

The Human Obesity Gene Map: The 2005 Update

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Abstract

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This paper presents the 12th update of the human obesity gene map, which incorporates published results up to the end of October 2005. Evidence from single-gene mutation obesity cases, Mendelian disorders exhibiting obesity as a clinical feature, transgenic and knockout murine models relevant to obesity, quantitative trait loci (QTL) from animal cross-breeding experiments, association studies with candidate genes, and linkages from genome scans is reviewed. As of October 2005, 176 human obesity cases due to single-gene mutations in 11 different genes have been reported, 50 loci related to Mendelian syndromes relevant to human obesity have been mapped to a genomic region, and causal genes or strong candidates have been identified for most of these syndromes. There are 244 genes that, when mutated or expressed as transgenes in the mouse, result in phenotypes that affect body weight and adiposity. The number of QTLs reported from animal models currently reaches 408. The number of human obesity QTLs derived from genome scans continues to grow, and we now have 253 QTLs for obesity-related phenotypes from 61 genome-wide scans. A total of 52 genomic regions harbor QTLs supported by two or more studies. The number of studies reporting associations between DNA sequence variation in specific genes and obesity phenotypes has also increased considerably, with 426 findings of positive associations with 127 candidate genes. A promising

observation is that 22 genes are each supported by at least five positive studies. The obesity gene map shows putative loci on all chromosomes except Y. The electronic version of the map with links to useful publications and relevant sites can be found at <http://obesitygene.pbrc.edu>.

Key words: human obesity gene map, association, linkages, Mendelian disorders, quantitative trait loci, candidate genes

Introduction

This paper represents the 12th in a series (1–11) on the status of the human obesity gene map, the 11th report published in *Obesity*. As in previous reports, we reviewed the literature published up to the end of October 2005 searching for the relevant publications through a variety of sources: PubMed using a combination of key words, authors, and journals; continuous reviews of obesity and genetics journals; personal collection of reprints; and papers made available to us by colleagues from around the world. Publications dealing with a wide variety of phenotypes pertaining to obesity, such as BMI, body fat mass, percentage of body fat, abdominal fat, fat-free mass, skinfolds, resting metabolic rates, plasma leptin levels, and other components of fat distribution and energy balance, were retained. As in previous reports, negative findings are not systematically reviewed but are briefly introduced when such data were available to us.

Each collaborating author was assigned one section of the report for an in-depth review. In addition to an introduction and a brief discussion (C.B.), the report includes sections dealing with monogenic obesity cases (G.A.), Mendelian disorders exhibiting obesity as clinical feature (J.W.), murine gene-deficient [knockout (KO)¹/flox], transgenic models in which altered expression of a gene (or genes) results in phenotypes relevant to obesity and quantitative

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¹ Nonstandard abbreviations: KO, knockout; QTL, quantitative trait locus; *MC4R*, melanocortin receptor 4; BDNF, brain-derived neurotrophic factor; *NTRK2*, neurotrophic tyrosine receptor kinase 2; AHO, Albright Hereditary Osteodystrophy; BW, body weight; MGI, Mouse Genome Informatics; LOD, logarithm of the odds ratio; WHR, waist-to-hip ratio; cM, centimorgan(s); WC, waist circumference.

trait loci (QTL) from murine models (A.Z.), QTLs from other animal model studies and gene-drug interactions (Y.C.), association studies in humans with specific candidate genes (T.R.), and human linkage studies including genome scans performed to identify QTLs of obesity or obesity-related phenotypes (L.P.). The other collaborating author (B.W.) is involved in the management of the database, the generation of the tables and the map from the database, and the electronic version of the human obesity gene map (<http://obesitygene.pbr.edu>). Readers are referred to previous publications (9,11) for detailed information on the electronic version of the map and on browsing and querying capabilities of the online Obesity Gene Map Database.

As in the past, the published references for each entry in the current human obesity gene map are provided for convenience. We are using gene symbols and chromosomal locations given in the Entrez Gene database (<http://www.ncbi.nlm.nih.gov/>) available from the National Center for Biotechnology Information. The appendix provides a complete list of genes and map locations cited in this paper.

Although the authors have taken every possible effort to provide correct information, in the rapidly changing world of genetics and bioinformatics and the ever-present world of human fallibility, it is almost inevitable that inaccuracies will emerge. The full responsibility for errors is ours. Furthermore, we seek your indulgence in errors of omission and hope you will notify us of any oversights. All correspondence to maximize the precision and quality of the map is welcomed and, indeed, solicited.

Sadly, we have to inform the readership that this is likely to be the last time that we are able to publish the review of the human obesity gene map. We have tried unsuccessfully to obtain the funding to support the enormous amount of work that is necessary every year to prepare this popular review. The printed version of the map in *Obesity* is highly cited, and the e-version is accessed ~200,000 times a year by ~40,000 unique users based mainly in academic institutions and pharmacological or biotechnology companies. Although we recognize that the yearly review in its printed and electronic versions is a valuable tool for those involved in this field, the project has become too large to be handled solely by us without support staff.

Monogenic Effects and Mendelian Disorders

Monogenics Section

The majority of disorders previously summarized in Table 2 have now been associated with a candidate gene or a genetic defect. Therefore, this year they are being merged with the monogenic obesity cases into a new table, Table 1.

This year, there has been relatively nominal reporting of monogenic cases of obesity. The majority of the monogenic obesity cases remain those with a genetic defect (mutation,

deletion, or insertion) in the melanocortin receptor 4 (*MC4R*) gene. Table 1 summarizes all of the cases that were reported in previous years. A publication by Farooqi and O'Rahilly (12) elegantly summarizes cases of monogenic obesity that received treatment for the mutated gene that resulted in improvement of the health status of the patients. These cases were covered in the 2004 Obesity Gene Map report. The same group recently described a new rare mutation in the receptor of the neurotrophin brain-derived neurotrophic factor (*BDNF*) gene, *TrkB* (13).

Neurotrophic Tyrosine Receptor Kinase 2 (NTRK2)

In humans, the receptor of the murine BDNF gene, *TrkB*, is encoded by the *NTRK2* gene. A study was reported by Yeo and colleagues (13) whereby a de novo heterozygous mutation arose in a child with severe early-onset obesity and hyperphagia. The A-to-G transition resulted in amino acid substitution of the tyrosine residue at position 722 by a cysteine (Y722C) (Table 1). An additional cohort of 192 alleles and the proband's parents were screened for the presence of this rare mutation, but nobody was found to carry it. In vitro functional studies showed that the mutation impaired activation of MAPK when cells were treated with BDNF (13). This new rare mutation provides another example of single-gene mutations in genes involved in energy balance regulation that result in severe and early onset obesity. In another preliminary study of 288 individuals with a history of early onset obesity, five missense mutations were identified in *NTRK2* (A74T, I98V, M354V, P660L, T821L) that have yet to be functionally characterized and described in greater detail (13).

Mendelian Disorders

Since last year's review, there has been limited development in the area of Mendelian disorders related to obesity, although many novel mutations in known genes have been reported. Updated references on new mutations for the Albright hereditary osteodystrophy (AHO), Bardet-Biedl, Berardinelli-Seip congenital lipodystrophy, Borjeson-Forssman-Lehmann, familial partial lipodystrophy, multiple endocrine neoplasia (type 1), and WAGR syndromes are provided (see Table 1).

In the present review, we now properly report AHO in the context of all disorders related to parathyroid hormone resistance, as described by DeSanctis et al. (131). To date, the AHO phenotype is always associated with mutations in *GNAS1*. In the AHO-like syndrome linked to 2q37, a French group narrowed down the critical region to a 4-megabase-pair interval delimited by D2S2338 (present) and D2S2253 (deleted) (149).

A new mutation was discovered for familial partial lipodystrophy, Dunnigan type (167). The affected 21-year-old woman had a great excess of subcutaneous fat on the face, neck, trunk, and abdomen, with relative lack on the gluteal region, arms, and legs. She was insulin resistant and had the

Table 1. Single-gene and obesity-related Mendelian disorders

OMIM no.	Syndrome	Locus	Candidate gene	Reference
Single-gene mutations with an obesity phenotype				
122561	Corticotropin-releasing hormone receptor 1	17q12-q22	<i>CRHR1</i>	(14)
602034	Corticotropin-releasing hormone receptor 2	7p14.3	<i>CRHR2</i>	(14)
601751	G-protein-coupled receptor 24	22q13.2	<i>GPR24</i>	(15)
164160	Leptin (obesity homolog, mouse)	7q31.3	<i>LEP</i>	(16–20)
601007	Leptin receptor	1p31	<i>LEPR</i>	(21)
601665	Melanocortin 3 receptor	20q13.2-q13.3	<i>MC3R</i>	(22–24)
155541	Melanocortin 4 receptor	18q22	<i>MC4R</i>	(25–47)
600456	Neurotrophic tyrosine kinase receptor type 2	9q22.1	<i>NTRK2</i>	(12,13)
176830	Proopiomelanocortin (adrenocorticotropin/ β -ipotropin/ α -melanocyte stimulating hormone/ β -melanocyte stimulating hormone/ β -endorphin)	2p23.3	<i>POMC</i>	(48,49)
162150	Proprotein convertase subtilisin/kexin type 1	5q15-q21	<i>PCSK1</i>	(50,51)
603128	Single-minded homolog 1 (<i>Drosophila</i>)	6q16.3-q21	<i>SIM1</i>	(52,53)
Autosomal recessive				
203800	Alstrom syndrome	2p13.1	<i>ALMS1</i>	(54–59)
209901	Bardet-Biedl syndrome 1	11q13.1	<i>BBS1</i>	(60–66)
606151	Bardet-Biedl syndrome 2	16q13	<i>BBS2</i>	(61,63,67–70)
600151	Bardet-Biedl syndrome 3	3p13-p12	<i>BBS3</i> (<i>ARL6</i>)	(63,71–75)
600374	Bardet-Biedl syndrome 4	15q22.3-q23	<i>BBS4</i>	(61,76–81)
603650	Bardet-Biedl syndrome 5	2q31	<i>BBS5</i>	(63,82–84)
604896	Bardet-Biedl syndrome 6	20p12.2	<i>MKKS</i>	(63,68,71,85–88)
607590	Bardet-Biedl syndrome 7	4q27	<i>BBS7</i>	(67,89)
608132	Bardet-Biedl syndrome 8	14q32.1	<i>BBS8</i>	(89,90)
269700	Berardinelli-Seip congenital lipodystrophy 1	9q34.3	<i>AGPAT2</i>	(91–95)
606158	Berardinelli-Seip congenital lipodystrophy 2	11q13	<i>BSCL2</i>	(92,94,96–98)
212065	Carbohydrate-deficient glycoprotein syndrome type 1a	16p13.2	<i>PMM2</i>	(99)
216550	Cohen syndrome	8q22.2	<i>COH1</i>	(100–105)
601538	Combined pituitary hormone deficiency	5q35.3	<i>PROPI</i>	(106–108)
227810	Fanconi-Bickel syndrome	3q26.31	<i>SLC2A2</i>	(109–117)
139191	Isolated growth hormone (GH) deficiency	7p14	<i>GHRHR</i>	(118–120)
Triallelic digenic				
138090	Cortisone reductase deficiency	1pter-p36.13	<i>H6PD</i>	(121)
604931	Cortisone reductase deficiency	1q32-q41	<i>HSD11B1</i>	(121,122)
Digenic				
600917	Severe insulin resistance with obesity	3p25 7q31.1	<i>PPARG</i> <i>PPP1R3A</i>	(123)
Autosomal dominant				
100800	Achondroplasia	4p16.3	<i>FGFR3</i>	(124–127)
103580	AHO (Pseudopseudohypoparathyroidism)	20q13.2-q13.3	<i>GNAS</i>	(128–147)
103581	AHO 2	15q11-q13	<i>AHO2</i>	(148)
600430	Brachydactyly mental retardation syndrome	2q37.3	<i>STK25</i> <i>GPC1</i> <i>GPR35</i>	(149–155)
105830	Angelman syndrome with obesity	15q11-q12	<i>ANCR</i>	(156)
605746	Anisomastia	16q13-q21	<i>ANMA</i>	(157)
160980	Carney complex with primary pigmented nodular adrenocortical disease and Cushing's syndrome (CNC1)	17q24.3	<i>PRKARIA</i>	(158–164)
605244	Carney complex with primary pigmented nodular adrenocortical disease and Cushing's syndrome (CNC2)	2p16		(165)
604367	Familial partial lipodystrophy, Dunnigan, type 3	3p25	<i>PPARG</i>	(166–169)
151660	Familial partial lipodystrophy, type 2 (Dunnigan type)	1q23.1	<i>LMNA</i>	(170–180)
147670	Insulin resistance syndromes	19p13.3-p13.2	<i>INSR</i>	(181–188)
139250	Isolated GH deficiency (139250)	17q22-q24	<i>GH1</i>	(189)
131100	Multiple endocrine neoplasia, type 1 with Cushing's disease	11q13	<i>MEN1</i> (<i>Menin</i>)	(190–196)
122000	Posterior polymorphous corneal dystrophy (chromosome 1)	1p34.3-p32.3	<i>COL8A2</i>	(197)
605020	Posterior polymorphous corneal dystrophy (chromosome 20)	20p11.21	<i>VSI1</i>	(198,199)
176270	Prader-Willi syndrome	15q11.2 15q11.2 15q11.2	<i>IPW</i> <i>MKRN3</i> <i>PWCR1</i>	(200–212,216–218,220)

