Adipose differentiation is accompanied by changes in cellular morphology, a dramatic accumulation of intracellular lipid and activation of a specific program of gene expression. Using an mRNA differential display technique, we have isolated a novel adipose cDNA, termed adipoQ. The adipoQ cDNA encodes a polypeptide of 247 amino acids with a secretory signal sequence at the amino terminus, a collagenous region (Gly-X-Y repeats), and a globular domain. The globular domain of adipoQ shares significant homology with subunits of complement factor C3q, collagen α1(X), and the brain-specific factor cerebellin. The expression of adipoQ is highly specific to adipose tissue in both mouse and rat. Expression of adipoQ is observed exclusively in mature fat cells as the stromal-vascular fraction of fat tissue does not contain adipoQ mRNA. In cultured 3T3-F442A and 3T3-L1 preadipocytes, hormone-induced differentiation dramatically increases the level of expression for adipoQ. Furthermore, the expression of adipoQ mRNA is significantly reduced in the adipose tissues from obese mice and humans. Whereas the biological function of this polypeptide is presently unknown, the tissue-specific expression of a putative secreted protein suggests that this factor may function as a novel signaling molecule for adipose tissue.

Adipose tissue is highly specialized to play important roles in energy storage, fatty acid metabolism, and glucose homeostasis (1, 2). Adipocytes synthesize and store triglyceride in periods of nutritional abundance and mobilize the lipids in response to fasting (2, 3). Fat tissue is also involved in regulating blood glucose levels through the expression of the insulin-responsive glucose transporter, Glut4 (4, 5). Fat and muscle, in fact, constitute the two major sites for insulin-regulated glucose uptake.

At a molecular level, many genes involved in lipid metabolism and glucose homeostasis are prominently expressed in fat (1). These include fatty acid synthase (6), the fatty acid binding protein aP2 (7, 8), lipoprotein lipase (9), phosphoenolpyruvate carboxykinase (10), malic enzyme (11), glyceraldehyde-3-phosphate dehydrogenase (12), and Glut4 (4, 10). Receptors for lipogenic or lipolytic hormones such as insulin (13, 14), insulin-like growth factor 1, which is expressed in many tissues during development and plays an important role in cell proliferation (18). In adipocytes, however, insulin-like growth factor 1 is found to stimulate cell differentiation (19). More interestingly, insulin-like growth factor 1 is synthesized by adipocytes in response to growth hormone stimulation (20), thus potentially functioning in an autocrine or paracrine fashion to promote adipogenesis during development. Another signaling molecule from adipose tissue is TNF-α. TNF-α is secreted from fat, especially in obesity, and acts in an autocrine or paracrine manner to interfere with insulin action in fat and muscle (21, 22). The recent cloning and characterization of the ob gene product has further illustrated that adipose tissue secretes signaling molecules that function in an endocrine fashion (23). The ob gene product (leptin) is secreted from fat into the circulation and acts to regulate body weight, perhaps via a putative receptor in the cerebroventricular region of the brain (15, 23, 24). Hence, molecules secreted from adipose tissue are capable of modulating diverse functions in fat and other tissues, thus representing a new facet of adipose tissue physiology.

In this study, we have used mRNA differential display to do a novel adipose gene termed adipoQ. Sequence analysis suggests that adipoQ is a secreted protein that shares significant homology to subunits of complement factor C3q and contains a collagenous structure at the NH2 terminus and a globular domain at the COOH terminus. The expression of this novel gene is highly regulated during the adipose differentiation process and is expressed predominantly in adipose tissue in vivo. Moreover, a significant down-regulation in adipoQ mRNA was observed in fat tissues from obese mice and humans. Our results provide a potentially valuable new molecular tool to explore the physiology of adipose tissue in normal and pathological states.

**EXPERIMENTAL PROCEDURES**

Materials—DMEM, calf serum, and fortified calf serum were from HyClone, Inc. Insulin, dexamethasone, and isobutylmethylxanthine were purchased from Sigma, Inc. Reverse transcriptase (Moloney murine leukemia virus), Klenow fragment and T4 kinase were obtained from BRL, Inc. Isotopes including [32P]dCTP (6000 Ci/mmol) and 35S-dATP (1000 Ci/mmol) were from DuPont NEN. GenAMP kit containing Taq DNA polymerase was purchased from Perkin-Elmer.

