Molecular Characterization of Golgin-245, a Novel Golgi Complex Protein Containing a Granin Signature*

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The serum from a Sjögren’s syndrome patient with anti-Golgi antibodies was used as a probe to isolate a 4.6-kilobase pair cDNA insert from a HeLa cDNA library. Expression of the cDNA in Escherichia coli and the in vitro translation products of the cDNA yielded a recombinant protein that migrated in SDS-polyacrylamide gel electrophoresis at 180 kDa. This protein was immunoprecipitated by the human anti-Golgi serum and by immune rabbit serum but not by normal human serum or preimmune rabbit serum. Western blot analysis showed that the prototype human and immune rabbit sera recognized a 245-kDa protein, suggesting that the isolated clone contained a partial cDNA. The 5′-upstream sequence obtained by the rapid amplification of cDNA ends methodology using human placental cDNA and the combined HeLa cDNA contained 6965 base pairs and encoded a protein of 245 kDa and, like other Golgi autoantigens described earlier, is highly rich in coiled-coils. The deduced amino acid sequence included the decapetide ESLALEEL, which was identified as one of two signature sequences previously reported in a family of peptide hormones and neuropeptides known as “gramins.” This is the first report of a Golgi complex autoantigen that bears structural similarities to the granin family of proteins.

Human autoantibodies have proven to be valuable reagents in the identification and characterization of a number of intracellular macromolecules (1). Recently, human autoantibodies have been used to characterize and identify several Golgi complex proteins including golgin-95 and -160 (2) and giantin (3, 4). Macrogolgin was reported to be a new Golgi autoantigen (5), but subsequent studies noted that macrogolgin was identical to giantin (4). Sohda and his colleagues (6) reported a 372-kDa polypeptide that was immunoprecipitated by the human anti-Golgi serum and by immune rabbit serum but not by normal human serum or preimmune rabbit serum. Western blot analysis showed that the prototype human and immune rabbit sera recognized a 245-kDa protein, suggesting that the isolated clone contained a partial cDNA. The 5′-upstream sequence obtained by the rapid amplification of cDNA ends methodology using human placental cDNA and the combined HeLa cDNA contained 6965 base pairs and encoded a protein of 245 kDa and, like other Golgi autoantigens described earlier, is highly rich in coiled-coils. The deduced amino acid sequence included the decapetide ESLALEEL, which was identified as one of two signature sequences previously reported in a family of peptide hormones and neuropeptides known as “gramins.” This is the first report of a Golgi complex autoantigen that bears structural similarities to the granin family of proteins.

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The nucleotide sequence(s) reported in this paper has been submitted to the GenBank/EMBL Data Bank with accession number(s) U13906. § Scientist of the Alberta Heritage Foundation for Medical Research. To whom correspondence should be addressed: Faculty of Medicine, 3330 Hospital Dr. N.W., Calgary, Alberta T2N 4N1, Canada. Tel.: 403-220-4564; Fax: 403-283-5666; E-mail: fritzler@acs.ucalgary.ca.

acids (QLSSM) inserted at residue 215 of the giantin amino acid sequence; thus GCP372 and giantin are likely products of alternatively spliced mRNAs derived from the same gene.

In the present study, we used the serum from a Sjögren’s syndrome patient to isolate a cDNA clone encoding a novel Golgi complex protein, which we have designated golgin-245. Its amino acid sequence revealed the presence of multiple coiled-coil motifs, a sequence signature characteristic of a family of proteins known as gramins, and sequence similarity to the heavy chain of myosin.