Table 1. (continued)

OMIM no.	Syndrome	Locus	Candidate gene	Reference
		15q12	<i>SNRPN</i>	
		15q11.2	<i>MAGEL2</i>	
		15q11.2	<i>NDN</i>	
		15q11-q12	<i>GABRG3</i>	
603128	Prader-Willi-like syndrome (chromosome 6q)	6q16.3-q21	<i>SIM1</i>	(202,213–215, 219,220)
190160	Thyroid hormone resistance syndrome	3p24.1	<i>THRB</i>	(221)
181450	Ulnar-Mammary (Schinzel) syndrome	12q24.21	<i>TBX3</i>	(222–225)
194072	WAGR syndrome with obesity	11p13	<i>WT1</i>	(226–232)
		11p13	<i>PAX6</i>	
X linked				
301900	Borjeson-Forssman-Lehmann syndrome	Xq26.3	<i>PHF6</i>	(233–240)
303110	Choroideremia with deafness and obesity	Xq21.2	<i>CHM</i>	(241,242)
		Xq21.1	<i>DFN3</i>	
309550	Fragile X syndrome with Prader-Willi-like phenotype	Xq28	<i>FMR1</i>	(243–246)
300148	MEHMO syndrome	Xp22.13-p21.1	<i>MEHMO</i>	(247–249)
300218	Mental retardation X-linked, syndromic 7	Xp11.3-q22.1	<i>MRXS7</i>	(250)
300458	Mental retardation X-linked, syndromic 16	Xq28	<i>MECP2</i>	(251,252)
300238	Mental retardation, X-linked, syndromic 11	Xq26-q27	<i>MRXS11</i>	(253,254)
176270	Prader-Willi-like syndrome, X-linked	Xq23-q25	<i>PWLSX</i>	(255)
312870	Simpson-Golabi-Behmel 1	Xq26.2	<i>GPC3</i>	(82,256–264)
		Xq26.1	<i>GPC4</i>	
300209	Simpson-Golabi-Behmel 2	Xp22	<i>SGBS2</i>	(265)
309585	Wilson-Turner syndrome	Xq21.2-q22	<i>WTS</i>	(266,267)

metabolic syndrome and type 2 diabetes. She was heterozygous for a novel A>G mutation at position –14 of intron B, upstream of *PPARG* exon 1 within the promoter of the *PPARγ4* isoform, implicating this isoform as being potentially important in adipocyte biology.

Finally, in recent clinical reviews of large groups of Alstrom (58) and WAGR (229) syndrome patients, the central role of childhood obesity and hyperinsulinism in Alstrom syndrome was confirmed, as well as a significant prevalence of obesity (of 18%) in WAGR subjects. In this last syndrome, the new acronym WAGRO (obesity) has even been suggested (227).

Transgenics and KOs

The murine obesity gene map identifies 248 genes (Table 2) that, when mutated or expressed as transgenes in the mouse, result in phenotypes affecting body weight (BW) and adiposity. We include genes that promote obesity and genes that promote leanness, with the exception of genes that seem to promote failure-to-thrive phenotypes or mutant genes impacting developmental issues affecting multiple organs systems during embryogenesis or early growth. The list was compiled from the primary literature, accessible through PubMed and corroborated with information captured by the Mouse Genome Informatics (MGI) group (www.informatics.org). Official gene no-

menclature rules have been followed, even where the use of this nomenclature differs from the gene name used in the primary publication. We have attempted to capture common synonyms, but the list is not exhaustive. Readers are directed to MGI for a more complete list of synonyms and nomenclature history.

Of the new genes added to the list this year, three are imprinted. Maternal inheritance of the *Gnas* KO allele (400), a KO of the paternally expressed *Peg3* gene (493), and transgenic overexpression of the paternally expressed *Mest* (*Peg1*) in adipose tissue all promote obesity. Imprinted loci are well documented in the mouse genome, but the degree of imprinting can also be tissue dependent. Clearly, the role of imprinted genes in the development of obesity-related phenotypes must be considered in cases where simple Mendelian inheritance relationships seem uninformative. Three new genes listed for the first time this year are relevant to the molecular characterization of three well-known human obesity syndromes: Alstroms, Bardel-Biedl, and McKusick-Kaufman. The respective murine homologs, *Alms1*, *Bbs2*, and *Mkks*, all present obesity phenotypes when mutated in mice. Interestingly, *Bbs2*-deficient mice weigh less than controls at birth, suggesting an additional effect on early development. These three mutants will provide valuable model systems to study the roles of these genes in the development of these polygenic syndromes.