Cell Lines and Cell Culture—Murine fibroblastic 3T3-C2 cells and 3T3-F442A and 3T3-L1 preadipocytes were cultured as described (7, 25). Induction of adipocyte differentiation was performed essentially as described (26). Briefly, differentiation was initiated by administration of insulin at 5 μg/ml at confluence for 3T3-F442A cells and dexamethasone (1 μm), isobutylmethylxanthine (0.25 mm), and insulin (5 μg/ml) for 3T3-L1 cells. For 3T3-L1 cells, cells were treated with dexamethasone/isobutylmethylxanthine/insulin mix for 48 h and then were refed by DMEM medium containing 10% fetal calf serum and 5 μg/ml insulin.

1 The abbreviations used are: TNF-α, tumor necrosis factor; DMEM, Dulbecco's modified Eagle's medium; PCR, polymerase chain reaction.
Using this protocol, more than 90% of the cells in both cell lines acquire an adipocyte morphology 5-7 days after the initiation of differentiation. Culture medium was routinely changed every 2 days, and adipocyte differentiation was examined visually under the microscope.

mRNA Differential Display—mRNA differential display was performed essentially as described (27, 28). Briefly, total cellular RNA was isolated from 3T3-F442A adipocytes and differentiated 3T3-F442A adipocytes using the guanidine isothiocyanate extraction (29). 50 μg of total RNA was then treated with 20 units of RNase free-DNase (BRL, Inc.). Subsequently 0.2 μg of treated RNA was used in a reverse transcription reaction using each of the four 1-base-pair-anchored 3’ oligo(dT) primers (30) and 300 units of Mu-MLV reverse transcriptase (20 μl volume as recommended by the manufacturer). 2 μl of the reverse transcribed cDNA was used for each PCR reaction. PCR reaction was performed using the same 1-base-pair-anchored 3’ oligo(dT) primer and 10’5 arbitrary oligos of 10 nucleotides in length. The sequence of the 5’ arbitrary oligonucleotide that gave DD1 PCR product (see text) is 5’-AGTCATAC-5’. The 50-μl PCR reaction contained 10 mM Tris-HCl, pH 8.3, 50 mM KCl, 1.5 mM MgCl2, 0.01% gelatin, 2 μM dNTP (except dATP), 1 μl of α-32P-dATP (1300 Ci/mmol), 2 μM of 5’ arbitrary oligo and 3’ anchored dT oligo, and 0.2 μl of Taq polymerase. Parameters for PCR were 30 cycles of denaturing at 95 °C for 30 seconds, annealing at 40 °C for 1 min, and extension at 72 °C for 30 seconds. 5 μl of the PCR reaction mixture was loaded on a 8% sequence gel, and differentially amplified PCR fragments were visualized by exposing the dried sequence gel to x-ray film. Candidate PCR products were excised from the sequencing gel, and the DNA was eluted from the gel slices by boiling the gel slice in TE (10 mM Tris, pH 7.5, 1 mM EDTA) buffer for 10 min. The eluted DNA fragment was re-amplified by using the same primer pair and subsequently cloned into the TA cloning vector (Invitrogen, Inc.).

Library Screening, cDNA Cloning, and Sequencing—The cDNA library screening, restriction fragment analysis, subcloning, and sequencing analysis were performed as described (31). An adipocyte-specific λZAP II cDNA library was custom made by Stratagene Inc. as described (26). GenBank data base searches were performed using the Eugene program at the computer service in Dana-Farber Cancer Institute and overall homology, we termed this novel protein AdipoQ. The flag-epitope (DYKDDDDK, Kodak Scientific, Inc.) was incorporated into the COOH terminus. The NIH-3T3 fibroblasts were transient transfected with pSV-sport-flag-adipoQ as described (31). 24 h after transfection, cells were washed with phosphate-buffered saline, and DMEM medium with no serum was added. The number of candidate α-32P-dATP-labeled PCR products visualized in sequencing gels were expressed preferentially in mature adipocytes (data not shown), and we focused on one such product, DD1 (Fig. 1A). A partial cDNA clone for DD1 was obtained by PCR re-amplification (see “Experimental Procedures”) and sequenced. No significant sequence homology with any other genes in GenBank was apparent from this 200-base pair fragment. However, putative polyadenylation signals were present in this short nucleotide sequence. Northern analysis using this cDNA fragment revealed a mRNA expressed predominantly in differentiated fat cells (Fig. 1B). These data suggested that this cDNA fragment reflected a genuine mRNA species that was differentially regulated. A full-length clone of DD1 was subsequently obtained by screening a λZAP II cDNA adipocyte library with the partial cDNA clone. Sequence analysis revealed a single open reading frame in the full-length cDNA clone (Fig. 2A).