MATERIALS AND METHODS

Antibodies—The prototype serum was from a patient (FG) with idiopathic diffuse proliferative glomerulonephritis, renal failure requiring dialysis, and clinical features of Sjögren’s syndrome. The specificity of the autoantibodies for Golgi antigens was first identified on the basis of indirect immunofluorescence (IF) (2, 7). IF–IF was performed on commercially prepared HEP-2 cell (epithelial cells derived from a human laryngeal carcinoma) substrates (Immunno Concepts Inc., Sacramento) using a fluorescein-conjugated goat anti-human IgG (light and heavy chain) as described previously (2). A murine monoclonal antibody to the coatamer protein β-COP (Sigma) was used for co-localization studies. Double labeling and co-localization studies employed rhodamine-conjugated goat anti-rabbit or -mouse IgG (Pierce) antisera. Slides were viewed with a Zeiss Universal microscope fitted with a TEC-470 CCD video camera system (Optronics Engineering, Goleta, CA), and images were processed with a SONY color video printer and recorded on SONY video tape. Western Blotting—HeLa cells grown in culture flasks were extracted in buffer containing 50 mM Tris, pH 7.6, 150 mM NaCl, 20 mM EDTA, 0.5% Nonidet P-40, and protease inhibitors as described (2). Proteins or whole cell preparations were solubilized in SDS sample buffer, separated by discontinuous SDS-PAGE, and then transferred to nitrocellulose as described (2, 8). After transfer, the nitrocellulose sheets were blocked with 3% nonfat milk in phosphate-buffered saline containing 0.05% Tween 20 (PBST) and again washed with PBST to remove any unbound antibodies. Bound antibodies were traced by using 125I-protein A (2–4 × 10⁶ cpm/ml; ICN Radiochemicals, Irvine, CA) and visualized by exposing the air-dried nitrocellulose to X-Omat AR film (Eastman Kodak Co.).

cDNA Cloning, 5′-RACE, and Sequence Analysis—FG serum was used for immunoscreening 5 × 10⁶ recombinants from a HeLa Uni-ZAP XR cDNA library (Stratagene, La Jolla, CA, catalog no. 937216) as described (2). A clone ACJ F521 was isolated and subcloned into vivo into pBluescript plasmid using R408 helper phage as described in the manufacturer’s instructions (Stratagene). The nucleotide sequences were determined by using dye terminator sequencing and an automated sequencer from Applied Biosystems Inc. (Foster City, CA). Since ACJ F521 was a partial sequence, the RACE methodology was employed to obtain overlapping 5′- or 3′-ends from human placenta cDNAs (5′-RACE-PCR).
Ready cDNAs, Clontech) (9). DNA sequences were determined in both strands and compiled using Applied Biosystems Inc software SeqEd.

Northern Blotting—Total cellular RNA was isolated from HeLa cells (10). Ten μg of total HeLa RNA was fractionated by electrophoresis through 1% agarose containing 2.2 M formaldehyde and transferred to nitrocellulose membrane. The RNA blot was hybridized with 32P-labeled cDNA probes (11) and exposed to X-ray film for 2-14, and blots were washed at 53°C in 0.1 x SSC and 0.1% SDS.

RT-PCR Mapping of Golgin-245 mRNA—Total HeLa RNA was analyzed by RT-PCR to provide independent confirmation that the cDNA clones derived from 5'-RACE were derived from the same mRNA. Three sets of sense and antisense primers were designed, and their positions with respect to the putative full-length cDNA are indicated on Fig. 2a: H1, antisense 5'-GAAACGGGATCCCGGTGCGCGCAAGATG and sense 5'-CACTTCTTTTATTTGGCTACATGAGGGCGCCCTGAATG; H2, antisense 5'-AATTCCGGCTCAGAAGAATG and sense 5'-TGATGCGACCAAGTTGGAAG; H3, antisense 5'-GTGGGTACCTGGGATCAGGAAG and sense 5'-TGGTACCTGGGATCAGGAAGCTAGCAG. RT-PCR was performed using the “one-step” method as described (11) such that all reactions were added simultaneously, and the PCR steps were programmed to follow immediately after the RT reaction using a thermocycler (Eppendorf Inc., Fremont, CA). The RT reactions were performed at 50°C for 1 h using SuperScript II RNase H-minus reverse transcriptase (Life Technologies, Inc.). For primer pairs 44/45, 46/47, and 44/47, Taq polymerase (Life Technologies, Inc.) was used in PCR for 25 cycles (94°C for 1 s, 70°C for 2.5 min, and 94°C for 2 s). For primer pair 42/43, the Expand long template PCR system (Boehringer Mannheim) was employed to increase the chance for the expected 6.4-kb amplification product. The PCR was performed using 10 cycles of 58°C for 5 s, 68°C for 7 min, and 94°C for 5 s followed by 15 cycles of 58°C for 5 s, 68°C for 15 min, and 70°C for 5 s. Amplification products were analyzed by electrophoresis using 1.0 or 1.5% agarose gels using standard procedures.

