Facilitative glucose transporter isoform 4 (GLUT4) in rat adipocytes is largely sequestered in intracellular sites, and insulin recruits GLUT4 from these sites to the cell surface. The process is known to involve multiple intracellular compartments and associated proteins, many of which are yet to be identified. Recently, we purified three distinct insulin-sensitive intracellular GLUT4 compartments (G4TL, G4H, and G4L) in rat adipocytes and unraveled several new resident proteins in these compartments. Here, we describe one of them, a 62-kDa protein, purified and identified as rat adipose tissue carboxyl esterase (p62/CE) by matrix-assisted laser desorption/ionization time of flight mass spectrometry, reverse transcription-polymerase chain reaction, gene cloning, and immunological and enzymatic activity measurements. p62/CE in rat adipocytes was 80% cytosolic and 20% microsome-associated. It was found in all of the three insulin-sensitive intracellular GLUT4 compartments, and particularly enriched in G4TL, a compartment thought to represent GLUT4 endocytic vesicles. Significantly, an antibody against p62/CE introduced into rat adipocytes completely abolished the insulin-induced GLUT4 recruitment to the plasma membrane in host cells without affecting the basal GLUT4 distribution. Together, these findings suggest that p62/CE plays a key role in insulin-induced GLUT4 recruitment in rat adipocytes, probably by hydrolyzing acylglycerols or acyl-CoA esters to the respective free acids that are required for GLUT4 transport vesicle budding and/or fusion.

The uptake of glucose by muscle and adipose cells is primarily catalyzed by GLUT4, a facilitative glucose transporter isoform, and further regulated by hormones, specifically by insulin (1, 2). GLUT4 in these cells constantly and rapidly recycles between the cell surface and intracellular compartments, is found mostly in intracellular compartments, and is recruited to the plasma membrane in response to insulin (3–5). In morphological studies, GLUT4 is found in association with tubulovesicular structures, small vesicles, clathrin-coated lattices, and clathrin-coated vesicles (6, 7), indicating that multiple subcellular compartments are involved in GLUT4 sequestration, recycling, and recruitment. Exact roles of these intracellular compartments or organelles in GLUT4 regulation remain largely undefined. Biochemical studies, on the other hand, identified a number of proteins that are associated with immunopurified GLUT4-containing vesicles (8–18), implying that some of these proteins may play a role in GLUT4 regulation. Compartment-specific association of these proteins and their role in GLUT4 recycling and recruitment, however, are also unknown.

By using hypotonic lysis, differential centrifugation, and glycerol gradient velocity sedimentation, we (19) have recently separated GLUT4 in rat adipocytes into four distinct fractions, namely, TH, TL, H, and L. Data of endosomal and organelle-specific marker distribution revealed that the fraction TH contains both intracellular and plasma membrane GLUT4 compartments (designated as G4TH and G4PM, respectively), whereas GLUT4 in the fractions TL, H, and L represents exclusively intracellular compartments (designated as G4TL, G4H, and G4L, respectively). Furthermore, GLUT4 compartments in these fractions were distinct in response to insulin; insulin treatment reduced G4H and G4L compartments and increased G4TL compartment. Based on these findings, we suggested that G4H and G4L represent the putative GLUT4 storage endosomes and GLUT4 exocytic vesicles, respectively, while G4TL represents GLUT4 endocytic vesicles. G4TL, G4H, and G4L have been purified by immunoadsorption and shown to be distinct in protein composition.

In the present study, we describe an association of a 62-kDa protein with these immunopurified GLUT4 compartments. MALDI-TOF MS, RT-PCR, gene cloning, immunoreactivity, and enzyme activity assay identified this protein as a neutral carboxyl esterase expressed in rat adipocytes (20). Most importantly, the p62/CE antibody selectively abolished the insulin-induced GLUT4 redistribution to the cell surface in semipermeabilized rat adipocytes, without affecting the basal GLUT4 distribution. Based on these findings, we propose a working hypothesis that p62/CE modulates lipid metabolites in GLUT4 compartments and regulates membrane budding and fusion.
required for the insulin-induced GLUT4 recruitment in rat adipocytes.

