The yeasts enzymes involved in UDP-GlcNAc biosynthesis are potential targets for antifungal agents. GNA1, a novel member of the Gen5-related N-acetyltransferase (GNAT) superfamily, participates in UDP-GlcNAc biosynthesis by catalyzing the formation of GlcNAc6P from AcCoA and GlcN6P. We have solved three crystal structures corresponding to the apo Saccharomyces cerevisiae GNA1, the GNA1-AcCoA, and the GNA1-CoA-GlcNAc6P complexes and have refined them to 2.4, 1.3, and 1.8 Å resolution, respectively. These structures not only reveal a stable, β-intertwined, dimeric assembly with the GlcNAc6P binding site located at the dimer interface but also shed light on the catalytic machinery of GNA1 at an atomic level. Hence, they broaden our understanding of structural features required for GNAT activity, provide structural details for related aminoglycoside N-acetyltransferases, and highlight the adaptability of the GNAT superfamily members to acquire various specificities.

The Gen5-related N-acetyltransferases (GNATs) represent a large superfamily of functionally diverse enzymes that catalyze the transfer of an acetyl group from AcCoA to the primary amine of a wide range of acceptor substrates (for a review, see Ref. 1). Recently, three-dimensional structural information has become available with the structures of two aminoglycoside N-acetyltransferases from Serratia marcescens (SmAAT) (2) and Enterococcus faecium (EfAAT) (3); five histone acetyltransferases (HATs): PCAF (4), HAT1 (5), Tetrahymena GCN5 (tGCN5) (6, 7), yeast GCN5 (yGCN5) (8), and Hpa2 (9); and one arylalkylamine N-acetyltransferase, the serotonin N-acetyltransferase (AANAT) (10). These two structures also provide the first glimpses into the catalytic machinery of GNAT. Yet, a better understanding of the diverse modes of acceptor substrate recognition and the catalytic mechanism of the GNATs as well as further insights into the evolution of this superfamily still awaits additional structural studies of GNAT-substrate complexes.

Although the GNAT AcCoA binding site is well documented, the binding site of the acceptor substrate has been characterized in only two cases: the histone binding site in the tGCN5-CoA-H3 peptide complex structure (7) and the serotonin binding site in the AANAT-substrate analog complex structure (10). These two structures also provide the first glimpses into the catalytic machinery of GNAT. Yet, a better understanding of the diverse modes of acceptor substrate recognition and the catalytic mechanism of the GNATs as well as further insights into the evolution of this superfamily still await additional structural studies of GNAT-substrate complexes.

Glucosamine-6-phosphate N-acetyltransferase 1 (GNA1) is a novel amino-sugar N-acetyltransferase member of the GNAT superfamily. GNA1 holds a key position in the pathway toward de novo synthesis of the essential metabolite UDP-GlcNAc and is present in various eukaryotic organisms (Fig. 1A). The GNA1 murine homologue, Emeg32, which possesses an extra 31-residue NH2-terminal region compared with the yeast homologue, has recently been characterized (13). Emeg32 is essential for embryonic development, and its inactivation in mouse embryonic fibroblasts generates resistance to apoptotic stimuli and defects in cell proliferation (14). GNA1 has also been characterized in yeast (15) and shown to be essential to the survival of Saccharomyces cerevisiae in which it controls multiple cell cycle steps (16). In addition, Candida albicans GNA1 null mutants exhibit reduced virulence when injected into mice (17), making GNA1 a potential target for the development of new antifungal agents.

We present here three high resolution crystal structures of GNA1. The apo GNA1 and the binary GNA1-AcCoA complex structures were solved independently using MAD techniques and refined to 2.4 and 1.3 Å resolution, respectively (Table I). The structure of the GNA1-CoA-GlcNAc6P ternary complex was solved by molecular replacement and refined to 1.8 Å resolution (Table I). These three structures reveal GNA1 catalytic features and provide the first complete picture of an amino-sugar GNAT active site as a first step toward the development of specific inhibitors.

**EXPERIMENTAL PROCEDURES**

Expression and Purification of the Native and Selenomethionyl Proteins—Full-length GNA1 was amplified from genomic S. cerevisiae DNA, cloned into the pQE30 expression vector, and transformed into M15pREP4 (Qiagen) Escherichia coli cells. Protein expression was induced with 0.1 mM isopropyl-1-thio-β-D-galactopyranoside for 20 h at 37 °C. Selenomethionyl GNA1 was produced as previously published (18). The recombinant His-tagged native and selenomethionyl enzymes were purified via nickel affinity and anion exchange chromatographies, dialyzed against 10 mM Tris-HCl, pH 8, 150 mM NaCl, 1 mM dithiothreitol, and concentrated to 10 mg/ml. The activity of these enzymes was verified.