Affinity Purification of Antibodies—Uni-ZAP clone ACJF521 phage plaques were induced to produce recombinant protein with isopropyl-β-D-thiogalactopyranoside (Fisher Scientific)-impregnated nitrocellulose filters, and the filters with the bound recombinant proteins were then used for affinity purification as described previously (2).

In vitro Translation and Immunoprecipitation—One μg of plasmid ACJF521 was used for coupled in vitro transcription and translation (Tri-I, Promega, Madison, WI) in the presence of T3 and T7 RNA polymerase, rabbit reticulocyte lysate, and [35S]methionine and translation (TnT, Promega, Madison, WI) in the presence of T3 RNA polymerase, rabbit reticulocyte lysate, and [35S]methionine (Tran35S-label; ICN). Translation was carried out at 30°C for 1 h followed by SDS-PAGE of a 2-5% gel. The proteins were transferred to nitrocellulose and utilized for the production of rabbit antisera (11). Affinity-purified antibodies from unrelated phage plaques showed staining of the Golgi complex (Fig. 1a), which was similar to the staining demonstrated by the whole serum (Fig. 1b). In addition, the antisera directed against Golgin-160 (Fig. 1c), Affinity-purified antibodies from unrelated phase plaques did not stain any cell structures (data not shown). The staining obtained with the rabbit antibodies to recombinant ACJF521 co-localized to the same cellular structure as the murine monoclonal antibody to β-COP (Fig. 1, d and e, respectively).

These findings were in keeping with previous studies that have localized other autoantigens to the Golgi compartment (2, 3, 5, 6). Additional evidence supporting the localization of the reactive antigen to the Golgi complex came from HeLa cells treated with 7.5 μM brefeldin A. These studies demonstrated dramatic loss of staining by the prototype human and rabbit immune sera after 10 min of exposure to the drug and restoration of staining 90 min after removal of the drug (data not shown). The sensitivity of golgin-245 to brefeldin A resembles the rapid disappearance of β-COP (16), golgin-95, and golgin-160 (2) after exposure of tissue culture cells to the drug.

Isolation and Characterization of cDNAs Encoding Full-length cDNA—Sequence analysis of cDNA ACJF521 showed that it was composed of a long coding region followed by a noncoding region, a poly(A) tail (Fig. 2a), and features suggesting that it was a partial cDNA (also see below). To obtain the complete 5'-cDNA, the 5'-RACE methodology was used. In the first round of 5'-RACE, eight overlapping, independent clones were analyzed with the longest cDNA insert (29-9) containing 1856 bp and represented an open reading frame extending 5' from the ACJF521 cDNA (Fig. 2). However, clone 29-9 lacked a methionine start site consistent with Kozak's sequence (17). A second round of 5'-RACE was initiated with newly designed primers based on the 5'-sequence of 29-9. The sequences of four overlapping independent clones were determined, and the longest cDNA insert (2-14) contained 690 bp with an open reading frame extending 5' to clone 29-9 (Fig. 2). The combined sequence derived from overlapping clones 2-14, 29-9, and ACJF521 contained a total of 6965 bp (Figs. 2 and 3). The first ATG was found at nucleotide position 50 and is in excellent agreement with Kozak's sequence (17) for a methionine initiation codon. Based on the Genetics Computer Group program PEPTIDESORT analysis, the combined sequence encodes a protein of an open reading frame of 2083 amino acids with a predicted molecular mass of 244,527 daltons and a pl of 5.15. In keeping with previous nomenclature (2) the recombinant protein encoded by the combined cDNA was named golgin-245.

Independent confirmation that the 5'-RACE clones are derived from a single mRNA was obtained by Northern blotting when both 5'-fragment 2-14 and 3'-fragment ACJF521 hybridized to a common band of 7 kb (Fig. 2b), a size consistent with the full-length cDNA sequence reported here. A second independent confirmation was provided from the RT-PCR mapping data (Fig. 2c). The results showed that the 3 pairs of designed primers each deriving from different cDNA fragments produced RT-PCR products consistent with the expected sizes. Taken together, these data showed that the cDNA sequences reported here derived from a single mRNA of approximately 7 kb.