EXPERIMENTAL PROCEDURES

Materials—Collagenase (type I) was obtained from Worthington. 1F8 was purchased from Biogenesis Ltd. (Sandown, NH). Horseradish peroxidase-labeled protein A and anti-mouse IgG were from Zymed Laboratories Inc. (San Francisco, CA). Antibody against rat adipose tissue carboxyl esterase (p62/CE) was raised in rabbits using purified rat adipose tissue carboxylesterase (21). Oligonucleotides primers were from IDT (Lexington, KY).

Preparation and Hypotonic Lysis of Adipocytes—Adipocytes were isolated from epididymal fat pads of male Harlan Sprague-Dawley rats and stabilized as described (22). Adipocytes were then lysed as described (19) using hypotonic lysing medium (0.25 mM ATP, 2.5 mM MgCl₂, 0.1 mM MgCl₂, 0.1 mM NaCl, 0.05 mM NAD, and 1 mM KHCO₃, pH 7.4) (23).

Subcellular Fractionation by Differential Centrifugation and Glycerol Gradient Velocity Sedimentation—The procedures have been described (19). Briefly, adipocyte lysates (from 3–5 rats) were centrifuged at 900 g for 15 min and the resulting pellet (900 g pellet) was resuspended in 30 ml of 5–30% glycerol sucrose cushion. The 900 g supernatant was centrifuged at 185,000 g for 2 h, and the pellet (185,000 g pellet) was resuspended in 600 ml of NaCl/HEPES buffer. Suspension of 900 g or 185,000 g pellet, without or after sonication (in a bath type sonicator, for 2 h at 4 °C), was then layered onto 9 ml of 5–30% glycerol cushion (24)

Northern Blot Analysis—Proteinase K digestion, essentially as described (22). 3 ml of LB broth (1.0% tryptone, 0.5% yeast extract, 0.5% NaCl, pH 7.5) containing ampicillin (100 μg/ml) was inoculated with single colony of Escherichia coli DH5α using pGEX-3X plasmid DNA. After incubation at 37 °C for 16 h, the cells were collected from the culture by centrifugation (11,000 rpm, 15 min, 4 °C), and used for double-stranded DNA preparation (Promega Wizard DNA purification kit). p62 full sequence flanked with Smal restriction site on both ends was generated by RT-PCR. Smal-restricted vector DNA and p62/Smal fragment were ligated at 14 °C and 16 h. The ligation mix was transformed into the competent E. coli DH5α or BL21. From the transformant E. coli colony, we purified double-stranded DNA and carried out DNA sequencing.

RESULTS

Carboxyl Esterase in GLUT4 Compartments in Rat Adipocytes

The GLUT4 compartments in fractions 8–10 were purified and used for double-stranded DNA preparation (Promega Wizard DNA purification kit). p62 full sequence flanked with Smal restriction site on both ends was generated by RT-PCR. Smal-restricted vector DNA and p62/Smal fragment were ligated at 14 °C and 16 h. The ligation mix was transformed into the competent E. coli colony, we purified double-stranded DNA and carried out DNA sequencing.

Carboxyl Esterase Activity—Carboxyl esterase activity against the water-soluble substrate para-nitrophenyl butyrate acid was determined by spectrophotometric method (28). In this procedure, the samples were diluted with 0.5 ml Tris-HCl (pH 7.4 or 8.0) and sodium acetate (pH 5.0) in the presence of 6% taurocholate. The assay was initiated by adding a freshly prepared para-nitrophenyl butyrate acid solution (100 μg/ml in sodium acetate, pH 5.0). Carboxyl esterase activity was then monitored and compared with control autohydrolysis of the substrate using an Amersham Pharmacia Biotech spectrophotometer set at 405 nm.

Streptolysin O (SLO) Permeabilization and Antibody Treatment—Adipocytes were permeabilized with SLO as described (29) with minor modifications. Adipocytes were washed with cold intracellular buffer (140 mM potassium glutamate, 20 mM HEPES, 5 mM MgCl₂, 5 mM EGTA, 5 mM NaCl, pH 7.2) and incubated with intracellular buffer containing 200 units/ml of SLO at room temperature for 15 min. Adipocytes were washed twice with intracellular buffer containing 1 mg/ml of bovine serum albumin and ATP-regenerating system (40 IU/ml creatine phosphokinase, 5 mM creatine phosphate, and 1 mM ATP) and incubated with 0.5 mg/ml anti-carboxyyl esterase antibody or normal rabbit IgG for 40 min at 37 °C. When specified, adipocytes were treated with 100 nM insulin for the last 20 min of the permeabilization protocol above.

