cells do not generate H7 and accumulate H6 (17), which is the immediate precursor of H7 (Fig. 1). PIG-F is a highly hydrophobic ER membrane protein of 20 kDa (17) with no significant homology to any other proteins with known functions. In the absence of PIG-F, PIG-O is unstable and its expression level is only 20–25% of that in the presence of PIG-F (16). The PIG-F expression level is not dependent upon PIG-O. Therefore, PIG-F stabilizes PIG-O. PIG-F, however, does not bind to PIG-N (16). In the present study, we have shown that PIG-F binds to hGPI7 (Fig. 5, A and B). The observation that PIG-F binds to hGPI7 and PIG-O but not to PIG-N parallels the higher sequence identity of hGPI7 to PIG-O compared with PIG-N.

We also found that PIG-F stabilizes hGPI7 (Fig. 5C) similarly to its stabilization of PIG-O. The expression level of hGPI7 in the absence of PIG-F was one-half that in the presence of PIG-F. The complexes of PIG-O and PIG-F and hGPI7 and PIG-F are formed separately. Moreover, PIG-O and hGPI7 compete for binding with PIG-F (Fig. 6A). Therefore, when the amount of PIG-F is limited, the level of PIG-O is influenced by PIG-F. A higher level of hGPI7 results in a lower level of PIG-O and hence in lower generation of H7.

It is not known whether Gpi7p and/or Gpi13p are associated with Gpi11p, an S. cerevisiae homolog of PIG-F (13). The profile of accumulated GPI mannosides in Gpi11p-defective cells is similar to that in Gpi7p-defective cells, but not to that in Gpi13p-defective cells. It is therefore possible that Gpi7p, but not Gpi13p, is dependent upon Gpi11p in S. cerevisiae.

The biological significance of GPI7 in yeast has been well studied. Most of the genes involved in GPI biosynthesis are known to have abnormalities, among the two forms of yeast GPI-anchored proteins (36). Approaches to identifying GPI-anchored proteins are based on the observation that PIG-F binds to GPI-anchored proteins (36). A possible mechanism for generating GPI-anchored proteins is to increase the number of GPI-anchored proteins, which might have been selected for by their function in growth (37). The presence of PIG-F in the absence of PIG-F was one-half that in the presence of PIG-F. The complexes of PIG-O and PIG-F and hGPI7...
proteins, the expressions of cell wall proteins with the ceramide-type GPI are severely affected, whereas the expressions of plasma membrane proteins with the diacylglycerol-type GPI, such as Gas1p, are not affected (37). Whether defective diacylglycerol-to-ceramide remodeling of GPI in the gpi7-null mutant (9). It was recently reported that Gpi7p is essential for separation of the daughter cells after cytokinesis. The GPI-anchored protein Egt2p, which is normally concentrated in the septum, was mislocalized in the cytokinesis. Gpi7p but may be involved in processes such as remodeling of cell wall, mammalian GPI7 cannot have the same role as yeast GPI or targeted transport of GPI-anchored proteins.

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Association of GPI7 with PIG-F
GPI7 Is the Second Partner of PIG-F and Involved in Modification of Glycosylphosphatidylinositol

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