Analysis of the putative protein sequence identified a hydrophobic leader from amino acid residues 2 to 17, presumably representing a signal peptide. A region of collagenous repeats (Gly-X-Y) was present from amino acid 45 to 110, with 22 individual Gly-X-Y repeats. Comparisons with genes in GenBank identified several regions of homology to the subunits (A, B, and C chains) of complement factor C1q (36, 37), a tissue-specific collagen α1(X) (38), and a brain-specific protein cerebellin (39). The identity with the C1q chains is approximately 31% in the globular COOH-terminal region (Fig. 2B), with the homology localized primarily in two segments of unchanged, hydrophobic regions (F1 and F2). In addition, adipoQ and C1q A, B, and C chains have a similar size of 240-250 amino acids (Fig. 2B). The number of Gly-X-Y repeats is similar as well, with 22 such repeats for adipoQ and 26-29 for the C1q chains. The similarity of this protein to collagen α1(X) and cerebellin is found mainly at the COOH-terminal globular domain (Fig. 2C) with 38 and 25% identity over a 130-amino acid region. Collagen α1(X), however, encodes a much larger protein (680 amino acids) with a long collagenous segment (154 Gly-X-Y repeats). Cerebellin, on the other hand, is a smaller polypeptide with 193 amino acid residues and does not contain a collagenous domain (39). Because of the similarity between this novel protein and all three components of C1q molecules in size, domain structure, and overall homology, we termed this novel protein adiponectin.
Novel Adipose-specific Gene

Expression of AdipoQ mRNA from Mouse and Human—To examine the tissue distribution of adipoQ mRNA, we performed Northern analysis using various tissue RNAs from both mouse and rat (Fig. 4, A and B). A single abundant mRNA species was present in mouse adipose tissue and very little adipoQ mRNA could be detected in other tissues (Fig. 4A). AdipoQ mRNA is at least 50–100-fold more abundant in adipose tissue than in any other tissues examined in mice. The distribution of adipoQ mRNA in rat is also highly restricted to adipose tissue (Fig. 4B). Interestingly, in rat three distinct adipoQ mRNAs of 2.5, 1.8, and 1.2 kilobases in size were detected, and all three mRNAs were adipose-specific. Whether these three distinct rat mRNA species encode the same or slightly different proteins remains to be determined. A single 4-kilobase adipoQ mRNA can also be detected in a human fat sample using mouse adipoQ cDNA as a probe (Fig. 4C). The expression of adipoQ mRNA in adipose tissue is highly restricted to mature fat cells in rat, and little or no expression was detected in the stromal-vascular fraction isolated from fat pads (Fig. 4B, lanes 10 and 11). This is consistent with the increased adipoQ expression observed during adipocyte differentiation in established cell lines.

Expression of AdipoQ in Lean and Obese Adipose Tissues from Mouse and Human—To investigate whether adipoQ gene expression is altered in obesity, we examined adipoQ mRNA levels in adipose tissue samples from lean (ob/+) and obese (ob/ob) mice. Fat samples from obese and lean human individuals were also examined. As shown in Fig. 5A, a large (70–90%) increase in adipoQ expression was detected in obese fat compared with lean fat (Fig. 5A, lane 4 vs. lane 2). The expression of adipoQ mRNA was also increased in fat samples from obese human individuals compared with lean human individuals (Fig. 5B, lanes 4 vs. 2). These results suggest that adipoQ expression is upregulated in obesity.

The expression of adipoQ mRNA in adipose tissue is highly regulated. This kinetics is similar to or slightly later than that of the ap2 mRNA and parallels the expression of adipin mRNA (data not shown). It is also worth noting that the adipoQ mRNA is a very abundant message and can be readily detected in total RNA.