Other Methods—DNA sequencing was done at the SUNY at Buffalo Sequencing Laboratory. Protein was determined with a BCA kit (Pierce) according to the manufacturer’s instructions. Adipocyte membrane fragments enriched with plasma membranes (PMs) and low density microsomes (LDMs) were prepared after homogenization as described (3).

RESULTS

Particulate fractions T₁₄, T₁₅, H, and L (19) were prepared from rat adipocytes by hypotonic lysis, differential centrifugation, and glyceral gradient velocity sedimentation (see “Experimental Procedures”). The GLUT4 compartments in fractions T₁₄, H, and L (G4T₁₄, G4H, and G4L, respectively) were purified by immunoadsorption using 1F8 (see “Experimental Procedures”). GLUT4 in the fraction T₁₄ (G4T₁₄) was not immuno-

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Fig. 1. Protein composition and GLUT4 content of GLUT4 compartments immunopurified from fractions T1, H, and L. Fractions T1, H, and L were isolated from basal (+) or insulin-stimulated (-) adipocytes, and each (100 μg of protein) was subjected to immunoadsorption with 1F8 (1F8) or normal mouse IgG (NI) (see "Experimental Procedures"). Adsorbed materials were eluted with SDS-containing Laemmli buffer without β-mercaptoethanol for 1 h at room temperature. After adding β-mercaptoethanol (1% final), the proteins were resolved in SDS-PAGE in 8% gel and subjected to silver staining for protein analysis (upper panel) and GLUT4-immunoblots (lower panel). Bands a and b (upper panel) were 120- and 62-kDa proteins, respectively, that were purified and analyzed by MALDI-TOF MS.

The immunoadsorption procedure described in Fig. 1 was scaled up to purify proteins in sufficient quantities for sequence identification, using fraction H because of its large protein yield and GLUT4 compartment size. Proteins in 1F8-immunosorbed materials were separated by SDS-PAGE and visualized by silver staining as in Fig. 1. Selected protein bands (marked as a and b in Fig. 1) in gel were excised and digested with trypsin, and resulting peptide fragments were analyzed by MALDI-TOF MS (see "Experimental Procedures"). Such an analysis performed for 62-kDa protein (b in Fig. 1) is shown in Table I. From the linear tryptic spectrum, 19 potent peptide fragments were matched with rat hepatic neutral cholesterol ester hydrolase (nCEH; EC 3.1.1.13) (GenBankTM accession no. L46791) (30). Similarly, MALDI-TOF MS of tryptic fragments of 120-kDa protein (a in Fig. 1) indicated its potential identity with KIAA0339 (GenBankTM accession no. AB002337), a protein containing ATP/GTP binding site motif A, whose function is not known (31). Further characterization of this protein will be considered in a separate report.

To confirm the expression of nCEH in rat adipocytes, we performed RT-PCR in rat adipocytes using three different primer sets (see "Experimental Procedures") designed from three highly conserved regions of nCEH sequence, CE-S1, -2, and -3 (27). Using β-actin as a control, RT-PCR using the primer sets for nCEH produced corresponding size of DNA from rat adipose tissue mRNA (Fig. 2A). To further confirm the identity of p62 with nCEH, we sequenced the CE-S1, CE-S2, and CE-S3-amplified DNA segments from poly(A)+ RNA of rat adipose tissue obtained above. The sequence was found to be 99.7% identical to that of rat hepatic nCEH mRNA (with five nucleotide differences out of 1695 nucleotides).