**Crystallization**—Crystals of the native and selenomethionyl protein...
were grown at 20 °C using the hanging-drop vapor diffusion method by mixing equal volumes of the protein solution and the reservoir, which contained 17–22% polyethylene glycol 600 and 0.2/0.4 M imidazole/maleate, pH 5.1. Crystals obtained from the apo protein belong to the orthorhombic space group P2₁2₁2₁ and contain 6 molecules/asymmetric unit. Those obtained from GNA1 preincubated overnight with 1 mM AcCoA were positioned similarly. Because the GNA1-CoA-GlcN6P structure was solved at 2.5 Å; superimposition of the two ternary complexes (GNA1-CoA-GlcNAc6P and GNA1-CoA-GlcN6P) revealed that GlcN6P (the substrate) and GlcNAc6P (the reaction product) were bound in the same orientation. Four density maps were improved by solvent flattening, non-crystallographic symmetry averaging, and phase extension with the program DM (20). The apo and AcCoA-complexed GNA1 structures were solved in the P2₁2₁2₁ space group in the unit cell, which contains 6 molecules/asymmetric unit. Those obtained from GNA1 preincubated overnight with 1 mM AcCoA or CoA belong to the monoclinic space group C₂ and contain 4 molecules/asymmetric unit. For data collection, the crystals were transferred to a reservoir solution containing 32% polyethylene glycol 600 and 20% 2-propanol. Crystals of apo GNA1 and of the GNA1-CoA-GlcNAc6P complex soaked in the reservoir solution supplemented with 1 mM CoA-GlcNAc6P-complexed GNA1 have been deposited in the Protein Data Bank (accession codes 1I12, 1I11, and 1I1D).

**RESULTS AND DISCUSSION**

**Overall Structure**—The three-dimensional structures of GNA1 in its apo state and complexed forms with AcCoA or with CoA and GlcNAc6P have been solved and refined at 2.4, 1.3, and 1.8 Å resolution, respectively. Overall, the electron density is well defined for these structures (Fig. 1B) except for a surface loop comprising residues Gln-52 to Lys-57 (cf. “Experimental Procedures”). As predicted from sequence analysis, GNA1 shares structural similarities with other GNAT superfamily members (1, 11). The GNA1 fold consists of a central core, composed of a mixed 5-stranded β-sheet flanked by 4 α-helices, and a COOH-terminal strand β₆, which is projected away from the central core (Fig. 2A).

The GNA1 structure is dimeric in the crystal as well as in solution, as attested from gel filtration data (not shown). The crystalline dimer is made of two intertwined GNA1 monomers in which strand β₆ of one subunit exchanges with the identical strand from the other subunit (Fig. 2A). A β-strand exchange between subunits in a dimer is an unusual feature among GNATs and has been observed only in the HAT Hpa2 structure (9). In all other structurally characterized GNAT, except Hat1 and 1.8 Å resolution, overall, the electron density is well defined for these structures (Fig. 1B) except for a surface loop comprising residues Gln-52 to Lys-57 (cf. “Experimental Procedures”). As predicted from sequence analysis, GNA1 shares structural similarities with other GNAT superfamily members (1, 11). The GNA1 fold consists of a central core, composed of a mixed 5-stranded β-sheet flanked by 4 α-helices, and a COOH-terminal strand β₆, which is projected away from the central core (Fig. 2A).

**Table I**

| Structural statistics | Apo | AcCoA | CoA-GlcNAc6P |
|-----------------------|-----|-------|--------------|
| **Resolution (Å)**    | 2.4 | 1.3   | 1.8          |
| **Completeness (%)**  | 99.3| 99.0  | 99.6         |
| **Anomalous completeness** | 94.7| 93.0  | 94.9         |
| **R_factor**          | 25  | 21.021| 16.423       |
| **R_free**            | 10  | 9.7   | 12.5         |

a Numbers in parentheses refer to values for the highest resolution shell.

b \( R_{	ext{factor}} = \sum |I(I-I_{\text{ref}})| / \sum I \), where I is the observed intensity and \( I_{\text{ref}} \) is the average intensity for all equivalent reflections.

c \( R_{	ext{free}} = \sum |I(I-I_{\text{ref}})| / \sum I \), where I and \( I_{\text{ref}} \) are the intensities of Bijvoet positive and negative reflections, respectively.

d Values in parentheses refer to the number of reflections used in the random test set.

e r.m.s.d., root mean square deviation.