**Expression of AdipoQ in Lean and Obese Adipose Tissues from Mouse and Human—To investigate whether adipoQ gene expression is altered in obesity, we examined adipoQ mRNA levels in adipose tissue samples from lean (ob/+) and obese (ob/ob) mice. Fat samples from obese and lean human individuals were also examined. As shown in Fig. 5A, a large (70–90%) increase in adipoQ expression was detected in obese fat compared with lean fat (Fig. 5A, lane 4 vs. lane 2). The expression of adipoQ mRNA was also increased in fat samples from obese human individuals compared with lean human individuals (Fig. 5B, lanes 4 vs. 2). These results suggest that adipoQ expression is upregulated in obesity.**
Fig. 2. Nucleotide and deduced amino acid sequences of adipoQ: homology to components of C1q, collagen α1(X), and cerebellin.

A, nucleotides are numbered from the 5’ end of the sequence. The amino acid sequence is derived from the longest open reading frame. The **bold** region indicates the collagen-like domain. Putative polyadenylation signals are **underlined**. The accession number for adipoQ in GenBank is U49915.

B, homology of adipoQ with murine C1q. The **bold** region indicates identical amino acids in all four sequences. Two interruptions in collagenous region of C1q-B and C1q-C chains are indicated by an *asterisk* (see text). Symbols # and ‡ mark the conserved cysteines.

C, intron/exon structure of the adipoQ gene. Apparent size of introns and exons corresponds to the genomic sequence. Intron and exon boundaries are indicated by a bar and a box, respectively.

D, T7 and T3 transcriptional vectors (sizes are indicated in kb).

E, in vitro, CM, and Cell lysis of flag-adipoQ vector.

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**Novel Adipose-specific Gene**
reduction in adipoQ mRNA expression was observed in fat tissue from the obese mice. In contrast, the expression of another adipose-specific gene, aP2, was not affected by obesity (Fig. 5A). A more dramatic reduction (>50-fold) in adipins mRNA expression was observed in the same mouse samples (Fig. 5A), in agreement with published results (49). We also examined adipoQ expression in fat samples from four obese (BMI = 39 ± 1.4) and three normal human individuals (BMI = 21 ± 0.3) (Fig. 5B). A reduction of 50–80% in adipoQ mRNA was observed in obese human fat tissue samples (Fig. 5B, lanes 1–4) as compared with the normal controls (Fig. 5B, lanes 5–7). Thus, expression of adipoQ mRNA is clearly dysregulated in obesity of both mouse and humans.

**DISCUSSION**

Adipose tissue was traditionally thought to be a relatively passive depot for lipid storage and mobilization and was viewed to be solely at the receiving end of hormonal and neuronal signals. Consistent with this, receptors for hormones such as insulin, adrenocorticotropic hormone, and epinephrine are passively linked to adipose tissue. This cytokine is produced by adipose cells in vivo and in vitro (1). However, recent investigations suggest that fat tissue is much more actively involved in the energy balance system by secreting molecules that signal to and perhaps regulate the functions of other tissues and organs (3, 15, 23). One clear example of this is the production of TNF-α by adipose tissue. This cytokine is produced by fat cells mainly in the context of animal and human obesity (21). It interferes with insulin action in both muscle and fat and plays a major role in systemic insulin resistance, at least in part through a reduction in the tyrosine kinase activity of the insulin receptor (50). Another example of this is the recently cloned obese (ob) gene product. The ob protein is synthesized mainly by adipocytes and is secreted into the circulation. Injection of this protein indicates that it influences (directly or indirectly) food intake and thermogenesis (15, 24, 51). Another secreted molecule from adipose tissue with signaling potential is adipin. Originally identified as an adipocyte-specific serine protease (52), adipin has been shown to encode a critical component of the alternative complement pathway (factor D) (53). Moreover, the proximal part of this complement pathway is shown to be activated in adipose tissue (54), generating small bioactive molecules such as the anaphylatoxin C3a that could affect systemic functions. Most recently, C3a has been shown to regulate triglyceride synthesis in fibroblasts and adipocytes (55). These data suggest that many important physiological functions may be controlled through secreted proteins from adipose tissue.