The deduced amino acid sequences were used to search the GenBank, EMBL, and NBRF databases (August 30, 1995) for homologous sequences. Golgin-245 demonstrated 97% identity with a sequence tag (X76942) identified as 72.1 protein con-
taining 149 amino acids. The location of the X76942 sequence in relation to golgin-245 is shown in Fig. 2a. There were no apparent transmembrane, nuclear localization, or other signal motifs. Like golgins-95 and -160, 20% identity of amino acid sequences was found with the myosin heavy chain and several other proteins (CENP-E, CENP-F) that contain coiled-coil domains. Since the regions of similarity are widely spread throughout the sequences of the proteins, it is assumed that they are related to the coiled-coil domain structure. There was no significant sequence similarity with β-adaptin, β-COP, previously reported Golgi complex autoantigens, or other known mammalian Golgi components. It has been previously noted that golgin-95 and golgin-160 also share significant sequence similarity to several cytoskeleton-related proteins including desmin, the 150-kDa dynein-associated protein (18), the myosin family proteins (myosin heavy chain, tropomyosin), and kinesin (19). Kinesin, a microtubule-stimulated ATPase (20), has recently been reported to be a motor for microtubule-mediated Golgi-to-endoplasmic reticulum membrane traffic (21). In support of this conclusion, it has been demonstrated that antibodies to kinesin and myosin bind to the Golgi complex (22, 23). To extend the comparison, golgin-160 and macrogolgin have sequence similarity to the yeast cytoskeleton-related protein USO1, a yeast protein that is required for protein transport from the endoplasmic reticulum to the Golgi apparatus (24). This evidence suggests that the Golgi complex autoantigens are functionally and structurally related.

The decapeptide -ESLALEELEL- (amino acids 388–397; Figs. 3 and 4) was identified as a granins_1 “signature,” which has the consensus pattern [DE]-[SN]-L-[SAN]-X(2)[DE]XEL (25, 26). The granins are a family of acidic proteins present in the secretory granules of a wide variety of endocrine and neuroendocrine cells (25, 26). Two consensus sequences have been reported. The granins_1 signature is located at the carboxyl
terminus of the proteins and has been identified in all granin family proteins with the exception of murine secretogranin 2. In contrast, the second signature sequence, granins_2, has been described in only two members of the granins family, chromogranin A and B, and is characterized by two cysteine residues bound together near the amino terminus of the protein. The predicted pI of golgin-245 (5.15) is similar to the pI of granins, which range between 4.9 and 5.6 (26). The relatively higher content of glutamic acid (17%) compared with aspartic acid (4%) is another feature of golgin-245 that is shared with most granins (26). The function of these proteins or these signatures has not been clearly defined, but current evidence suggests that one of their functions is processing or packaging of neuropeptides. The similarities between granins and golgin-245 are remarkable and suggest that they have similar functions.

Secondary structure analysis using the program "COILS" (15) identified coiled-coils throughout the length of the protein (Fig. 4). This feature is of interest because all Golgi complex autoantigens cloned to date are predominantly coiled-coil structures. Large domains of coiled-coils have also been noted in other autoantigens including the nuclear mitotic antigen (NuMA) (27), lamin B (28), and myosin heavy chain (29), and one or two coiled-coils have been noted in the 52-kDa SS-A/Ro (30) and 80/86-kDa Ku antigens (31). This motif has also been described in a number of other prokaryotic and eukaryotic proteins, most notably Golgi complex proteins (32, 33), the streptococcal M protein (29), products of the nuclear oncogenes c-fos and c-jun (34), viral fusogenic proteins (35), and the envelope glycoproteins of HIV-1 and other viruses (36). Although certain proteins with coiled-coils are able to bind DNA and are thought to be predominantly regulatory in their function (37), this structure also mediates the dimerization of certain trans-activators (38) and is essential for recombination of certain viral proteins (39) and oligomerization of viral envelope proteins (40). These observations suggest that the Golgi complex autoantigens, including golgin-245, may participate in protein-protein interactions.
In immunoblotting, recombinant protein from the ACJF521 clone was reactive with the prototype serum, with antibody affinity-purified from the ACJF521 recombinant protein and with immune rabbit serum (data not shown). This was clearly demonstrated by parallel experiments, which used the in vitro translated product derived from the cDNA clone ACJF521 (Fig. 5A). The translation product protein was approximately 180 kDa (lane 1) and was immunoprecipitated by the prototype human serum (lane 3) and immune rabbit sera (lane 4) but not by normal human serum (lane 2) or preimmune rabbit sera (lane 5). Immunoblot analysis of HeLa cell extracts (Fig. 5B) with the rabbit serum directed against golgin-245 (lane 3) and the prototype human serum (lane 4) demonstrated reactivity with a 225-kDa (lane 3) protein whereas the preimmune rabbit serum (lane 1) showed no reactivity. A prototype serum with antibodies directed against the 225-kDa NuMA protein (1) was included as a control to obtain a more accurate assessment of the molecular mass of the reactive protein (lane 2).