The stereochemistry of the refined models was analyzed by PROCHECK (25); no residue was found in the disallowed regions of the Ramachandran plot. The coordinates of apo, AcCoA-, and CoA-GlcNAc6P-complexed GNA1 have been deposited in the Protein Data Bank (accession codes 1I12, 1I11, and 1I1D). Figs. 1, 2, 4, and 5 were generated with SPOCK (26) and Raster3D (27) except for Fig. 1A, which was computed with Asperts (28).
The black shown in are identified by Subunit 1 residues involved in the GlcNAc6P and AcCoA binding sites for SmAAT and Hpa2 is based on a structural comparison with GNA1. The presence of this N-myristoyltransferase (30) but is a unique feature within the structurally characterized GNATs.

Superimposition of the apo and AcCoA-complexed GNA1 structures shows that AcCoA binding induces subtle structural rearrangements that are confined to the edges of the cleft and result in a slightly narrower cleft. Residues 102–109 in the α3–β5 loop and 134–143 in the β5–α4 loop plus the N-cap of α4 move by −1.3 and 1.1 Å, respectively, toward the center of the cleft (Fig. 2A). Whether these conformational changes, induced upon cofactor binding, are a prerequisite for acceptor substrate binding as shown for other GNATs (31–33) needs to be ascertained by kinetic studies. A detailed comparison with other GNATs reveals that these rearrangements differ from those reported for (i) tGCN5, in which the cofactor-binding cleft opens slightly upon AcCoA binding to accommodate the histone tail (7); and (ii) AANAT, in which a major rearrangement of the α1-loop-α2 region occurs upon AcCoA binding to complete the serotonin binding site (10). Therefore, although the binding of AcCoA is similar among GNATs, it induces different conformational changes that can contribute to the specific binding of the acceptor substrate.

The Acceptor Substrate Binding Site—The GNA1 aminoglycoside binding site exhibits an atypical architecture, as it is built at the dimer interface and involves residues from the exchanged β-strand, two features found only in a few intertwined oligomeric structures such as that of bovine seminal ribonuclease (34). GlcNAc6P binds at the base of the AcCoA cleft within a small pocket that is lined mostly with electronegative residues except for a patch of positively charged residues that specifically accommodate the 6-phosphate group (Fig. 2B). Remarkably, the GlcNAc6P acetyl group is positioned similarly to the cofactor acetyl group in the GNA1-AcCoA complex structure (Fig. 2B). The sugar-6-phosphate establishes numerous hydrogen bonds, mainly via side chain atoms, together with a few hydrophobic contacts such as that found between Leu-27 and the β-face of the sugar ring (Fig. 2C). Superimposition of the structures of the cofactor-complexed SmAAT (2) or EfAAT (3) (the two structurally characterized aminoglycoside GNATs) on that of the GNA1-AcCoA-GlcNAc6P complex shows that SmAAT Phe-51, EfAAT Trp-25, and GNA1 Leu-27 are identically positioned, an observation that supports a common functional role for these residues, thereby identifying an aminoglycoside recognition feature of GNATs.

Catalytic Mechanism—Several reports on GlcN6P N-acetyltransferases suggest that catalysis requires sulfhydryl group-containing residues, such as a cysteine that could act as a nucleophile in a two-step mechanism involving the formation of a covalent acetyl-cysteine enzyme intermediate (35, 36). In contrast, structural and kinetic data available for GNATs support a mechanism proceeding through a direct nucleophilic reaction of acceptor substrate on AcCoA (1, 32, 33).

In the GNA1-AcCoA complex structure, the cysteine residue closest to the AcCoA acetyl group lies 6.5 Å apart, too far to play a role in acetyl transfer. Furthermore, no other appropriate nucleophile residue is found in the proximity of the acetyl group, which makes the formation of an acetyl-enzyme intermediate very unlikely and supports the hypothesis of a single-step mechanism as suggested for GNATs. Consistent with this hypothesis is the position of the amino group of the product GlcNAc6P (similar to that of the substrate GlcN6P; cf. “Experimental Procedures”), which is ideal to allow a direct nucleophilic attack at the AcCoA carbonyl (Fig. 2B). In addition, the nucleophilic character of the amine is enhanced by the hydrogen bond it establishes with the backbone carbonyl of Asp-134 (Fig. 2C). The AcCoA carbonyl is polarized via hydrogen bonds to the backbone amides of Asp-99 and Ile-100, located in the oxanion hole, a feature that facilitates the nucleophilic attack...
and stabilizes the negative charge building up on the oxygen atom of the tetrahedral reaction intermediate (Fig. 3). Finally, the Tyr-143 hydroxyl group, which lies within hydrogen bond distance of the AcCoA sulfur atom (Fig. 2B), could serve to stabilize the thiolate anion of the departing CoA molecule.