Table 2. Murine models of obesity

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
2(89)	<i>A^k</i>	20q11.2-q12	<i>ASIP</i>	Agouti. Expression limited to adipose cells	Transgene: aP2 promoter regulating expression of murine wild-type agouti cDNA	(268,269)
2(89)	<i>A^k</i>	20q11.2-q12	<i>ASIP</i>	Agouti, also known as BAP20 mouse. Ubiquitous expression. Unexpectedly high in skeletal muscle	Increased BW and fat mass Transgene: human β actin promoter regulating expression of murine wild-type agouti cDNA	(270)
2(89)	<i>A^k</i>	20q11.2-q12	<i>ASIP</i>	Agouti. Ubiquitous expression	Obesity Transgene: murine <i>Pgk1</i> promoter regulating expression of murine wild-type agouti cDNA	(270)
10(44)	<i>Abca7^g</i>	19p13.3	<i>ABCA7</i>	ATP-binding cassette, subfamily A (ABC1), member 7	Obesity Reduced fat and circulating high-density lipoprotein and total cholesterol in females	(271)
5	<i>Acacb^g</i>	12q24.1	<i>ACACB</i>	Acetyl CoA carboxylase β , also known as Acc2	Reduced adiposity; resistant to diet-induced obesity	(272,273)
11(38)	<i>Acadvl^g</i>	17p13-p11	<i>ACADVL</i>	Acyl-CoA dehydrogenase, very long chain	Lipid accumulation in myocytes; impaired temperature regulation	(274,275)
7(F4)	<i>Adam12^g</i>	10q26.3	<i>ADAM12</i>	A disintegrin and metallopeptidase domain 12 (meltrin α)	Adult-onset fat mass gain Moderate resistance to diet-induced obesity due to an impairment in the increase of the number of adipocytes in high-fat-fed mice	(276)
16(53.4)	<i>Adamts1^g</i>	21q21.2	<i>ADAMTS1</i>	A disintegrin-like and metalloprotease (reprolysin type) with thrombospondin type 1 motif, 1	Reduced BW and adiposity	(277)
17	<i>Adcyap1^g</i>	18p11	<i>ADCYAP1</i>	Adenylate cyclase activating polypeptide 1	Wasting; reduced adiposity	(278)
11(19)	<i>Adra1b^g</i>	5q23-q32	<i>ADRA1B</i>	Adrenergic receptor, $\alpha 1b$	Accelerated weight gain on high-fat diet	(279)
19(50)	<i>Adra2a^k</i>	10q24-q26	<i>ADRA2A</i>	Transgene expresses adrenergic receptor $\alpha 2$ in adipose cells	Transgene: aP2-driven human <i>ADRA2A</i> cDNA When expressed in <i>Adra3b</i> -deficient mice, leads to obesity. When expressed in mice heterozygous for <i>Adra3b</i> , there is no adipose phenotype	(280)
19(51)	<i>Adrb1^g</i>	10q24-q26	<i>ADRB1</i>	Adrenergic receptor, $\beta 1$	Obesity in conjunction with mutations in <i>Adrab2</i> and <i>Adrab3</i>	(281)
19(51)	<i>Adrb1^k</i>	10q24-q26	<i>ADRB1</i>	Transgene insertion 1, Susan R. Ross, expresses adrenergic receptor $\beta 1$ in adipose cells	Transgene: aP2-driven expression of human <i>ADRB1</i> cDNA Reduced adiposity and partially resistant to diet-induced obesity	(282)
18(34)	<i>Adrb2^g</i>	5q31-q32	<i>ADRB2</i>	Adrenergic receptor, $\beta 2$	Reduced adiposity Obesity in conjunction with targeted mutations in <i>Adrab1</i> and <i>Adrab3</i>	(281)
8(10)	<i>Adrb3^g</i>	8p12-p11.2	<i>ADRB3</i>	Adrenergic receptor, $\beta 3$	Increased body fat. Mildly obese on chow. High obesity on high-fat diet. Obesity in conjunction with mutations in <i>Adrab2</i> and <i>Adrab3</i>	(283)
8(10)	<i>Adrb3^g</i>	8p12-p11.2	<i>ADRB3</i>	Adrenergic receptor, $\beta 3$	Increased adiposity on chow or high-fat diets	(284)
11	<i>Aebp1^k</i>	7p13	<i>AEBP1</i>	AE-binding protein 1	Transgene; expressed in adipose tissue; obesity in females	(285)
8(D1-D2)	<i>Agryp^g</i>	16q22	<i>AGRP</i>	Agouti-related protein	Age-related lean phenotype	(286)
8(D1-D2)	<i>Agryp^k</i>	16q22	<i>AGRP</i>	Agouti-related protein; expressed ubiquitously	Transgene: β actin promoter regulating expression of human <i>AGRP</i> cDNA; elevated weight gain and obesity	(287)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
8(D1-D2)	<i>Agrp</i> ^k	16q22	<i>AGRP</i>	Agouti-related protein	Transgene. Post-embryonic deletion of <i>AGRP</i> -expressing neurons Lean	(288)
13(16)	<i>Agtr1a</i> ^g			Angiotensin II receptor, type 1a	Resistant to diet-induced obesity	(289)
X(12.5)	<i>Agtr2</i> ^g	Xq22-23	<i>AGTR2</i>	Angiotensin II receptor, type 2	Resistant to diet-induced obesity	(290)
16(15)	<i>Ahsg</i> ^g	3q27	<i>AHSG</i>	α -2-HS-glycoprotein	Resistant to diet-induced obesity	(291)
1(51.7)	<i>Akp3</i> ^g	2q37.1	<i>ALPI</i>	Alkaline phosphatase 3, intestine, not Mn requiring; also known as IAP	Accelerated weight gain on high-fat diet	(292)
12(57)	<i>Akt1</i> ^k	14q32.3	<i>AKT1</i>	Thymoma viral proto-oncogene 1	Transgene, cDNA of constitutively active Akt expressed in skeletal muscle from the human skeletal actin promoter Reduced in adiposity	(293)
7(6.5)	<i>Akt2</i> ^g	19q13.1-13.2	<i>AKT2</i>	Thymoma viral proto-oncogene 2	Reduction in adiposity, especially in young females. Age-related adipocyte loss in both sexes	(294)
6	<i>Alms1</i> ^g	2p13	<i>ALMS1</i>	Alstrom syndrome 1 homolog (human)	Gene trapped Obesity	(295)
15(B1)	<i>Amacr</i> ^g	5p13.2-q11.1	<i>AMACR</i>	α -methylacyl-CoA racemase	Reduction in BW and adiposity on phytol-supplemented diet	(296)
17	<i>Angptl4</i> ^g	19p13.3	<i>ANGPTL4</i>	Angiopietin like 4, also known as fasting-induced adipocyte factor (FIAF)	Reduction in body fat gain upon transfer from germ-free to conventional housing	(297)
9	<i>Angptl6</i> ^g	19p13.2	<i>ANGPTL6</i>	Angiopietin like 6, also known as adipopietin-related growth factor (AGF)	The 20% of mice that survive development manifest obesity and insulin resistance	(298)
9	<i>Angptl6</i> ^k	19p13.2	<i>ANGPTL6</i>	Angiopietin like 6, also known as adipopietin-related growth factor (AGF)	Transgenic: ubiquitous expression using the chicken β -actin promoter and cytomegalovirus (CMV)-enhancer Reduced adiposity on chow and resistance to diet-induced obesity on high-fat diets	(298)
7(4)	<i>Apoc1</i> ^k	19q13.2	<i>APOC1</i>	Apolipoprotein C1. transgene insertion 1, Louis M. Havekes-overexpressing human <i>APOC1</i> gene	Transgenic (Tg) mice expressing the human <i>APOC1</i> gene from its own promoter Moderate reduction in adiposity relative to non-Tg mice. When crossed with the Lep background, however, Tg mice were protected against obesity and insulin resistance	(299)
9(27)	<i>Apoc3</i> ^g	11q23.1-q23.2	<i>APOC3</i>	Apolipoprotein C-III	Obesity on high-fat diet	(300,301)
4(B1)	<i>Aqp7</i> ^g	9p13	<i>AQP7</i>	Aquaporin 7	Increased gonadal fat pad mass	(302)
4(B1)	<i>Aqp7</i> ^g	9p13	<i>AQP7</i>	Aquaporin 7	Adult-onset obesity	(303)
X(36)	<i>Ar</i> ^c	Xq11.2-q12	<i>AR</i>	Androgen receptor	Floxed gene + Cre transgene expressed from the cytomegalovirus promoter Obesity. Decreased energy expenditure	(304,305)
10(B5)	<i>Arid5b</i> ^g	10q21.2	<i>ARID5B</i>	AT-rich interactive domain 5B (Mrf1 like), also known as Mrf2	Reduced adiposity on chow. Resistant to diet-induced obesity	(306)
11(B4)	<i>Aspa</i> ^c	17pter-p13	<i>ASPA</i>	Aspartoacylase (aminoacylase) 2	Reduced adiposity	(307)
14(C3)	<i>Atp12a</i> ^g	13q12.12	<i>ATP12A</i>	ATPase, H ⁺ /K ⁺ -transporting, non-gastric, α polypeptide	Increased weight loss on potassium-free diet	(308)
18	<i>Atp8b1</i> ^f	18q21-q22	<i>ATP8B1</i>	ATPase, class I, type 8B, member 1	Targeted knock-in Increased weight loss on bile salt-supplemented diet	(309)
12	<i>Batf</i> ^k	14q24.3	<i>BATF</i>	Regulator of transcription factor B-ZIP	Loss of all adipose tissue	(310)
19	<i>Bbs1</i> ^g	11q13.1	<i>BBS1</i>	Bardet-Biedl syndrome 1 homolog (human)	KO due to gene trap insertion in exon 11 Reduced BW at birth. Obesity at 10 weeks in 10% of the mutants	(311)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
8	<i>Bbs2</i> ^g	16q21	<i>BBS2</i>	Bardet-Biedl syndrome 2 homolog (human)	Reduced BW at birth. Increased abdominal fat mass at 4 months	(312)
9(33)	<i>Bbs4</i> ^c	15q22.3-23	<i>BBS4</i>	Bardet-Biedl syndrome 4 homolog (human)	Low BW at weaning, adult-onset obesity after weaning	(311)
9(33)	<i>Bbs4</i> ^g	15q22.3-23	<i>BBS4</i>	Bardet-Biedl syndrome 4 homolog (human)	Low BW at weaning, adult-onset obesity after weaning	(81)
2(62)	<i>Bdnf</i> ^g	11p13	<i>BDNF</i>	BDNF. The mutation is homozygous lethal	Mature-onset obesity in heterozygotes. Can be treated by food restriction	(313,314)
X(A7.1)	<i>Brs3</i> ^g	Xq26-q28	<i>BRS3</i>	Bombesin-like receptor 3	Obesity	(315)
2	<i>Bub1b</i> ^g	15q15	<i>BUB1B</i>	Budding uninhibited by benzimidazoles 1 homolog, β (<i>S. cerevisiae</i>)	Age-dependent loss of body fat; reduced lifespan	(316)
17(34.3)	<i>C3</i> ^g	19p13.3	<i>C3</i>	Complement component 3; acylation-stimulating protein	Females possess a lean phenotype and are resistant to diet-induced obesity	(317,318)
13(D1)	<i>Cart</i> ^g	5q13.2	<i>CART</i>	Cocaine- and amphetamine-regulated transcript	Increased susceptibility to diet-induced obesity	(319,320)
6(A2)	<i>Cav1</i> ^g	7q31.1	<i>CAV1</i>	Caveolin 1	Decreased adiposity; resistant to diet-induced obesity	(321)
6(48.3)	<i>Cav3</i> ^g	3p25	<i>CAV3</i>	Caveolin 3	Increased adiposity	(322)
9(26)	<i>Cbl</i> ^g	11q23.3	<i>CBL</i>	Casitas B-lineage lymphoma, also known as c-cbl	Reduced adiposity	(323)
5(34)	<i>Cckar</i> ^g	4p15.1-15.2	<i>CCKAR</i>	Cholecystokinin (CCK) A receptor	Resistant to CCK-mediated inhibition of food intake but normal long-term weight regulation; increased cholesterol absorption on lithogenic diet; altered thermogenic regulation	(324–326)
17(28.8)	<i>Ccnd3</i> ^g	6p21	<i>CCND3</i>	Cyclin D3	Resistant to diet-induced obesity	(327)
5(2)	<i>Cd36</i> ^g	7q11.2	<i>CD36</i>	CD36 antigen/fatty acid translocase	Altered metabolic adaptation to dietary modulation	(328)
18(6)	<i>Cdh2</i> ^k	18q11.2	<i>CDH2</i>	Cadherin 2, also known as N-cadherin or Ncad. Truncated gene used in this construct acts as a dominant negative allele	Transgene: expressing truncated <i>Cdh2</i> using an osteoblast-specific promoter, <i>Og2</i>	(329)
17(15.2)	<i>Cdkn1a</i> ^g	6p21.2	<i>CDKN1A</i>	Cyclin-dependent kinase inhibitor 1A (P21)	Increased adiposity	(330)
6(62)	<i>Cdkn1b</i> ^g	12p13.1-p12	<i>CDKN1B</i>	Cyclin-dependent kinase inhibitor 1B (P27)	Increased adiposity	(330)
7(12)	<i>Cebpa</i> ^g	19q13.1	<i>CEBPA</i>	CCAAT/enhancer-binding protein (C/EBP), α	Reduced adiposity	(331)
7(12)	<i>Cebpa</i> ^d	19q13.1	<i>CEBPA</i>	CCAAT/enhancer-binding protein (C/EBP), α	KO + gene replacement. A <i>Cebpb</i> knock-in was generated by replacing the entire coding region of the <i>Cebpa</i> locus with the <i>Cebpb</i> coding region	(332)
2(95.5)	<i>Cebpb</i> ^g	20q13.13	<i>CEBPB</i>	CCAAT/enhancer-binding protein (C/EBP), β	Lean and resistant to diet-induced obesity	(333)
16(9)	<i>Cebp</i> ^g	8p11.2-11.1	<i>CEBPD</i>	CCAAT/enhancer-binding protein (C/EBP), Δ	Reduced adiposity	(334)
13(7)	<i>Chrm3</i> ^g	1q41-q44	<i>CHRM3</i>	Muscarinic receptor M3	Reduced adiposity	(335)
18	<i>Cidea</i> ^g	18p11.21	<i>CIDEA</i>	Cell death-inducing DNA fragmentation factor, α subunit-like effector A	Reduced adiposity and resistant to diet-induced obesity	(336)
5(43)	<i>Clock</i> ^b	4q12	<i>CLOCK</i>	Clock	ENU-generated mutant	(337)
4(13.9)	<i>Cnr1</i> ^c	6q14-q15	<i>CNR1</i>	Cannabinoid receptor 1 (brain), also known as CB1 receptor	Obesity Floxed gene + ubiquitously expressed Cre Reduced adiposity	(338)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
4(13.9)	<i>Cnr1</i> ^g	6q14-q15	<i>CNR1</i>	Cannabinoid receptor 1 (brain), also known as CB1 receptor	On standard chow at 20 weeks, the BWs and adiposity are 24% and 60% lower, respectively, than control mice	(339)
5	<i>Corin</i> ^g	4p13-12	<i>CORIN</i>	Corin	Resistant to diet-induced obesity	(340)
8(32.6)	<i>Cpe</i> ^g	4q32.3	<i>CPE</i>	Carboxypeptidase E	Increased BW KO: floxed and deleted	(341)
19(2)	<i>Cpt1a</i> ^g	11q13.1-13.2	<i>CPT1A</i>	Carnitine palmitoyltransferase 1a, liver	Obesity Homozygotes are lethal	(342)
3(8)	<i>Crh</i> ^k	8q13	<i>CRH</i>	Corticotropin-releasing factor hormone, also known as CRF Expression of the transgene, however, is restricted to endogenous Crh-expressing cells due to a tissue-specific enhancer present within the Crh cDNA sequence	Fasting hypoglycemia in heterozygotes Increased fasting serum free fatty acids Murine corticotropin-releasing hormone cDNA expressed from the mouse metallothionein promoter Transgenic mice exhibit elevated ACTH release, high circulating levels of CRH and adrenal corticosterone. They display excess fat accumulation and muscle atrophy	(343)
6(28)	<i>Crhr2</i> ^g	7p14.3	<i>CRHR2</i>	Corticotropin-releasing hormone receptor 2	Normal adiposity on low-fat diet. Lower feed efficiency on high-fat diet (higher food intake but same weight gain as wild-type mice)	(344)
9	<i>Cyb5r4</i> ^g	6pter-q22.33	<i>CYB5R4</i>	Cytochrome b5 reductase 4	Reduced adiposity, increased food intake, hyperglycemia and hypoinsulinemia at 7 weeks	(345)
9(31)	<i>Cyp19a1</i> ^g	15q21.1	<i>CYP19A1</i>	Cytochrome P450, family 19, subfamily a, polypeptide 1, also known as aromatase	Elevated gonadal fat pad weight; obesity prevented by cholesterol feeding	(346,347)
2(15.5)	<i>Dbh</i> ^d	9q34	<i>DBH</i>	Dopamine β hydroxylase. Dopamine-deficient (DD) mice are homozygous for this mutation and also for a KO of the endogenous tyrosine hydroxylase (Th) locus. The Th knock-in in the <i>Dbh</i> gene restores tyrosine hydroxylase activity	KO + gene replacement. A <i>Th</i> knock-in was generated by inserting the <i>Th</i> gene into the mutated <i>Dbh</i> locus These DD mice do not possess altered adiposity on chow diet. However, when the Th ^{-/-} and <i>Dbh</i> <tm2(Th)Rpa mutations are homozygous in mice that are also homozygous for the Lep ^{ob} mutation, there is a significant reduction in BW and adiposity gain	(348,349)
11	<i>del(17)(p11.2)</i> ^k	17p11.2	<i>del(17)(p11.2)</i>	Smith-Magenis syndrome	Transgenic: chromosomal rearrangement	(350)
15(46.9)	<i>Dgat1</i> ^g	8q24.3	<i>DGAT1</i>	Acyl CoA: diacylglycerol O-acyltransferase 1	Obesity Lean and resistant to diet-induced obesity	(351)
15(46.9)	<i>Dgat1</i> ^k	8q24.3	<i>DGAT1</i>	Acyl CoA: diacylglycerol O-acyltransferase 1	Transgenic; expressed in adipose cells	(352)
15(46.9)	<i>Dgat1</i> ⁱ	8q24.3	<i>DGAT1</i>	Acyl CoA: diacylglycerol O-acyltransferase 1	Fatty liver but no obesity on high-fat diet Adenovirus-mediated overexpression in liver	(353)
4(C7)	<i>Dhcr24</i> ^g	1p33-31.1	<i>DHCR24</i>	24-dehydrocholesterol reductase	Increased gonadal but not subcutaneous fat mass Reduction in subcutaneous and mesenteric fat	(354)
12(54)	<i>Dlk1</i> ^k	14q32.3	<i>DLK1</i>	Δ -like 1 homolog (<i>Drosophila</i>), also known as Pref-1. Expressed as fusion to human immunoglobulin- γ constant region in liver	Transgene. Murine cDNA expressed from albumin promoter Reduced adiposity	(355)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
12(54)	<i>Dlk1</i> ^k	14q32.3	<i>DLK1</i>	Δ -like 1 homolog (<i>Drosophila</i>), also known as Pref-1. Expressed as fusion to human immunoglobulin- γ constant region in adipocytes	Transgene. Murine cDNA expressed from aP2 promoter Reduced adiposity	(355)
14	<i>Dnajc3</i> ^g	13q32	<i>DNAJC3</i>	DnaJ (Hsp40) homolog, subfamily C, member 3	Increased food intake; decreased adiposity	(356)
1(H2)	<i>Dpt</i> ^e	1q12-23	<i>DPT</i>	Dermatopontin	Increased subcutaneous adipose number and volume	(357)
16(23.3)	<i>Drd3</i> ^g	3q13.3	<i>DRD3</i>	Dopamine receptor 3	Increased adiposity and diet-induced obesity	(358)
11	<i>dup(17)(p11.2)(p11.2)</i> ^k	17p11.2	<i>dup(17)(p11.2)(p11.2)</i>	Smith-Magenis syndrome	Transgenic: chromosomal rearrangement Obesity	(350)
8(8)	<i>Eif4ebp1</i> ^g	8p12	<i>EIF4EBP1</i>	Eukaryotic translation initiation factor 4E-binding protein 1	Reduced adiposity	(359)
5	<i>Ereg</i> ^g	4q13.3	<i>EREG</i>	Epiregulin	Increased weight loss with dextran sulfate sodium exposure	(360)
19(3)	<i>Esrra</i> ^g	11q13	<i>ESRRA</i>	Estrogen-related receptor α	Reduced BW and adiposity. Resistant to diet-induced obesity	(361,362)
3(13.9)	<i>Fabp4</i> ^g	8q21	<i>FABP4</i>	Fatty acid-binding protein 4, adipocyte	Like the control strain, homozygous mutants become obese on a high-fat diet but remain insulin sensitive	(363)
3(A1-A3)	<i>Fabp5</i> ^g	8q21.13	<i>FABP5</i>	Fatty acid-binding protein 5, adipocyte, also known as Mal1	Less adiposity than controls on high-fat diet	(364,365)
7(B2)	<i>Fgf21</i> ^k	19q31.1-qter	<i>FGF21</i>	Fibroblast growth factor 21	Transgene. Expressed human gene in liver Resistant to diet-induced obesity	(366)
2(H2)	<i>Fkhl18</i> ^g	20q11.1-11.2	<i>FKHL18</i>	Forkhead-like 18 (<i>Drosophila</i>)	Resistant to diet-induced obesity	(367)
7(5)	<i>FosB</i> ^k	19q13.32	<i>FOSB</i>	FBJ osteosarcoma oncogene B	Transgene: expressing Δ FosB, an alternative spliced mRNA variant of FosB under the control of the neuron-specific enolase promoter	(368,369)
2(84)	<i>Foxa2</i> ^g	20p11	<i>FOXA2</i>	Forkhead box A2, also known as Hnf3b or Tcf3b	Decreased fat mass KO + reporter Homozygous KO mice are embryonic lethal Heterozygotes rapidly develop obesity on a high-fat diet	(370)
8(65.5)	<i>Foxc2</i> ^k	16q22-q24	<i>FOXC2</i>	Forkhead Box C2 expressed in adipose cells	Transgene: human <i>FOXC2</i> cDNA expressed from the aP2 promoter Reduced adiposity (lipid content) on chow diet and resistance to diet-induced obesity	(371,372)
17(E5)	<i>Fshr</i> ^g	2p21	<i>FSHR</i>	Follicular-stimulating hormone receptor	Obesity	(373)
6	<i>Fxyd4</i> ^g	10q11.21	<i>FXYD4</i>	FXYD domain-containing ion transport regulator 4	Increased food intake but reduced BW	(374)
19(2)	<i>Gal</i> ^g	11q13.2	<i>GAL</i>	Galenin	Exaggerated obesity in NPY-deficient mice	(375)
10(43)	<i>Gamt</i> ^g	19p13.3	<i>GAMT</i>	Guanidinoacetate methyltransferase	Decreased adiposity	(376)