Next, CE-S1, -2, and -3 above were labeled by random priming with [32P]dATP and used as probes for Northern blot analysis. A typical result of the Northern blot with CE-S1 is illustrated in Fig. 2B, again using β-actin as a control. p62 mRNA was detected in rat adipose tissue at appropriate size (about 1.9 kb), demonstrating that a nCEH-like protein p62 (hereafter referred to as p62/CE) is indeed expressed in rat adipose tissue. We cloned p62/CE (GenBankTM accession number AF171640). Deducing amino acid sequence of cloned p62/CE was identical to that of rat liver carboxyl esterase (ES-10) (32) with an exception of one residue (Asn instead of Lys at position 265). It was approximately 2.5-fold greater at pH 8 than at pH 5 (not illustrated), indicating that p62/CE is a neutral carboxyl esterase (20).

A carboxyl esterase, known as FAEE synthase/CE, has been purified from rat adipose tissue by one of us (20) and partially characterized. A polyclonal antibody was raised using the purified protein of this carboxyl esterase (21). The antibody has been shown to be highly specific to the enzyme; it reacted with only one protein in rat adipose tissue extract with no other cross-reactive protein. Furthermore, the antibody inhibited CE activity in rat adipose tissue but not other carboxyl ester-hydrolyzing enzymes in the tissue including lipoprotein lipase, hormone-sensitive lipase, cholesterol esterase, and cholinesterase.

This antibody cross-reacted with purified recombinant p62/CE in immunoblot (Fig. 2C). Although the DNA sequence has not been reported, the N-terminal 27-amino acid sequence of FAEE synthase/CE has been known (21), which is identical to that of p62/CE, suggesting that the two are the same protein.

| Mass | Matching sequences with nCEH |
|------|----------------------------|
| 2845.8 | Glu106-Lys129 |
| 1628.5 | Phe65-Lys78 |
| 1709.1 | Leu172-Arg186 |
| 3232.6 | Ala243-Lys274 |
| 1375.8 | Thr275-Lys296 |
| 2891.5 | Thr275-Lys296 |
| 1534.7 | Glu287-Lys309 |
| 2662.7 | Ser378-Lys400 |
| 3998.2 | Ser378-Lys412 |
| 1071.7 | Tyr401-Lys410 |
| 2808.0 | Asp537-Lys560 |
| 3918.9 | Thr641-Lys673 |
| 927.5 | Tyr657-Arg686 |
| 2863.0 | Tyr657-Lys686 |
| 1244.8 | Ile537-Lys546 |
| 2604.5 | Ile537-Arg564 |
| 1621.4 | Leu537-Lys551 |
| 1136.7 | Leu537-Lys551 |
| 830.4 | Glu541-Arg549 |
PCR, Northern blot, and Western blot analyses. A contained 3.5 μl of PCR products which were analyzed in a 1% agarose gel. CE-S1–5 (lane 3), CE-S2–5’ and CE-S2–3’ primers (lane 4), and CE-S3–5’ and CE-S3–3’ primers (lane 5), respectively. Lanes 1 and 6 were DNA size markers, while lane 2 contained a β-actin fragment as a control. B, the CE-S1 labeled with [3P]dATP from A was used as a probe for Northern blot analysis to detect cholesterol esterase mRNA poly(A)⁺ RNA of rat adipose tissue as described under “Experimental Procedures.” Blots were analyzed by PhosphorImager analysis (Molecular Dynamics, Inc., Sunnyvale, CA) after 3-day exposure. The images have been digitally processed to facilitate qualitative comparisons. C, recombinant p62/CE was produced from BL21 cells containing pGEX-3X/p62 construct. Recombinant p62/CE protein (100 ng) was subjected to 10% SDS-PAGE separation and then Western blot using the antibody specific to p62/CE as described under “Experimental Procedures.” Molecular marker positions are indicated by arrowheads.

The antibody inhibited the esterase activity of purified p62/CE recombinant protein in vitro (Fig. 3). The antibody also reacted with the rat liver carboxyl esterase (ES-10) and rat hepatic nCEH (21). We refer to this antibody as anti-p62/CE antibody.