The adipoQ molecule identified in this study has several features that suggest that it could function as a signaling molecule from adipocytes. First, adipoQ contains a hydrophobic signal peptide sequence and is homologous to several secreted proteins such as C1q A, B, and C chains, collagen α1(X), and cerebellin. Consistent with this, adipoQ is secreted from fibroblasts after transfection of a expression vector. Second, the expression of adipoQ is highly regulated during differentiation and is restricted to adipose tissue in vivo. Finally, the expression of adipoQ is affected by obesity in rodents and humans, suggesting a dysregulation in this pathological state. These properties closely parallel those of other important signaling molecules secreted from adipose tissue including the ob gene product and TNF-α.

Given these unique sequence features and expression patterns, it is tempting to speculate on the possible functions of adipoQ. The sequence homology with C1q provides a possible clue. C1q is the first component of the classical complement activation pathway (46). It is composed of three homologous subunits: the A, B, and C chains. Each chain has a NH2-
terminal collagenous segment (Gly-X-Y repeats) of 78–84 amino acids and a globular carboxyl region of approximately 130 amino acids. A functional C1q molecule contains six sub-units of each chain heteroligomerized along the collagenous helix. C1q interacts with the aggregated IgGs and initiates the complement cascade by proteolytically activating factors C2 and C4 (56). However, recent evidence suggests that C1q can also regulate other functions such as cell-mediated cytotoxicity (57), phagocytosis (58), chemotaxis (59), and interleukin-1 production (60) via a receptor-mediated mechanism. A putative receptor for C1q has been isolated and characterized in several human and murine cells including macrophages, lymphocytes, and fibroblasts (61). The collagenous region of C1q has been shown to be important for ligand-receptor interaction (62, 63). AdipoQ and C1q share significant similarities in the structure of this domain. Thus, it is possible that adipoQ could bind to the same or a similar receptor, thereby eliciting a biological response.

It is also possible that adipoQ may participate in the complement activation processes. Surrogate complement activation has been previously shown to be achieved by mannan-binding protein, a carbohydrate binding protein with a domain structure similar to that of C1q (64–66). However, because adipoQ lacks several key cysteines (see “Results”) in the regions that are important for C1q function, it is not clear whether adipoQ functions in complement system. Further experiments will be needed to address this issue.

The identification of adipo-Q, a novel adipose tissue-specific protein, poses many questions regarding its molecular and biochemical properties that are yet to be examined. More importantly, its biological role in adipose tissue and in the overall energy balance systems remain to be defined. The production and purification of this novel protein should open a new avenue to studying adipose tissue physiology in normal and pathological states.

While this manuscript was under review, Scherer et al. (67) identified a adipocyte-specific protein named Acrp30 using a

![Fig. 4. Expression of adipoQ mRNA in various tissues from mice, rats, and humans. A, 10 μg of total RNA from different mouse tissues was analyzed by Northern blot. Tissues are designated as follows: B, brain; Fa, fat; H, heart; I, intestine; K, kidney; L, liver; M, muscle; P, pancreas; S, spleen. The blot was sequentially hybridized to the adipoQ and the aP2 DNA probes. B, expression of adipoQ mRNA in rat tissues. 10 μg of total RNA from different rat tissues was analyzed by Northern blot. Tissue designation was identical to A, with two additional samples: mature fat cell from rat fat pads (floaters, FL), and the stromal-vascular fraction of rat fat pads (SV). C, comparison of adipoQ mRNA expression in mice, rats, and human fat. 10 μg of total fat tissue RNAs were analyzed by Northern blot. M, mouse; R, rat; H, human fat. Ethidium bromide (EtBr) stainings were used to normalize RNA loading.](http://www.jbc.org/)

![Fig. 5. Expression of adipoQ mRNA in lean and obese fat samples from mice and humans. A, 10 μg of total RNA from lean (ob+/+)

lanes 2 and 3) and obese (ob/ob) mice fat pads (lanes 1) was analyzed by Northern blot. The same blot was hybridized with probes for adipoQ, adipisin, and aP2. B, 10 μg of total fat RNA from three lean human individuals (lanes 5–7) and four obese individuals (lanes 1–4) was analyzed for adipoQ expression. Expression of the TNF receptor type 1 (TNFR1) mRNA was not altered in these conditions and was used as a control for RNA loading (22).](http://www.jbc.org/)
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random cDNA sequencing approach. AdipoQ is identical to Acrp30 in protein sequence.

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