8(5)	<i>Gas6</i> ^g	13q34	<i>GAS6</i>	Growth arrest specific 6	Resistant to diet-induced obesity	(377)
11(60)	<i>Gast</i> ^g	17q21	<i>GAST</i>	Gastrin	Obesity	(378)
11(E2)	<i>Gcgl</i> ^g	17q25	<i>GCGR</i>	Glucagon receptor	Decreased white and brown adipose mass	(379)
11(1)	<i>Gck</i> ^k	7p15.3-p15.1	<i>GCK</i>	Glucokinase. Expressed in skeletal muscle	Transgene. Murine Gck cDNA expressed from the murine myosin light-chain 1 promoter Resistant to diet-induced obesity	(380)
11(1)	<i>Gck</i> ^k	7p15.3-p15.1	<i>GCK</i>	Glucokinase. Expressed in liver	Transgene. Murine Gck cDNA expressed from the murine Pepck promoter Increased weight gain on a high-fat diet	(381)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
6(60.6)	<i>Gdf3ⁱ</i>	12p13.1	<i>GDF3</i>	Growth differentiation factor 3	Adenovirus-mediated overexpression Increase in BW and adiposity on high-fat diet but no phenotype on chow	(382)
1(27.8)	<i>Gdf8^g</i>	2q232.2	<i>GDF8</i>	Growth differentiation factor 8, also known as myostatin	Reduced adiposity and increased muscle mass Loss of <i>Gdf8</i> expression also results in a significant reduction in adipose mass accumulation in agouti lethal yellow (<i>A^y</i>) and <i>Lep^{ob}</i> mutants	(383,384)
1(27.8)	<i>Gdf8^k</i>	2q232.2	<i>GDF8</i>	Growth differentiation factor 8, also known as myostatin	Transgene Overexpressed in muscle Resistant to diet-induced obesity	(385)
6(35.5)	<i>Gfpt1^k</i>	2p13	<i>GFPT1</i>	Glutamine fructose-6-phosphate transaminase 1	Transgene Overexpressed in adipose cells Increased adiposity	(386)
14(D3-E1)	<i>Gfra2^g</i>	8p21	<i>GFRA2</i>	Glial cell line-derived neurotrophic factor family receptor $\alpha 2$	Growth retardation accompanied by reduced fat mass and elevated basal metabolic rate	(387)
11(65)	<i>Gh^k</i>	17q24.2	<i>GH</i>	Growth hormone (GH)	Transgene. Bovine GH overexpressed from metallothionein promoter	(388)
11(65)	<i>Gh^k</i>	17q24.2	<i>GH</i>	Growth hormone (GH)	Resistant to diet-induced obesity Transgene. Bovine GH expressed in central nervous system	(389)
15(4.6)	<i>Ghr^f</i>	5p13-12	<i>GHR</i>	Growth hormone (GH) receptor	Obese Knock-in. Independent deletion of two domains designated m569 and m391	(390)
2(89)	<i>Ghrh^k</i>	20q11.2	<i>GHRH</i>	Growth hormone (GH)-releasing hormone	Increased adiposity in males Transgenic mouse expressing human <i>GHRH</i> cDNA from the mouse metallothionein I promoter	(391)
6(E3)	<i>Ghrl^g</i>	3p26-p25	<i>GHRL</i>	Ghrelin	Increased adiposity KO (lacZ fusion) On a high-fat diet, homozygous mutants tend to have a decrease in percentage body fat and an increase in percentage lean body mass without any significant difference in BW compared with wild-type mice	(392)
3	<i>Ghsr^g</i>	3q26.31	<i>GHSR</i>	Growth hormone (GH) secretagogue receptor	Decreased BW	(393)
3	<i>Ghsr^k</i>	3q26.31	<i>GHSR</i>	Growth hormone (GH) secretagogue receptor Expression limited to GHRH (GH-releasing hormone-expressing neurons)	Transgene. Rat <i>GHRH</i> 5' and 3' genomic sequences driving expression of human <i>GHSR</i> cDNA Decreased BW and adiposity	(394)
7	<i>Gipr^g</i>	19q13.3	<i>GIPR</i>	Gastric inhibitory polypeptide receptor	Resistant to diet-induced obesity; reduced adiposity in aged mice; lower respiratory exchange ratio and higher fat oxidation in the light phase	(395–397)
2(104)	<i>Gnas^g</i>	20q13.2-q13.3	<i>GNAS</i>	Imprinted locus GNAS (guanine nucleotide-binding protein, α -stimulating) complex locus	KO (exon 2) Loss of maternal expression leads to obesity, with increased lipid per cell in white and brown adipose tissue, whereas loss of paternal expression leads to a lean phenotype, with decreased lipid in adipose tissue	(398,399)
2(104)	<i>Gnas^g</i>	20q13.2-q13.3	<i>GNAS</i>	Imprinted locus GNAS (guanine nucleotide-binding protein, α -stimulating) complex locus	KO (exon 1) Maternal inheritance of the mutant allele gives larger BW in heterozygous mice	(400)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
19(4)	<i>Gng3^g</i>	11p11	<i>GNG3</i>	Guanine nucleotide-binding protein (G-protein), $\gamma 3$ subunit	KO. Floxed and deleted Female homozygotes have reduced inguinal and retroperitoneal fat pads	(401)
19(52)	<i>Gpam^g</i>	10q25.2	<i>GPAM</i>	Glycerol-3-phosphate acyltransferase, mitochondrial	Reduced BW and adiposity	(402)
2(33)	<i>Gpd2^g</i>	2q24.1	<i>GPD2</i>	Glycerol phosphate dehydrogenase 2, mitochondrial	Reduced BW and adiposity	(403)
2(33)	<i>Gpd2^g</i>	2q24.1	<i>GPD2</i>	Glycerol phosphate dehydrogenase 2, mitochondrial	Enhanced adipose and BW gain of females on a high-fat diet. This effect was not observed in males	(404)
12	<i>Gphb5^k</i>	14q23.2	<i>GPHB5</i>	Glycoprotein hormone $\beta 5$, also known as GPB5 and OGH	Transgene. Ubiquitous Resistant to diet-induced obesity	(405)
19	<i>Gpr10^g</i>	10q26.13	<i>GPR10</i>	G-protein-coupled receptor 10	Adult-onset obesity	(406)
5(F)	<i>Gpr109a^g</i>	12q24.31	<i>GPR109A</i>	G-protein-coupled receptor 109a	Absence of nicotinic acid-induced inhibition of free fatty acid release from adipocytes	(407)
15	<i>Gpr24^g</i>	22q13.3	<i>GPR24</i>	G-protein-coupled receptor 24, also known as Mch1r	Lean and resistant to diet-induced obesity	(408)
7	<i>Gpr40^g</i>	19q13.1	<i>GPR40</i>	G-protein-coupled receptor 40	Resistant to diet-induced obesity-mediated changes	(409)
1(A1)	<i>Gpr7^g</i>	8p22-q21.13	<i>GPR7</i>	G-protein-coupled receptor 7	Adult-onset obesity	(410)
9(57)	<i>Gpx1^k</i>	3p21.3	<i>GPX1</i>	Glutathione peroxidase 1	Transgene consisting of complete genomic <i>Gpx1</i> gene Increased BW and adiposity	(411,412)
7	<i>Grm5^g</i>	11q14.3	<i>GRM5</i>	Glutamate receptor, metabotropic 5	Lower BW and reduced adipose gain on high-fat diet	(413)
6(A3)	<i>Grm8^g</i>	7q31.3-q32.1	<i>GRM8</i>	Glutamate receptor, metabotropic 8	Increased fat mass	(414)
16(B4)	<i>Gsk3b^k</i>	3q13.3	<i>GSK3B</i>	Glycogen synthase kinase 3 β	Transgene expressing human cDNA in skeletal muscle Increased BW and adiposity in male transgenic mice	(415)
6(48.7)	<i>Gt(ROSA)26Sor^k</i>		Unknown	Gene trap ROSA 26, Philippe Soriano	Transgene. Conditional activation of Akt in skeletal muscle Decreased adipose mass and increased muscle mass after treatment with tamaxiphen	(293)
10(43)	<i>Gtrgeo22^g</i>	19p13.3	<i>C19orf20</i>	Gene trap 22, Philippe Soriano	Reduced BW and adiposity	(416)
11(61.2)	<i>Hcr1^k</i>	17q21	<i>HCRT</i>	Hypocretin (orexin). Loss of orexin-containing neurons	Transgene. Expression of Mjd (Ataxin 3) with expanded repeats in orexinergic neurons Late-onset obesity (C57BL/6J and DBA/2 mixed genetic background)	(417)
11(61.2)	<i>Hcr1^k</i>	17q21	<i>HCRT</i>	Hypocretin (orexin). Loss of orexin-containing neurons	Transgene. Expression of Mjd (Ataxin 3) with expanded repeats in orexinergic neurons No weight difference between mutant and wild type when both on a C57BL/6J genetic background	(418)
2(71)	<i>Hdc^g</i>	15q21-q22	<i>HDC</i>	Histidine decarboxylase	Increased BW and adiposity	(419)
5(20)	<i>Hdh^k</i>	4p16.3	<i>HD</i>	Huntington disease (HD) gene homolog, also known as Huntington, R6/2	Transgenic. Human exon 1 of the <i>HD</i> gene carrying ~141 to 157 CAG repeats expressed from the endogenous <i>HD</i> gene promoter Despite an overall growth retardation, the transgenic mice have increased adiposity Adult-onset wasting syndrome	(420)
13(46)	<i>Hexb^g</i>	5q13	<i>HEXB</i>	Hexosaminidase B	Lean	(421)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
10(67.5)	<i>Hmga2</i> ^g	12q15	<i>HMGA2</i>	High mobility group AT-hook 2	Resistant to diet-induced obesity	(422)
6(49)	<i>Hrh1</i> ^g	3p25	<i>HRH1</i>	Histamine receptor H1	Late-onset obesity	(423)
2	<i>Hrh3</i> ^g	20q13.3	<i>HRH3</i>	Histamine receptor H3	Increased adiposity and BW	(424)
1	<i>Hsd11b1</i> ^g	1q32-q41	<i>HSD11B1</i>	Hydroxysteroid 11- β dehydrogenase 1	Resistant to diet-induced obesity	(425)
1	<i>Hsd11b1</i> ^k	1q32-q41	<i>HSD11B1</i>	Hydroxysteroid 11- β dehydrogenase 1	Transgenic. Promoter aP2-specific expression	(426)
8(50.8)	<i>Hsd11b2</i> ^k	16q22	<i>HSD11B2</i>	Hydroxysteroid 11- β dehydrogenase 2	Increased adiposity Transgenic. Human gene expressed in adipose cells	(427)
X(66)	<i>Htr2c</i> ^g	Xq24	<i>HTR2C</i>	5-Hydroxytryptamine (serotonin) receptor 2C	Resistant to diet-induced obesity Late-onset obesity	(428)
9(7)	<i>Icam1</i> ^g	19p13.2	<i>ICAM1</i>	Intercellular adhesion molecule-1	Late-onset obesity Accelerated adiposity on a high-fat diet (N4 mice)	(429)
9(7)	<i>Icam1</i> ^g	19p13.2	<i>ICAM1</i>	Intercellular adhesion molecule-1	Transient increased adiposity after 11 days of high-fat diet but reduced BW and adiposity relative to controls after 50 days of high-fat diet (N8 mice)	(430)
9(7)	<i>Icam1</i> ^k	19p13.2	<i>ICAM1</i>	Intercellular adhesion molecule-1 (ICAM-1)	Transgene. Expressing soluble <i>ICAM-1</i> in liver Increased weight gain on a Western-type diet	(431)
1(29.8)	<i>Idh1</i> ^k	2q33.3	<i>IDH1</i>	Isocitrate dehydrogenase 1 (NADP ⁺), soluble, also known as IDPc Expression limited to liver and adipose tissue	Transgene, <i>Idh1</i> cDNA expressed from the rat cytosolic <i>Pepck</i> promoter Obesity	(432)
12(21.5)	<i>Ifrd1</i> ^k	7q22-q31	<i>IFRD1</i>	Also known as Tis7, interferon-related developmental regulator 1	Transgene. Expressed in gut small intestine	(433)
15	<i>Igfbp6</i> ^k	12q13	<i>IGFBP6</i>	Insulin-like growth factor-binding protein 6	Increased adiposity Transgene (human). Expressed in glial cells	(434)
2(10)	<i>Il1rn</i> ^g	2q14.2	<i>IL1RN</i>	Interleukin 1 receptor antagonist	Down-regulation of uncoupling protein 1 Reduced BW	(435)
2(10)	<i>Il1rn</i> ^g	2q14.2	<i>IL1RN</i>	Interleukin 1 receptor antagonist	Reduced adiposity, resistant to diet-induced obesity and resistant to obesity due to monosodium glutamate treatment	(436)
5(17)	<i>Il6</i> ^g	7p21	<i>IL6</i>	Interleukin 6	Increased adiposity and BW	(437)
5(17)	<i>Il6</i> ^g	7p21	<i>IL6</i>	Interleukin 6	No effect on adiposity. Reduced BW in 3-month-old mice	(438)
7(F1)	<i>Inpp1</i> ^g	11q23	<i>INPPL1</i>	Polyphosphate phosphatase-like 1 (SHIP-2)	Resistant to diet-induced obesity	(439)
8(1)	<i>Insr</i> ^c	19p13.3-p13.2	<i>INSR</i>	Insulin receptor not expressed in muscle cells	Floxed gene and muscle-specific Cre expression	(440)
8(5)	<i>Irs2</i> ^c	13q34	<i>IRS2</i>	Insulin receptor substrate 2. Conditional KO in pancreatic β cells and hypothalamus	Increased adipose depots and obesity Floxed gene + Cre transgene expressed from the rat insulin promoter	(441)
8(5)	<i>Irs2</i> ^c	13q34	<i>IRS2</i>	Insulin receptor substrate 2. Conditional KO in pancreatic β cells and hypothalamus	Fat mass, increased Floxed gene + Cre transgene expressed from the rat insulin promoter	(442)
3(52)	<i>Kcna3</i> ^g	1p13.3	<i>KCNA3</i>	Potassium voltage-gated channel, shaker-related subfamily, member 3, also known as Kv1.3	Fat mass, increased Reduced BW and resistant to diet-induced obesity	(443)
7(41)	<i>Kcnj11</i> ^g	11p15.1	<i>KCNJ11</i>	Potassium inwardly rectifying channel, subfamily J, member 11, also known as Kir6.2	Increased BW (10%) and epididymal fat pad (70%) weight	(444)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
14(E2.1)	<i>Klf5^g</i>	13q22.1	<i>KLF5</i>	Kruppel-like factor 5	Deficiency in white adipose tissue development	(445)
6(10.5)	<i>Lep^k</i>	7q31.3	<i>LEP</i>	Mouse <i>Lep</i> cDNA was cloned 3' of the liver-specific Apoe promoter and 5' of liver-specific enhancer sequence. Serum leptin expression is 200- to 300-fold higher than in wild-type mice	Transgene: Apoe promoter expressing murine leptin cDNA Absence of fat pads	(446)
6(10.5)	<i>Lep^k</i>	7q31.3	<i>LEP</i>	This transgene consists of a mouse <i>Lep</i> cDNA fused to the human APCS promoter with hormone expression targeted to the liver. In mutants carrying 30 copies of the transgene serum, leptin expression is about 12-fold higher than in wild-type mice	Transgene: human APCS promoter expressing murine leptin cDNA Decreased BW. Absence of adipose tissue	(447)
4(46.7)	<i>Lepr^f</i>	1p31	<i>LEPR</i>	Selective loss of long form of leptin receptor	Obesity	(448)
4(46.7)	<i>Lepr^k</i>	1p31	<i>LEPR</i>	Transgene insertion 1, Gerard Karsenty. An isoform of <i>Lepr</i> cDNA lacking the transmembrane domain found in all other isoforms was cloned 3' of the liver-specific Apoe promoter and 5' of liver-specific enhancer sequence	Transgene: Apoe promoter expressing murine-soluble leptin receptor cDNA Mice carrying this transgene possess normal body fat. However, in mice heterozygous for the <i>lep^{ob}</i> mutation, the transgenic mice show significantly increased adiposity	(446)
4(46.7)	<i>Lepr^c</i>	1p31	<i>LEPR</i>	Neuronal-specific deletion of leptin receptor	Floxed gene + <i>Cre</i> transgene expressed from the Syndecan 1 promoter Obesity	(449,450)
4(46.7)	<i>Lepr^k</i>	1p31	<i>LEPR</i>	Leptin receptor. Mutation due to insertion of rabbit smooth muscle myosin heavy chain promoter region	Mutation due to transgene insertion Obesity	(451)
4(46.7)	<i>Lepr^k</i>	1p31	<i>LEPR</i>	Leptin receptor	Transgenic: neuron-specific expression Rescue of obesity of <i>Lepr^{db}</i> mutant mice	(452)
4(46.7)	<i>Lepr^k</i>	1p31	<i>LEPR</i>	Leptin receptor	Transgenic: neuron-specific expression (50% and 75%) Adiposity and obesity are proportional to hypothalamic <i>LEPR</i> deficiency, but fertility and cold tolerance remain intact	(449)
7(23)	<i>Lhb^k</i>	19q13.32	<i>LHB</i>	Luteinizing hormone β polypeptide	Obesity in females	(453)
19	<i>LipI^g</i>	10q23.2-q23.3	<i>LIPA</i>	Lysosomal acid lipase 1	Decreased BW, fatty liver, loss of brown and white fat depots; ectopic fat deposition into liver, spleen, and bowel	(454,455)
9(39)	<i>Lipc^g</i>	15q21-23	<i>LIPC</i>	Lipase, hepatic	KO with background strain effects Increased adiposity mediated by background susceptibility	(456)
7(5.5)	<i>Lipe^g</i>	19q13.2	<i>LIPE</i>	Lipase, hormone sensitive	Reduced fat pad size, heterogenous adipocyte size, increased brown fat.	(457,458)
7(5.5)	<i>Lipe^k</i>	19q13.2	<i>LIPE</i>	Lipase, hormone sensitive	Resistant to diet-induced obesity Transgene: human <i>LIPE</i> expressed in adipose tissue Corrects adipose defects of <i>Lipe</i> -deficient mice	(459)
12(9)	<i>LpinI^h</i>	2p25.1	<i>LPIN1</i>	<i>Lipin 1</i> , gene responsible for phenotype of fatty liver dystrophic mouse	Spontaneous null allele Reduced adiposity on chow. Resistant to diet-induced obesity	(460)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
12(9)	<i>Lpin1^k</i>	2p25.1	<i>LPIN1</i>	Lipin 1	Transgene. Expressed in adipose tissue	(461)
12(9)	<i>Lpin1^k</i>	2p25.1	<i>LPIN1</i>	Lipin 1	Obesity due to increased fat storage Transgene. Expressed in skeletal muscle	(461)
7(61)	<i>Mapk3^g</i>	6p12-p11.2	<i>MAPK3</i>	Mitogen-activated protein kinase 3, also known as ERK1, Prkm3, p44	Obesity due to changes in energy expenditure N6 mice. Reduced adiposity, resistant to diet-induced obesity	(462)
2(E1)	<i>Mapk8ip1^g</i>	11p11.2	<i>MAPK8IP1</i>	Mitogen-activated protein kinase 8 interacting protein 1. Acts as an inhibitor of c-Jun N-terminal kinase	Reduced adiposity	(463,464)
2(100)	<i>Mc3r^g</i>	20q13.2-q13.3	<i>MC3R</i>	Melanocortin receptor 3	Obesity	(465)
2(100)	<i>Mc3r^g</i>	20q13.2-q13.3	<i>MC3R</i>	Melanocortin receptor 3	Obesity	(466)
18(E1)	<i>Mc4r^g</i>	18q22	<i>MC4R</i>	Melanocortin receptor 4	Obesity	(467)
18(E1)	<i>Mc4r^k</i>	18q22	<i>MC4R</i>	Melanocortin receptor 4	Transgene. Expressed in paraventricular hypothalamic nucleus and subpopulation of amygdala Prevents 60% of obesity, rescues hyperphagia but not reduced energy expenditure of <i>Mc4r</i> -deficient mutant mice	(468)
6(7.5)	<i>Mest^k</i>	7q32	<i>MEST</i>	Mesoderm-specific transcript	Transgene. Expressed in adipose tissue	(469)
2	<i>Mkks^g</i>	20p12	<i>MKKS</i>	McKusick-Kaufman syndrome protein	Increased adiposity	(470)
5	<i>MLXip^g</i>	7q11.23	<i>MLXIPL</i>	MLX interacting protein-like	Obesity Lean; rapid death on high-sucrose and high-fructose diets	(471)
10(40.9)	<i>Mmp11^g</i>	22q11.23	<i>MMP11</i>	Matrix metalloproteinase 11	Obesity	(472)
10(70)	<i>Mmp19^g</i>	12q14	<i>MMP19</i>	Matrix metalloproteinase 19	Accelerated BW and adipose mass gain on a high-fat diet	(473)
8(45)	<i>Mt1, Mt2^g</i>	16q13	<i>MT1A</i>	Metallothionein I and II	KO; both mutations generated with the same targeting construct	(474,475)
15(32)	<i>Myc^k</i>	8q24.12-q24.13	<i>MYC</i>	Myelocytomatosis oncogene. Expression limited to liver	Increased BW. Adult-onset obesity Transgene. Murine c-myc expressed under the control of the <i>Pepck</i> promoter	(476)
9	<i>Ncb5or^g</i>	6pter-q22.33	<i>NCB5OR</i>	NADPH cytochrome B5 oxidoreductase	Resistant to diet-induced obesity Reduced adipose mass	(345)
3	<i>Nhlh2^g</i>	1p12-p11	<i>NHLH2</i>	Nescient helix loop helix 2, also known as neural transcription factor 2 or NSCL2	Adult-onset obesity	(477)
5	<i>Nmu^g</i>	4q12	<i>NMU</i>	Neuromedin U	Elevated BW and obesity	(478)
5	<i>Nmu^k</i>	4q12	<i>NMU</i>	Neuromedin U	Transgene. Expressed ubiquitously	(479)
11(45.6)	<i>Nos2^g</i>	17q11.2-12	<i>NOS2A</i>	Nitric oxide synthase 2, inducible, macrophage, also known as iNOS	Lean and hypophagic Reduced adiposity	(480)
11	<i>Npb^g</i>	17q25.3	<i>NPB</i>	Neuropeptide B; ligand for GPR7	Mild obesity	(481)
6(26)	<i>Npy^g</i>	7p15.1	<i>NPY</i>	Neuropeptide Y	No obesity phenotype except with Galenin (<i>Gal</i>) KO	(375)