Direct testing of the identity of p62 purified from the GLUT4 compart-ments by p62/CE antibody. Protein p62 was purified from GLUT4-containing vesicles in fraction H after immunoadsorption with 1F8 as described in the legend to Fig. 1. The p62 was microeluted from SDS gel and subjected to immunoprecipitation with 3 μg of rat carboxyl esterase antibody (αCE) and protein A-agarose beads. 3 μg of normal rabbit IgG (NI) were used for nonimmune control. 1/2 of the supernatant (Sup) and 1/2 of the precipitates (Pt) were separated on SDS-PAGE, and p62/CE was detected by immunoblotting using rat carboxyl esterase antibody (A) and by silver stain (B). Positions of molecular size markers are indicated in kDa. Similar results were reproduced in two other experiments.

The antibody inhibited the esterase activity of purified p62/CE recombinant protein in vitro (Fig. 3). The antibody also reacted with the rat liver carboxyl esterase (ES-10) and rat hepatic nCEH (21). We refer to this antibody as anti-p62/CE antibody and have used it here for characterization of p62/CE in rat adipocytes.

Direct testing of the identity of p62 purified from the GLUT4 compartment by p62/CE antibody. Protein p62 was purified from GLUT4-containing vesicles in fraction H after immunoadsorption with 1F8 as described in the legend to Fig. 1. The p62 was microeluted from SDS gel and subjected to immunoprecipitation with 3 μg of rat carboxyl esterase antibody (αCE) and protein A-agarose beads. 3 μg of normal rabbit IgG (NI) were used for nonimmune control. 1/2 of the supernatant (Sup) and 1/2 of the precipitates (Pt) were separated on SDS-PAGE, and p62/CE was detected by immunoblotting using rat carboxyl esterase antibody (A) and by silver stain (B). Positions of molecular size markers are indicated in kDa. Similar results were reproduced in two other experiments.
rat adipocytes and studied its effect on the steady-state distribution of endogenous GLUT4 between the PM and the LDM. Basal and insulin-stimulated adipocytes were permeabilized using SLO in the absence and in the presence of normal rabbit IgG or anti-p62/CE antibody (0.5 mg/ml each) (see “Experimental Procedures”), and PM and LDM fractions were isolated and subjected to immunoblot with GLUT4 antibody. In IgG-incorporated adipocytes, insulin increased the PM GLUT4 content 5–6-fold, with a concomitant reduction (by 25–30%) in the LDM GLUT4 level (Fig. 7). These are essentially identical to the GLUT4 subcellular distributions observed in intact (non-permeabilized) adipocytes (3). Similar insulin effects on PM and LDM GLUT4 levels were observed in SLO-treated adipocytes in the absence of nonimmune IgG or antibody (data not illustrated). In p62/CE antibody-incorporated adipocytes, however, there was no observable difference in GLUT4 levels in PM or LDM between the basal and insulin-stimulated states (Fig. 7), indicating that the p62/CE antibody completely abolished insulin-stimulated GLUT4 redistribution. This occurred without affecting basal GLUT4 level (Fig. 7).

**DISCUSSION**

GLUT4 recycling and its insulin-induced recruitment to the cell surface in adipocytes are known to involve multiple protein functions in multiple intracellular compartments (5, 6). Clear identification of the individual intracellular GLUT4 compartments and the associated protein functions involved in these processes has been elusive. Recent success in separation and partial purification of three distinct, intracellular GLUT4 compartments from hypotonically lysed rat adipocytes (19) allowed us to detect and study novel association of certain proteins with GLUT4 compartments that hitherto escaped from identification. Among these was a 62-kDa protein (Fig. 1), which we have identified here as adipose tissue neutral carboxyl esterase, p62/CE. This identity was first suggested by MALDI-TOF MS tryptic fragmentation spectrum profile analysis (Table I) and subsequently confirmed by RT-PCR (Fig. 2A). Northern blotting (Fig. 2B), gene cloning (GenBank™ accession no. AF171640), enzyme activity assay (Fig. 3), and immunological cross-reactivity (Figs. 2C and 4). Adipose tissue carboxyl esterase (FAEE synthase/CE) has been purified and partially characterized (20, 21). It is a member of the mammalian carboxyl esterase (CE) superfamily (EC 3.1.1.1) (27) and hydrolyzes a variety of esters including endogenous substrates such as short- and long-chain acylglycerols and long-chain acyl-CoA esters to the respective free acids with broad substrate specificity.