The first striking difference resides within the GNA1 β-bulge which, when compared with the β-bulges in other GNAT structures, shows a markedly different hydrogen bonding pattern. The GNAT β-bulge is an irregularity of the antiparallel β structure in which two residues on one strand are facing a single residue on the other strand (37). Such a β-bulge is formed when two consecutive residues in a β-strand direct their backbone carbonyl or amide toward the same side of the strand, thereby breaking the typical alternated pattern of a β-structure. In GNA1, the backbone amides of Asp-99 and Ile-100 are projected toward the active site and form the oxynion hole, whereas the carbonyls of Glu-98 and Asp-99 point toward β3. In all other members of the GNAT superfamily (except EfaAT in which a proline perturbs the bulge conforma-
(3)), the situation is reversed; the oxyanion hole is absent because the two consecutive backbone amides are now directed toward strand β3 (establishing an hydrogen bond with the backbone carbonyl of the facing residue), whereas two consecutive main chain carbonyls are found pointing into the active site (Fig. 4). These two carbonyls have been suggested to play a role in acceptor substrate binding in tGCN5 (7) and in the stabilization of catalytic water molecules in AANAT (10); this indicates that, in addition to a common role in structuring the cofactor binding site, the β-bulge could also fulfill other non-conserved catalytic functions.

The second important difference concerns the deprotonation of the acceptor substrate amino group prior to the reaction. In the case of tGCN5 and AANAT, a chain of well ordered water molecules, or “proton wire,” connecting the acceptor substrate amino group to the proposed catalytic bases tGCN5 Glu-122 or AANAT His-120 was suggested to be involved in this proton removal (7, 10). In the GNA1-CoA-GlcNAc6P complex, a similar proton wire is observed, leading to Glu-98 in which the side chain occupies a similar position as that of tGCN5 Glu-122 and AANAT His-120. However, the E98A mutation does not abolish the GNA1 activity (15), suggesting that Glu-98 might not function as the general base. Nevertheless, deprotonation prior to the reaction might not be necessary in the case of GNA1, because the pKₐ of GlcN6P (−7.75) is lower than that of other GNAT acceptor substrates such as lysine (8.95) or serotonin (−10). This hypothesis is also supported by the fact that the optimum pH of purified mammalian GlcN6P N-acetyltransferases lies in the alkaline range (35, 36) and by the lower Kₐ value of S. cerevisiae GNA1 for GlcN6P at pH 8 than at pH 7.5 (13), suggesting that GNA1 may preferentially bind the basic/deprotonated form of GlcN6P.

Substrate Specificity among GNATs—Although the GNAT enzymes share structural similarities, they have distinct acceptor specificities, consistent with their implication in various biological processes. A comparative analysis of the complexes of GNA1-CoA-GlcNAc6P, tGCN5-CoA-H3 peptide (7), and AANAT-bisubstrate analog (7, 10) highlights the structural determinants responsible for the substrate specificities among GNATs. Importantly, this knowledge is essential for the design of specific inhibitors for medical applications.

The structural comparison of these three complexes reveals that both the NH₂- and COOH-terminal regions diverge between the different GNAT structures and are important for substrate specificity. The NH₂-terminal structural differences concern the α1-loop-α2 region and are relatively minor, whereas more dramatic changes occur in the COOH-terminal end. In AANAT, the α4-β6 loop orients toward the active site as it folds back on its own subunit. This loop, along with the

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**Fig. 3.** Schematic representation of the proposed GNA1 catalytic mechanism.

**Fig. 4.** The β-bulge structure in GNA1 and comparison with another GNAT superfamily member. The structure of GNA1-CoA-GlcNAc6P (thick sticks with light gray carbons) is superimposed with that of AANAT (thin sticks with dark gray carbons) in the vicinity of the β-bulge. Nitrogen and oxygen atoms from the protein main chain or the acetylated amine of GlcNAc6P are shown in black. Light and dark gray dotted lines indicate hydrogen bonds in the structures of GNA1 and AANAT, respectively.
in UDP-GlcNAc biosynthesis (which implies a physiological link between UDP-GlcNAc and cell cycle progression) or if it is the consequence of an additional function of GNA1.