	<i>Npy^{-/-} + Gal^{-/-} -^g</i>			Double homozygote for neuropeptide Y and galenin deficiency	KO: compound double homozygous mutant strain	(375)
8(33)	<i>Npy1r^g</i>	4q31.3-q32	<i>NPY1R</i>	Neuropeptide Y receptor Y1	Obesity	(482)
8(32.5)	<i>Npy5r^g</i>	4q31-q32	<i>NPY5R</i>	Neuropeptide Y receptor Y5	Obesity Increased adiposity leading to mild adult-onset obesity	(483)
7	<i>Nr1h2^g</i>	19q13.3-13.2	<i>NR1H2</i>	Nuclear receptor subfamily 1, group H, member 2, also known as LXR β	Reduced adiposity	(484)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
1(92.6)	<i>Nr1i3^g</i>	1q23.3	<i>NR1I3</i>	Nuclear receptor subfamily 1, group I, member 3	Accelerated adipose loss on calorie-restricted diet	(485)
18(20)	<i>Nr3c1^c</i>	5q31	<i>NR3C1</i>	Nuclear receptor subfamily 3, group C, member 1; also known as glucocorticoid receptor	Floxed gene and rat nestin-driven Cre Neuronal-specific ablation leads to increased adiposity preweaning, leading to reduced adiposity in older mice due to altered food intake and metabolic efficiency	(486)
2(107)	<i>Ntsr1^g</i>	20q13-20q13	<i>NTSR1</i>	Neurotensin receptor 1	Adult-onset increase in BW and adiposity	(487)
10(8)	<i>Oprm1^g</i>	6q24-q25	<i>OPRM1</i>	Opioid receptor, $\mu 1$, also known as MOR, MOR-1	Resistant to diet-induced obesity	(488)
1(98.6)	<i>Parp1^g</i>	1q41-q42	<i>PARP1</i>	ADP-ribosyltransferase [NAD ⁺ , poly(ADP-rose)polymerase] 1, also known as Adprt1 or Adprp	Age-onset obesity in a mixed genetic background	(489,490)
13(44)	<i>Pcsk1^g</i>	5q15-q21	<i>PCSK1</i>	Proprotein convertase subtilisin/kexin type 1	Increased adipose mass in heterozygous mice	(491)
X	<i>Pcsk1n^k</i>	Xp11.23	<i>PCSK1N</i>	Proprotein convertase subtilisin/kexin type 1 inhibitor	Transgene: expressing Pcsk1n cDNA using the β -actin promoter Adult-onset obesity	(492)
7(6.5)	<i>Peg3^g</i>	19q31.4	<i>PEG3</i>	Paternally expressed 3, also known as Zfp102, End4, Pw1	Obesity	(493)
11(31)	<i>Pemt^g</i>	17p11.2	<i>PEMT</i>	Phosphatidylethanolamine <i>N</i> -methyltransferase	Liver abnormalities on high-fat diet	(494,495)
13(50)	<i>Pik3r1^g</i>	5q13.1	<i>PIK3R1</i>	Phosphatidylinositol 3-kinase, regulatory subunit, polypeptide 1 (p85 α)	Smaller adipocytes and reduced adiposity	(496)
11(58.2)	<i>Pip5k2b^c</i>	17q12	<i>PIP5K2B</i>	Phosphatidylinositol-4-phosphate 5-kinase, type II, β	Reduced BW and adiposity; resistant to diet-induced obesity	(497)
5(F1/G1)	<i>Pla2g1b^g</i>	12q23-q24.1	<i>PLA2G1B</i>	Phospholipase A2, group IB, pancreas	Normal BW and fat pad weight on chow diet; resistant to diet-induced obesity when fed a western diet. KO mice also displayed increased lipid content in the stool, thus displaying decreased fat absorption	(498)
7	<i>Plin^g</i>	15q26	<i>PLIN</i>	Perilipin	Reduced adiposity. Resistance to diet-induced obesity	(499)
9	<i>Plscr1^g</i>	3q23	<i>PLSCR1</i>	Phospholipid scramblase 1	Elevated adiposity	(500)
11(43)	<i>Plscr3^c</i>	17p13.1	<i>PLSCR3</i>	Phospholipid scramblase 3	Elevated BW and adipose mass	(501)
10(47)	<i>Pmch^g</i>	12q23-q24.1	<i>PMCH</i>	Promelanin-concentrating hormone, also known as MCH	Reduced BW and adiposity; resistant to diet-induced obesity	(502,503)
12(4)	<i>Pomc1^g</i>	2p23.3	<i>POMC</i>	Pro-opiomelanocortin- α	Obesity on chow and high-fat diets	(504,505)
5(75)	<i>Por^g</i>	7q11.2	<i>POR</i>	P450 (cytochrome) oxidoreductase	Conditional KO (liver specific) Increased liver weight and fatty liver	(506,507)
15(48.8)	<i>Ppara^g</i>	22q13.31	<i>PPARA</i>	Peroxisome proliferator-activated receptor α	Moderate elevation in gonadal fat in chow-fed females; significant increase in adiposity relative to wild-type mice in both males and females in high-fat diet-fed mice	(508)
15(48.8)	<i>Ppara^k</i>	22q13.31	<i>PPARA</i>	Peroxisome proliferator-activated receptor α	Transgene. Expressed in muscle Resistant to diet-induced obesity	(509)
17(13.5)	<i>Ppard^c</i>	6p21.2-p21.1	<i>PPARD</i>	Peroxisome proliferator activator receptor Δ , also known as peroxisome proliferator-activated receptor β . Specific loss of Ppard from adipose cells	Floxed gene and aP2-driven Cre Resistant to diet-induced obesity and reduced adiposity in <i>Lepr^{db}</i> homozygous mutants	(510)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
17(13.5)	<i>Ppard</i> ^c	6p21.2-p21.1	<i>PPARD</i>	Peroxisome proliferator activator receptor Δ , also known as peroxisome proliferator-activated receptor β , overexpressed in skeletal muscle	Floxed transgene. Cre-Lox strategy to overexpress Ppard in skeletal muscle using HAS-Cre Reduction in fat mass and adipocyte cell size	(511)
17(13.5)	<i>Ppard</i> ^e	6p21.2-p21.1	<i>PPARD</i>	Peroxisome proliferator activator receptor Δ , also known as peroxisome proliferator-activated receptor β	On a high-fat diet, KO mice develop greater adiposity than controls despite a lower overall total BW	(512)
6(52.7)	<i>Pparg</i> ^c	3p25	<i>PPARG</i>	Loss of peroxisome proliferator-activated receptor γ from adipose cells	Floxed gene and aP2-driven Cre Reduced adiposity and resistant to diet-induced obesity	(513,514)
6(52.7)	<i>Pparg</i> ^c	3p25	<i>PPARG</i>	Loss of peroxisome proliferator-activated receptor γ from muscle	Floxed gene and muscle creatine kinase (MCK)-driven Cre Increased adiposity	(515)
6(52.7)	<i>Pparg</i> ^c	3p25	<i>PPARG</i>	Loss of peroxisome proliferator-activated receptor γ from β cells	Floxed gene and rat insulin promoter-driven Cre Attenuated β cell hyperplasia in response to a high-fat diet	(516)
6(52.7)	<i>Pparg</i> ^c	3p25	<i>PPARG</i>	Peroxisome proliferator-activated receptor γ	KO + reporter Selective loss of PPARG2 isoform leads to reduced BW, smaller adipocytes, and resistance to diet-induced obesity	(517)
6(52.7)	<i>Pparg</i> ^f	3p25	<i>PPARG</i>	Peroxisome proliferator-activated receptor γ	Knock-in expressing dominant negative allele Lethal in homozygotes. Heterozygotes are lean and resistant to diet-induced obesity	(518)
5(C1)	<i>Ppargc1a</i> ^g	4p15.1-15.2	<i>PPARGC1A</i>	Peroxisome proliferative-activated receptor, γ , coactivator 1 α	Resistant to diet-induced obesity; cold sensitive	(519)
5(C1)	<i>Ppargc1a</i> ^g	4p15.1-15.2	<i>PPARGC1A</i>	Peroxisome proliferative-activated receptor, γ , coactivator 1 α	Increased adiposity in young females and old males	(520)
18	<i>Ppargc1b</i> ^k	5q32	<i>PPARGC1B</i>	Peroxisome proliferative-activated receptor, γ , coactivator 1 β	Transgene. Murine cDNA expressed from the chicken β -actin promoter Resistant to diet-induced obesity	(521)
6(A2)	<i>Ppp1r3a</i> ^g	7q31.1	<i>PPP1R3A</i>	Protein phosphatase 1, regulatory (inhibitor) subunit 3A	Increased BW and obesity	(522)
19(C3)	<i>Ppp1r3c</i> ^g	10q23-q24	<i>PPP1R3C</i>	Protein phosphatase 1, regulatory (inhibitor) subunit 3C, also known as PTG	Homozygous mutants are embryonic lethal. Heterozygotes show increased intramyocellular lipid stores and elevated circulating leptin, triglycerides, and free fatty acids	(523)
11	<i>Ppy</i> ^k	17q21	<i>PPY</i>	Pancreatic polypeptide	Transgenic. Mouse Ppy cDNA expressed from the chicken β -actin hybrid promoter Reduced BW and adiposity	(524,525)
4	<i>Prkaa2</i> ^g	1p31	<i>PRKAA2</i>	Protein kinase, adenosine monophosphate-activated, α 2 catalytic subunit. No expression in adipocytes	KO. Floxed gene + aP2 expressed Cre Increased adiposity	(526)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
1	<i>Prkag3^k</i>	2q35	<i>PRKAG3</i>	Transgene insertion 1, Leif Andersson. Expression of the mutated protein was seen in the skeletal muscle. Levels of the endogenous PRKAG3 protein were reduced, resulting in no significant change in overall PRKAG3 protein expression	An Myl1 promoter and enhancer expressing a full-length mouse Prkag3 cDNA (encodes protein kinase, AMP-activated, γ 3, noncatalytic subunit) with a single missense mutation (R225Q) Transgenic mice have lowered adiposity and are protected against high-fat diet-induced triglyceride accumulation and insulin resistance	(527)
12(B1)	<i>Prkar2b^g</i>	7q22	<i>PRKAR2B</i>	Protein kinase, cAMP-dependent regulatory, type II β , also known as RII β	Decreased adiposity; resistant to diet-induced obesity	(528,529)
2(2)	<i>Prkcq^k</i>	10p15	<i>PRKCQ</i>	Protein kinase C, θ	Obesity	(530)
19	<i>Prkhr^g</i>	10q26.13	<i>PRLHR</i>	Prolactin-releasing hormone receptor	Obesity	(406)
1(106.3)	<i>Prox1^g</i>	1q32.2-q32.3	<i>PROX1</i>	Prospero-related homeobox 1	Obesity in heterozygotes	(531)
2(H3)	<i>Ptpn1^g</i>	20q13.1-q13.2	<i>PTPN1</i>	Protein tyrosine phosphatase, non-receptor type 1	Reduced adiposity	(532,533)
5(F1/G1)	<i>Ptpn11^g</i>	12q24	<i>PTPN11</i>	Protein tyrosine phosphatase, non-receptor type 11	Forebrain-specific KO Obesity	(534)
2(73.1)	<i>Ptpns1^g</i>	20p13	<i>PTPNS1</i>	Protein tyrosine phosphatase, non-receptor-type substrate 1, also known as SHPS-1	Decreased BW and fatty livers	(535)
11	<i>Pttg1^g</i>	5q35.1	<i>PTTG1</i>	Pituitary tumor-transforming 1	Reduced BW and cessation of weight gain after 6 months in males accompanied by loss of epididymal fat mass	(536)
11(B2)	<i>Rai1^g</i>	17p11.2	<i>RAI1</i>	Retinoic acid induced 1	Homozygote lethal. Heterozygotes are obese	(537)
9(50)	<i>Rasgrf1^g</i>	15q24	<i>RASGRF1</i>	RAS protein-specific guanine nucleotide-releasing factor 1	Reduced BW and adiposity	(538)
1(69.9)	<i>Ren1^k</i>	1q32	<i>REN</i>	Renin 1	Transgene: human REN gene expressed from endogenous promoter Late-onset obesity	(539)
8(0.4)	<i>Retn^k</i>	19p13.2	<i>RETN</i>	Resistin/ADSF/Fizz3. Adipocyte-specific overexpression of dominant negative Retn	Transgene: aP2 promoter expressing Retn fused to the human IgG γ constant region Increased adiposity but enhanced glucose disposal and insulin sensitivity	(540)
16(33)	<i>Retnlb^k</i>	3q13.1	<i>RETNLB</i>	Resistin-like β ; expressed in liver	Hyperlipidemia and fatty liver on high-fat diet	(541)
X(65.7)	<i>Rps6ka3^g</i>	X p22.2-p22.1	<i>RPS6KA3</i>	Ribosomal protein S6 kinase polypeptide 3	Reduced BW and adiposity; resistant to diet-induced obesity	(542)
11	<i>Rps6kb1^g</i>	17q23.2	<i>RPS6KB1</i>	Ribosomal protein S6 kinase, polypeptide 1, S6K1	Resistant to diet-induced obesity	(543)
4	<i>Rsc1a1^g</i>	1p36.1	<i>RSC1A1</i>	Regulatory solute carrier protein, family 1, member 1	Obesity	(544)
1(88.1)	<i>Rxrg^g</i>	1q22-q23	<i>RXRG</i>	Retinoid X receptor γ	Resistant to diet-induced obesity	(545)
19(43)	<i>Scd1^g</i>	10	<i>SCD1</i>	Stearyl-CoA desaturase 1	Reduced BW and adiposity; resistant to diet-induced obesity	(508,546)
12(1)	<i>Sdc1^k</i>	2p24.1	<i>SDC1</i>	Syndecan 1	Transgenic. Mouse Sdc1 cDNA expressed from the cytomegalovirus promoter/enhancer Adult-onset obesity	(547)
4(60.8)	<i>Sdc3^g</i>	1pter-p22.3	<i>SDC3</i>	Syndecan 3	Reduced adiposity on chow. Resistant to diet-induced obesity	(548)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
5	<i>Serpine1^g</i>	7q21.3-q22	<i>SERPINE1</i>	Serine (or cysteine) proteinase inhibitor, clade E, member 1, also known as plasminogen activator inhibitor, type I	Resistant to diet-induced obesity	(549)
5	<i>Serpine1^g</i>	7q21.3-q22	<i>SERPINE1</i>	Serine (or cysteine) proteinase inhibitor, clade E, member 1, also known as plasminogen activator inhibitor, type I	Same BW gain as control mice on high-fat diet. No difference in subcutaneous fat mass but elevated gonadal adipose mass. Mutant and litter mates are 80% B6 and 20% 129 composite	(550)
5	<i>Serpine1^k</i>	7q21.3-q22	<i>SERPINE1</i>	Serine (or cysteine) proteinase inhibitor, clade E, member 1, also known as plasminogen activator inhibitor, type I. Expressed in adipose cells	Transgene. Murine cDNA expressed from the aP2 promoter Resistant to diet-induced obesity	(551,552)
8(9.5)	<i>Sfrp1^g</i>	8p12-p11.1	<i>SFRP1</i>	Secreted frizzled-related sequence protein 1	KO + reporter Reduced adiposity in males	(553)
7(61)	<i>Sh2bpsm1^g</i>	16p11.2	<i>SH2B</i>	SH2-B PH domain-containing signaling mediator 1, also known as SH2-B	Obesity	(554)
10(26.5)	<i>Sim1^c</i>	6q16.3-q21	<i>SIM1</i>	Single-minded 1	Floxed gene and EIIa-expressed Cre Obesity in heterozygous mice	(555)
10(26.5)	<i>Sim1^g</i>	6q16.3-q21	<i>SIM1</i>	Single-minded 1	Obesity in heterozygous mice	(556)
11(40)	<i>Slc2a4^g</i>	17p13	<i>SLC2A4</i>	Solute carrier family 2 (facilitated glucose transporter), member 4 (encodes GLUT4)	Reduced adiposity	(557)
11(40)	<i>Slc2a4^k</i>	17p13	<i>SLC2A4</i>	Solute carrier family 2 (facilitated glucose transporter), member 4 (encodes GLUT4)	Transgene. Expressed in adipose tissue Increased fat mass	(558)
6	<i>Slc6a1^k</i>	3p25-p24	<i>SLC6A1</i>	γ -aminobutyric acid transporter 1	Transgenic. Mouse Slc6a1 brain-derived cDNA expressed from the cytomegalovirus promoter/enhancer Obesity	(559)
15(31.7)	<i>Soat2^g</i>	12q13.13	<i>SOAT2</i>	Sterol O-acyltransferase 2	Resistant to fatty liver but elevated circulating triglycerides and high-density lipoprotein cholesterol	(560)
11(E2)	<i>Socs3^c</i>	17q25.3	<i>SOCS3</i>	Suppressor of cytokine signaling 3. Cerebrum- and hypothalamus-specific loss of Socs3	Floxed gene + transgenic Cre expressed from rat nestin promoter Decreased BW and resistant to diet-induced obesity	(561)
11(E2)	<i>Socs3^c</i>	17q25.3	<i>SOCS3</i>	Suppressor of cytokine signaling 3. Cerebrum and hypothalamus-specific loss of Socs3	Floxed gene + transgenic Cre expressed from Syndecan I promoter Decreased BW and resistant to diet-induced obesity	(561)
17(8)	<i>Sox8^g</i>	16p13.3	<i>SOX8</i>	SRY box-containing gene 8	Decreased fat mass	(562)
11(29.9)	<i>Sparc^g</i>	5q31.3-q32	<i>SPARC</i>	Secreted acidic cysteine-rich glycoprotein (osteonectin)	Increased adiposity with no effect on BW	(563)
11	<i>Srebf1^k</i>	17p11.2	<i>SREBF1</i>	Transgene expresses sterol regulatory element-binding factor 1 in adipose cell	Transgene: aP2-driven human SREBF1c cDNA Reduced BW and adiposity	(564)
11	<i>Srebf1^k</i>	17p11.2	<i>SREBF1</i>	Sterol regulatory element-binding factor 1, transcript 1a, also known as Srebp1. Expressed in adipose cells	Transgene expressing the human SREBF1-1a cDNA using the murine aP2 promoter Adipocyte hypertrophy and fatty liver	(565)
11	<i>Srebf1^k</i>	17p11.2	<i>SREBF1</i>	Sterol regulatory element-binding factor 1, transcript 1c, also known as Srebp1. Expressed in adipose cells	Transgene expressing the human SREBF1c cDNA using the murine aP2 promoter Loss of all adipose tissue	(564)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
11(60.5)	<i>Stat3^c</i>	17q21.31	<i>STAT3</i>	Pancreatic- and hypothalamic-specific deletion of Stat3; signal transducer and activator of transcription 3	Floxed gene + Cre transgene expressed from the rat insulin II promoter Obesity. Transplantation of wild-type pancreatic islets into the mutants did not alleviate obesity, suggesting that lack of hypothalamic Stat3 expression is responsible for the obesity	(566)
11(60.5)	<i>Stat3^c</i>	17q21.31	<i>STAT3</i>	Neuronal-specific deletion of Stat3; signal transducer and activator of transcription 3	Floxed gene + Cre transgene expressed from the rat Nestin promoter Obesity	(567)
11(60.5)	<i>Stat5b^g</i>	17q11.2	<i>STAT5B</i>	Signal transducer and activator of transcription 5B	Increased adiposity	(568)
7(6.5)	<i>Tgfb1^k</i>	19q13.31	<i>TGFB1</i>	Transforming growth factor, β 1	Transgenic. Human TGFB1 cDNA expressed from the rat PEPCK promoter Reduced adiposity; lipodystrophy	(569)
11(57)	<i>Thra^g</i>	17q11.2	<i>THRA</i>	Thyroid hormone receptor α	KO; gene replacement with dominant negative mutant allele Increased BW and adiposity	(570)
14(B1)	<i>Tkt^g</i>	3p14.3	<i>TKT</i>	Transketolase	Homozygous mutants are embryonic lethal. Heterozygotes display reduced BW and adiposity	(571)
17(19.1)	<i>Tnf^g</i>	6p21.3	<i>TNF</i>	TNF	Reduction in BW and adiposity	(572)
17(19.1)	<i>Tnf^k</i>	6p21.3	<i>TNF</i>	TNF. Non-cleavable mutant protein expressed in TNF-deficient mice	Elevated BW and adipose fat mass	(573)
7(51.5)	<i>Tub^g</i>	11p15.5	<i>TUB</i>	Tubby candidate gene	Late-onset obesity	(574)
3(47.1)	<i>Txnip^g</i>	1q21.1	<i>TXNIP</i>	Thioredoxin-interacting protein	Increased fat-to-muscle ratio	(575)
2(67.4)	<i>Ubr1^g</i>	15q13	<i>UBR1</i>	Ubiquitin protein ligase E3 component n-recogin 1	Lean	(576)
8(38)	<i>Ucp1^g</i>	4q28-q31	<i>UCP1</i>	Uncoupling protein 1, mitochondrial	Temperature-dependent resistance to diet-induced obesity on C57BL/6J genetic background	(577)
8(38)	<i>Ucp1^k</i>	4q28-q31	<i>UCP1</i>	Transgene insertion 1, Frederic Bouillaud. The transgene consists of a rat uncoupling protein 1 (UCP1) cDNA sequence under the control of a mouse Ckmm promoter. Expression limited to skeletal and cardiac muscle	Transgene: murine muscle creatine kinase promoter expressing rat UCP1 cDNA Lower BW and reduced adiposity	(578)
8(38)	<i>Ucp1^k</i>	4q28-q31	<i>UCP1</i>	Uncoupling protein 1 (UCP1), mitochondrial	Transgene. UCP1 promoter expressing the diphtheria toxin gene Ablation of UCP1 expressing tissues leads to obesity	(579,580)
7(50)	<i>Ucp2/Ucp3^k</i>	11q13	<i>UCP2/UCP3</i>	Uncoupling protein 2 (UCP2), mitochondrial; uncoupling protein 3 (UCP3), mitochondrial	Transgene. Murine bacterial artificial chromosome containing the genomic UCP2 and UCP3 genes Reduced adiposity	(581)
7(50)	<i>Ucp3^k</i>	11q13	<i>UCP3</i>	Uncoupling protein 3 (UCP3), mitochondrial. Expression limited to skeletal muscle	Transgene. Murine UCP3 cDNA expressed from the mouse Mck promoter No difference on chow, but a 4-week exposure to a high-fat diet revealed transgenic mice have less weight gain and reduced adipose gain	(582)