The association of p62/CE with GLUT4 compartments is unequivocal both in protein staining (Fig. 1) and in immunoblotting (Fig. 6). p62/CE, like many other CE isoforms, contains the C-terminal four amino acids capable of conferring retention in the ER (32). There are exceptions to this ER retention signal rule, however (30); ES-10 and nCEH, for example, contain the N-terminal cleavable signal sequence and the C-terminal ER retention signal sequence, HVEL, yet ES-10 is exclusively found in ER lumen, whereas nCEH is mostly (about 80%) in the cytosol. It is quite possible that p62/CE may also reside in the ER, and this by itself would not necessarily contradict the...
presence of p62/CE in cytosol and its association with GLUT4 vesicles. Please also note that GLUT4-containing compartments purified by 1F8 in fractions T_1L, H, and L were free of ER, yet showed p62/CE immunoreactivity. In preliminary experiments, we have noted that 1 M KCl or alkaline wash reduces p62/CE association with GLUT4 vesicles, suggesting cytoplasmic, rather than luminal, interaction of p62/CE with the vesicles.

Quantitative assessment of the association of p62/CE with each of the GLUT4-compartments, however, was less than unequivocal, a problem not uncommon in assessment of extrinsic membrane protein binding to membranes. Notably, immunoblot data showed no clear insulin effect on p62/CE association in each GLUT4 compartment (Fig. 6), while an apparent reduction in p62/CE protein staining intensity in G4H was observed in some (19) but not all insulin-stimulated adipocytes (Fig. 1). In protein staining data, p62/CE appeared more abundant in G4TL than in G4H or G4L (Fig. 1), although such a difference was not clearly seen in immunoblots (Fig. 6). However, when normalized against GLUT4 content, p62/CE was significantly more concentrated in G4TL in both assays (Figs. 1 and 6), G4TL is a compartment composed exclusively of small vesicles, free of integrin but enriched in transferrin receptor and Rab5, and thought to represent GLUT4 endocytic vesicles (19).

p62/CE antibody in permeabilized rat adipocytes completely abolishes insulin-induced recruitment of GLUT4 without affecting basal GLUT4 distribution (Fig. 7). This, together with the fact that the antibody inhibits the esterase activity of p62/CE or a p62/CE-catalyzed hydrolysis in the insulin-induced GLUT4 recruitment in adipocytes. The role of p62/CE in insulin-induced GLUT4 recruitment, however, is yet to be determined. An interesting possibility is that this neutral esterase hydrolyses certain lipid component(s) in nonacidic, GLUT4 compartments such as sorting and/or recycling endosomes and regulates GLUT4 transport vesicle sorting, budding, or fusion. The high p62/CE concentration observed in G4TL and G4H, the presumed endocytic vesicles and recycling/storage endosomes, respectively, is consistent with this possibility. Endosomal lipids are known to undergo enzymatic processing (33). The hydrolysis of endosomal cholesterol ester by nCEH, for example, was implicated in free cholesterol transport vesicle sorting, budding, or fusion in cholesterol homeostasis (34). Recently, insulin-sensitive phospholipid signal pathways have been implicated in insulin-induced GLUT4 recruitment in rat adipocytes (35). Thus, the activation of phospholipase D-dependent phosphatidylcholine hydrolysis and de novo phosphatidic acid synthesis by insulin result in formation of phosphatidic acid and phosphatidylinositol (4,5)P_2-enriched microdomains in endosomes, which may promote vesicle formation, coat protein release, and/or fusion to target membranes (36). At least two more enzymes that are involved in lipid metabolism, namely acyl-CoA synthetase-1 (14) and l-3-hydroxyacyl-CoA dehydrogenase (37), have been suggested to play a role in GLUT4 regulation. How at the molecular level the endosomal lipid metabolism catalyzed by these enzymes and p62/CE regulates GLUT4 recycling and recruitment is an important question that deserves systematic investigation.

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Association of Carboxyl Esterase with Facilitative Glucose Transporter Isoform 4 (GLUT4) Intracellular Compartments in Rat Adipocytes and Its Possible Role in Insulin-induced GLUT4 Recruitment

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