The hypothesis of an additional HAT activity for GNA1 was addressed, but no HAT activity could be detected in vitro (15). Interestingly, a comparison of GNA1 with the related GNAT structures reveals that the closest structural homologue of GNA1 is the HAT Hpa2, which also adopts an intertwined dimeric structure (9). Superimposition of the two structures reveals differences in the relative arrangement of the two subunits, resulting in different acceptor substrate binding sites. A narrow open-ended channel, in which the histone tail could insert, is found in the Hpa2 structure instead of the rounded pocket of GNA1, which seems unlikely to accommodate an extended and bulky histone tail.

Could GNA1 fulfill an additional function via the noncovalent association with a particular cell compartment or with a protein partner? An association with the cytoplasmic face of organelle membranes has been described for EMe32 (the GNA1 murine homologue) which also co-purifies with the cdc48 homologue protein (p97/VCP) (13). Double-hybrid systematic experiments performed in S. cerevisiae revealed interactions between GNA1 and a priori unrelated or unknown proteins (38). Further biochemical experiments are needed to determine the biological relevance of these protein/membrane interactions.

UDP-GlcNAc is a key precursor of chitin (a component of the yeast and fungal cell wall) as well as of the glycosylphosphatidylinositol anchor of membrane-bound proteins and is essential to N-linked glycosylation and O-GlcNAc modification of proteins. Glycosyltransferases involved in N-glycosylation, such as the yeast GPT/alg7, which uses UDP-GlcNAc as a substrate, have been suggested to play a role in the cell cycle (39). In addition, a recent report shows that EMe32-dependent UDP-GlcNAc levels influence cell cycle progression and apoptosis signaling (14). Hence, the role of GNA1 in cell cycle progression appears to be linked to its key GlcN6P N-acetyltransferase activity in de novo UDP-GlcNAc biosynthesis. The structural data presented here have allowed us to propose a catalytic mechanism for GNA1, as well as providing a structural template for GNA1 homologues and related aminoglycosides GNATs. Finally, these results further exemplify the remarkable diversity of the GNAT superfamily and represent a critical step toward the development of specific inhibitors.

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Fig. 5. Structural comparison of substrate binding sites of AANAT, tGCN5, and GNA1. The molecular surfaces around the active site of the AANAT-bisubstrate analog (A), the tGCN5-CoA-H3Peptide (B), and the GNA1-CoA-GlcNAc6P complex structures (C) are viewed with a similar orientation. From top to bottom, the serotonin-like moiety and the acetyl group are shown in red, the H3 peptide backbone in yellow, with its reactive Lys-14 side chain in orange, and GlcNAc6P in purple. Structural divergences within the NH₂- and COOH-terminal regions are highlighted in dark blue and blue, respectively. The β3–β4 insertion loop unique to AANAT is shown in brown.

α1–α2 loop, almost covers the active site, thus facilitating the binding of a hydrophobic substrate (Fig. 5A). In tGCN5, the 20-residue segment inserted between α4 and β6 contributes to one side of the substrate binding canal, providing specific binding residues for the histone tail (Fig. 5B).

In GNA1, the shorter α4–β6 loop does not participate directly in acceptor substrate binding, but it forces strand β6 to extend and exchange with the identical strand of the other subunit in the dimer. Instead of using this loop, GNA1 exploits its intertwined oligomeric state to achieve specific binding, because an important part of the GNA1 active site consists of residues from the other subunit in the dimer (Fig. 5C). In contrast, this region is partly replaced in monomeric AANAT by a long loop inserted between strands β3 and β4, which contributes largely to one wall of the serotonin binding site (Fig. 5A). For the monomeric tGCN5, the short β3–β4 turn contributes to the canal-shaped active site designed to accommodate a long peptidic chain (Fig. 5B).

Role in the Cell Cycle—GNA1 was shown to control multiple cell cycle steps in S. cerevisiae (16). It is still unclear whether this role is related to the N-acetyltransferase activity of GNA1
Crystal Structure of S. cerevisiae GNA1

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The Crystal Structures of Apo and Complexed *Saccharomyces cerevisiae* GNA1 Shed Light on the Catalytic Mechanism of an Amino-sugar N-Acetyltransferase

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