Table 2. (continued)

Mouse chromosome (cM/band)	Mouse gene	Human chromosome	Human homolog	Gene description	Details	Reference
3(68.5)	<i>Unc5c</i> ^k	4q21-q23	<i>UNC5C</i>	unc-5 homolog C (<i>C. elegans</i>)	KO/transgene: a cDNA encoding telomerase reverse transcriptase under the control of the chicken β -actin promoter randomly inserted into intron 1 of <i>Unc5c</i> Reduction in BW and adiposity	(583)
5(79)	<i>Vgf</i> ^g	7q22	<i>VGF</i>	VGF nerve growth factor inducible	Reduction in BW and adiposity	(584)
19(20)	<i>Vldlr</i> ^g	9p24	<i>VLDLR</i>	Very-low-density lipoprotein receptor	Reduction in BW and adiposity	(585)
5	<i>Wbscr14</i> ^g	7q11.23	<i>WBSCR14</i>	Williams-Beuren syndrome chromosome region 14 homolog (human)	Lower adiposity on standard diet. Rapid death on feeding a high-fructose or high-sucrose diet	(471)
15(56.8)	<i>Wnt10b</i> ^k	12q13	<i>WNT10B</i>	Wingless-related MMTV integration site 10b	Transgene, cDNA expressed from the aP2 (Fabp4) promoter Reduced adiposity and resistant to diet-induced obesity. Loss of brown adipose tissue	(586,587)
15(56.8)	<i>Wnt10b</i> ^g	12q13	<i>WNT10B</i>	Wingless-related MMTV integration site 10b	Increased muscular adiposity	(588)
7(10.2)	<i>Zfp36</i> ^g	19q13.1	<i>ZFP36</i>	Zinc finger protein 36	Reduction in BW and adiposity	(589)