Antibodies directed against the Golgi complex have been reported in the sera of patients with systemic rheumatic diseases (i.e. SLE, Sjögren's syndrome) (7, 41–47), idiopathic cerebellar ataxia (48), paraneoplastic cerebellar degeneration (49), and viral infections including the Epstein-Barr virus (50) and the human immunodeficiency virus (51). In this study, we have used the antibodies from a patient with Sjögren's syndrome, a disease characterized by lymphocytic infiltrates and fibrosis of exocrine glands, to clone and characterize a unique Golgi complex protein.

**Fig. 3.** Nucleotide and deduced amino acid sequences of the cDNA clone encoding golgin-245 antigen. Sequencing of both DNA strands was performed with custom synthetic oligonucleotide primers. The open reading frame start at nucleotide 50 and ends at nucleotide 6298. The signature sequence for granins_1 at amino acids 388–397 and the polyadenylation signal AATAAA are both underlined. The combined nucleotide and amino acid sequences have been submitted to GenBank under accession number U31906.

**Fig. 4.** Structural features of golgin-245. Multiple coiled-coils are detected throughout the length of golgin-245 while only one short coiled-coil was found in the COOH-terminal domain of human chromogranin. The location of the granin_1 signature is indicated by the arrow.

Characterization of ACJ F521 Proteins—In immunoblotting, recombinant protein from the ACJ F521 clone was reactive with the prototype serum, with antibody affinity-purified from the ACJ F521 recombinant protein and with immune rabbit serum (data not shown). This was clearly demonstrated by parallel experiments, which used the in vitro translated product derived from the cDNA clone ACJ F521 (Fig. 5A). The translation product protein was approximately 180 kDa (lane 1) and was immunoprecipitated by the prototype human serum (lane 3) and immune rabbit sera (lane 4) but not by normal human serum (lane 2) or preimmune rabbit sera (lane 5). Immunoblot analysis of HeLa cell extracts (Fig. 5B) with the rabbit serum directed against golgin-245 (lane 3) and the prototype human serum (lane 4) demonstrated reactivity with a 225-kDa (lane 3) protein whereas the preimmune rabbit serum (lane 1) showed no reactivity. A prototype serum with antibodies directed against the 225-kDa NuMA protein (1) was included as a control to obtain a more accurate assessment of the molecular mass of the reactive protein (lane 2).

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**Unique Golgi Complex Protein Contains Granin Signature**

**Fig. 5. Immunoactivity of recombinant ACJ F521.** A, immunoprecipitation of [35S]methionine-labeled recombinant protein. The recombinant protein obtained from the coupled in vitro transcription and translation of the ACJ F521 cDNA migrated at \(~180 \text{ kDa}\) in a 4–20% gradient gel SDS-PAGE (lane 1) and was immunoprecipitated by the prototype serum FG (lane 3) and the immune rabbit sera (lane 4) but not by normal human serum (lane 2) or preimmune rabbit serum (lane 5). B, immunoblot analysis of HeLa whole cell extracts separated by 10% gel SDS-PAGE and probed using rabbit antibodies raised against golgin-245 recombinant protein. A serum with anti-NuMA (arrowhead, \(225 \text{ kDa}\)) is shown in lane 2; a 245-kDa protein and a less prominent higher mobility protein. All sera were diluted 1:200 except the prototype serum in lane 4, which is diluted 1:50. Molecular mass markers are shown on the right.

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