^a Antisense; ^b ethylnitrosourea (ENU); ^c floxed; ^d gene replacement; ^e gene trap; ^f knock-in; ^g knock-out; ^h spontaneous; ⁱ overexpression; ^j RNA interference; ^k transgenic.

One particularly interesting addition to the gene list is the murine *Clock* gene. The CLOCK transcription factor is a key component of the molecular circadian clock within pacemaker neurons of the hypothalamic suprachiasmatic nucleus. Characterization of murine *Clock* mutants reveals an obesity phenotype that is accelerated during feeding with high-fat diet. Causative factors include an attenuated diurnal feeding rhythm, hyperphagia, and perturbation of the expression of hypothalamic peptides associated with the regulation of feeding behavior and energy balance. The effects of the CLOCK transcription factor seem to be associated with growth and development only after weaning because no differences in BW are observed in newborn pups or 3- or 4-week weaned mice.

Animal QTLs

The murine QTL information in Table 3 has been completely revised this year. Primarily, the names assigned to quantitative trait loci (QTLs) have been changed to conform to currently utilized nomenclature, and, in an attempt to more specifically define the location of the QTL on the mouse genome, we have included the genetic location of the peak logarithm of the odds ratio (LOD) score (or other statistical measure utilized) and a confidence interval (usually the 1 LOD interval). Information presented has been summarized from the primary literature and also from the MGI group at the Jackson Laboratory (www.informatics.

jax.org). Clearly, the concept of QTL significance plays a large role in the identification of a QTL. We have attempted to adopt a uniform standard that identifies QTLs if they satisfy a genome-wide significance level below 0.05. QTLs that do not meet this are termed suggestive, and we have listed only suggestive QTLs that have either been corroborated in follow-up studies or replicated in another study using the same mouse strains. In cases of uncertainty, we have erred on the side of caution and listed the QTLs. For some recent studies, evidence for interactions between QTLs has been presented, despite no evidence of significance for the individual loci alone. Nomenclature rules may need to be revisited to describe these interactions. In the majority of the cases, QTL names listed in the table are not identical to those listed in the primary publication. In these cases, the names were changed by the MGI group to maintain conformity with existing nomenclature. Thus, names that have been listed in previous years may have been altered in this year's table.

QTLs may be identified from several different types of genetic crosses. We have listed this information in this year's table. Typically, F₂ intercrosses or backcrosses are utilized. However, there is likely to be an increasing use of recombinant inbred strains, advanced intercross lines, and congenic strains (that contain a specific donor genetic segment on a different background strain). It must be remembered that QTLs identified from phenotyping and genotyp-

Table 3. QTLs reported for animal polygenic models of obesity

Cross	QTL	Scores	Variance (%)	Phenotypes	Animal chromosome	QTL peak	Reference
Cow							
CGC × Hereford	<i>BTA17LW</i>	$F = 8$		−24 kg live weight	17		(590)
Chicken							
White Leghorn layer × commercial broiler	<i>BFc7 36-41</i>	$F = 13.29$	5.24	Abdominal fat weight (9 weeks)	7		(591)
	<i>BFc7 36-41</i>	$F = 11.50$	4.51	Abdominal fatness (9 weeks)	7		
	<i>BFc7 36-41</i>	$F = 11.08$	4.4	Fat distribution (9 weeks)	7		
Rhode Island Red layer × Rhode Island Red layer	<i>BWc4 200-207</i>	$p = 0.01$	25.8	BW (40 weeks)	4		(592)
White Leghorn × Rhode Island Red	<i>Gwchr4</i>	$p = 0.01$	17	Weight	4		(593)
	<i>Gwchr27</i>	$p = 0.01$	6	Weight	27		
White Plymouth Rock × White Plymouth Rock	<i>Bw5</i>	$F = 2.14$		Weight (5 weeks)	1		(594)
	<i>Bw7</i>	$F = 2.28$		Weight (7 weeks)	1		
White Plymouth Rock × Rhode Island Red layer	<i>Gfchr1</i>	LOD = 2.75	18.1	Fat (%), abdominal	1		(595)
	<i>Gwchr13</i>	LOD = 2.77	26.6	Weight	13		
Rhode Island Red layer × Rhode Island Red layer	<i>GAA01 263-287</i>			Weight	1		(596)
	<i>GAA02 23-28</i>			Weight	2		
Mouse (multiple crosses)							
(B6.129-Lip ^{tm1Unc} × SPRET/Ei)F1 × C57BL/6-Lip ^{tm1Unc}	<i>Bsbob</i>			Body fat and adiposity	2	81.7	(456,597)
(B6.129-Lip ^{tm1Unc} × SPRET/Ei)F1 × C57BL/6J	<i>Bsbob2</i>			BW, fat mass, adiposity; interacts with <i>Bsbob4</i> to regulate adiposity and BW	7	62 (60 to 63.5)	
(C57BL/6J × SPRET/Ei)F1 × C57BL/6J	<i>Bsbob3</i>			Interacts with <i>Bsbob4</i> to regulate total cholesterol	6	26.5	
C57BL/6J/Lip ^{−/−} × <i>Mus spretus</i> SPRET/Ei	<i>Bsbob4</i>			Interacts with <i>Bsbob3</i> to regulate total cholesterol; interacts with <i>Bsbob5</i> to regulate body fat	12	52	
	<i>Bsbob5</i>			Interacts with <i>Bsbob4</i> to regulate body fat	15	20.2	
		LOD = 3.6	26	Interaction between <i>Lipc</i> (chromosome 9) and chromosome 7 locus	7		
(BALB/cJ × C57BL/6J)F1 × (C3H/HeJ × DBA/2J)F1	<i>D3Mit127</i>	$p = 0.01$		Leptin, 27%	3	70.3	(598)
(C3H/He × <i>Mus spretus</i>)F1 × C57BL/6J	<i>Bw1</i>	LOD = 3.4	24	BW	X	18 cM (DXMit57-DXMit48)	(599)
	<i>Bw2</i>	LOD = 6.6		BW	X	DXMit109-DXMit16	
	<i>Bw3</i>	LOD = 4.3		BW	X	32 cM	
	<i>Bw1</i>			BW	X	Distal to DXNds1	
	<i>Bw2</i>			BW	X	DXMit60-DXMit16	
	<i>Bw3</i>			BW	X	DXMit3-DXMit12	

Table 3. (continued)

Cross	QTL	Scores	Variance (%)	Phenotypes	Animal chromosome	QTL peak	Reference
(C57BL/6-Lip ^{tm1Unc} × SPRET/Ei)F1 × SPRET/Ei	<i>Bsbob</i>	LOD = 3.7	3	Adiposity QTL present in BSBHLKO cross (BSB hepatic lipase-deficient animals) but not in BSB	2	79 (75.6 to 81.1)	(600)
B6.S(D2mit194-D2Mit311)	<i>Bsbob</i>			Confirmed to contain obesity QTL	2	26.7- to 32.1-megabasepair interval	
(C57BL/6J × SPRET/Pt)F1 × C57BL/6J	<i>Hlbsb1</i> <i>Hlbsb2</i>	LOD = 4.8	10.7 7	Hepatic lipase activity Hepatic lipase activity; interaction with <i>Hlbsb1</i>	7 3 to 7	64 (48 to 66)	(601)
(C57BL/6J × STRET/Ei)F1 × C57BL/6J	<i>Mob1</i> <i>Mob2</i> <i>Mob3</i> <i>Mob4</i>	LOD = 4.6 LOD = 4.8 LOD = 4.8 LOD = 3.4	6.5 7.1 7 5.9	Fat (%) Femoral fat Fat (%) Mesenteric fat	7 6 12 15	62 3.05 53 6.7	(602,603)
[High BW line (H) × low BW line (L)]F1 × high BW line (H)	<i>Bw19</i>	LOD = 137	20	Candidate gene <i>Gpc3</i> identified in 660-kilobasepair interval	X	DXMit226-DXMit68 (2 cM)	(604–606)
(<i>Mus m. castaneus</i> × C57BL/6J)F1 × <i>M. m. castaneus</i>	<i>Pbwg1</i>	LOD = 3.1 to 10.9	10	Stronger effect in females than males. Epistatic with <i>Pbwg12</i> in males	2	32.8 (26 to 44)	(607–609)
	<i>Pbwg2</i>	LOD = 3.1	3.9	Male specific	4	62 (39 to 86)	
	<i>Pbwg3</i>	LOD = 2.6 to 3.6	3		7	72 (40 to 104)	
	<i>Pbwg4</i>	LOD = 3.7	7.5	Female specific; BW 5 weeks	9	71 (46 to 96)	
	<i>Pbwg5</i>	LOD = 3.4	3.7	BW at 7 weeks	10	68 (42 to 94)	
	<i>Pbwg6</i>	LOD = 4.9	5.2	Stronger effect in males than females	13	53 (34 to 72)	
	<i>Pbwg7</i>	LOD = 3.1	6.9	Male specific	X	19 (0 to 48)	
	<i>Pbwg8</i>	LOD = 4	12.1	Male specific	6	32 (14 to 50)	
	<i>Pbwg9</i>	LOD = 3.8	4.5	Stronger effect in females than males	10	14 (0 to 36)	
	<i>Pbwg10</i>	$p = 2.6 \times 10^{-6}$		Interaction with <i>Pbwg8</i> in males	X	2.8	
	<i>Pbwg12</i>	$p = 9.1 \times 10^{-6}$		Male specific and epistatic with <i>Pbwg1</i> in males	12	34	
	<i>Pbwg13</i>	$p = 2.5 \times 10^{-6}$		Interaction with <i>Pbwg9</i> in females	5	81	
	<i>Pbwg14</i>	LOD = 2.8		BW at 8 weeks. Female specific	5	1 (0 to 74)	
	<i>Pbwg15</i>	LOD = 2.6 to 2.7		BW at 3 to 4 weeks	9	43	
	<i>Pbwg16</i>	LOD = 4.6		BW at 3 weeks	10	45	
	<i>Pbwg17</i>	LOD = 4.2 to 4.9		BW at 6 to 10 weeks	13	46	
	<i>Pbwg18</i>	LOD = 4.3		Female specific. Weight gain from 6 to 10 weeks	14	30	
	<i>Pbwg19</i>	LOD = 3		Male specific. BW at 5 weeks	16	2	
	<i>Pbwg20</i>	LOD = 3.1 to 6.3		BW at 3 to 4 weeks	19	0 (0 to 25)	

Table 3. (continued)

Cross	QTL	Scores	Variance (%)	Phenotypes	Animal chromosome	QTL peak	Reference
	<i>Pbwg21</i>	LOD = 2.6 to 3.6		Male specific. BW at 3 weeks and weight gain 3 to 6 weeks	X	27	
	<i>Pbwg22</i>	LOD = 2.9 to 4.3		Female specific. BW 9 to 10 weeks	X	35	
	<i>C10bw2</i>	LOD = 3.6		Female specific	9	17	
	<i>C10bw3</i>	LOD = 3.4		Male specific	11	57	
	<i>C10bw4</i>	LOD = 3.3		Female specific	13	46	
	<i>C10bw6</i>	LOD = 4.3		Female specific	X	18.9	
(Quackenbush-Swiss × C57BL/6J) × C57BL/6J	Not assigned	$p = 0.009$	40	BW and body length	10	56 to 65	(610)
129P3/J × C57BL/6J	<i>Bwq5</i>	LOD = 4.4	4.8	BW	2	81.7	(611)
	<i>Bwq6</i>	LOD = 4	4.3	BW	9	61	
	<i>Adip5</i>	LOD = 3.95	4.7	Adiposity; interaction with Adip9	9	26	
	<i>Adip6</i>	LOD = 3.32	4.4	Adiposity, interaction with Adip5	16	63.2	
129T2/SvEmsJ × EL/Suz	<i>Obq1</i>	LOD = 8	12.3	Adiposity	7	28	(612)
	<i>Obq2</i>	LOD = 4	6.3	Adiposity	1	15	
AKR/J × C57L/J	<i>Obq3</i>	LOD = 5.1	7	Adiposity	2	53 (34 to 78.7 cM)	(613)
	<i>Obq4</i>	LOD = 4.6	6.1	Adiposity	17	4 (0 to 7)	
AKR/J × SWR/J	<i>Dob1</i>	LOD = 4.4			4	50 (D4Mit5-D4Mit11)	(614,615)
		2004 = 4.8					
	<i>Dob2</i>	LOD = 4.8		Adiposity. QTL Not confirmed in (AKR × SWR) × SWR backcross mice	9	60 (D9Mit11-D9Mit18)	
		2004 = 3.9					
	<i>Dob3</i>	LOD = 3.9		Adiposity. QTL confirmed in (AKR × SWR) × SWR backcross	15	22.8 (D15Nds2-D15Mit22)	
B6.V-Lep ^{ob/ob} (leptin treated) × BALB/cJ	<i>Bwob</i>	LOD = 5			5	44	(616)
	<i>Mors1</i>	LOD = 5.6			1	101.5 to 106.3	
	<i>Mors2</i>	LOD = 3.4			3	52.5 to 71.8	
	<i>Mors3</i>	LOD = 3.8		Testosterone	14	27.5 to 30	
	<i>Mors4</i>	LOD = 3.4		Testosterone	14	15 to 27.5	
B10.UW H3 ^b we Pax1 un a ^l /Sn X		LOD = 4.61		Fat; adiposity adjusted for weight	2	67.8 to 82	(617)
BALB/cA and TSOD × TSOD	<i>Nidd5</i>	LOD = 5.91		BW and insulin	2	34.5	(618)
and BALC/cA	<i>Nidd6</i>	LOD = 4.65		BW	1	77	
BTBR.V-Lep ^{ob/ob} × B6.V-Lep ^{ob/ob}	<i>Mobe1</i>	LOD = 9.48	14.1	10-Week body mass	2	44 (41 to 47.1)	(619)
	<i>Mobe2</i>	LOD = 8	12.5	10-Week body mass	13	37	
	<i>Mobe3</i>	LOD = 3.6		10-Week body mass	5	65	
	<i>Mobe4</i>	LOD = 2.49		10-Week body mass	17	34.4	
C3H/He × NSY	Waiting for identification	LOD = 6.8		BMI	6	35.5 (32 to 60)	(620)
C57BL/6-insr ^{tm1Dac/} +, irs1 ^{tm1Jos/} + × 129S6/SvEvTac	<i>Elpt</i>	LOD = 3.7	33	Leptin. Interacts with hyperinsulinemia QTL, Hypn	7	50	(621)
C57BL/6J × 129S1/SvImJ	<i>Obq16</i>	LOD = 10		Females	8	48 (42 to 53)	(622)
	<i>Obq17</i>	LOD = 2.3		Females	1	74 (48 to 108)	
	<i>Obq18</i>	LOD = 2.9		Interacts with Obq16 (females)	9	65 (0 to 75)	
	<i>Obq19</i>			BMI (females)	17	8 (38 to 72)	
	<i>Mob2</i>	LOD = 2.6		(Females)	6	0 (0 to 10)	

Table 3. (continued)

Cross	QTL	Scores	Variance (%)	Phenotypes	Animal chromosome	QTL peak	Reference
C57BL/6J × 129S6/ SvEvTac	<i>D3Mit127</i>	LOD = 2.7	78		3	70.3	(623)
	<i>D10Mit162</i>	LOD = 2.9	28		10	59	
	<i>D12Mit231</i>	LOD = 3.2	25		12	48	
	<i>D14Mit192</i>	LOD = 3	52		14	40	
C57BL/6J × A/J	<i>Bw8q1</i>	LOD = 4.4	2	BW, 8 weeks	1	100 (77 to 102)	(624)
	<i>Bw8q2</i>	LOD = 3.3	4	BW, 8 weeks	4	66	
C57BL/6J × B6.A ^{Chr16}	<i>Diobq</i>	LOD = 4.3		Chromosome substitution strain	16	53.8 (29 to 55.2)	(625)
C57BL/6J × CAST/ Ei	<i>Mob5</i>	LOD = 5.8		Subcutaneous fat	2	95.5 (75 to 109)	(626,627)
	<i>Mob6</i>	LOD = 7.3		Subcutaneous fat. QTL confirmed in B6.CAST (73 to 83 cM) congenic mice	2	49.6 (35 to 85)	
	<i>Mob7</i>	LOD = 5.8		Retroperitoneal and subcutaneous fat	2	Peak at D2Mit9 37 (30 to 46)	(628)
	<i>Mob8</i>	LOD = 4.7		Body fat (%)	9	D9Mit8	
	Not assigned	LOD = 5.2		Leptin level (no obesity)	4		
	<i>Bdln2</i>	LOD = 4.3		Body length	15	15	
C57BL/6J × CAST/ EiJ	<i>Mnif1</i>	LOD = 8	7.1	Fat, intake	8	22 (10 to 30)	
	<i>Mnif2</i>	LOD = 6	5.4	Fat, intake	18	24 (10 to 58)	(629,630)
	<i>Mnif3</i>	LOD = 4	3.6	Fat, intake	X	18 (10 to 58)	
	<i>Mnic1</i>	LOD = 6.7	6	Carbohydrate intake	17	10 (3 to 24)	
	<i>Mnic2</i>	LOD = 3.4	3.1	Carbohydrate intake	6	46 (36 to 64)	
	<i>Mnic3</i>	LOD = 4.1	3.7	Carbohydrate intake	X	40 (14 to 61)	
	<i>Kcal1</i>	LOD = 7.7	6.8	Kilocalorie intake	18	20 (10 to 26)	
	<i>Kcal2</i>	LOD = 4.9	4.4	Kilocalorie intake	17	16 (8 to 37)	
	Not assigned	LOD = 3.3	3	6-Week weight	1	76 (46 to 84)	
	Not assigned	LOD = 3.3	4	6-Week weight	4	26 (24 to 30)	
C57BL/6J × DBA/ 2J	Not assigned	LOD = 3.2	4	6-Week weight	5	60 (57 to 64)	(631,632)
	Not assigned	LOD = 4.3	5	6-Week weight	5	35 (22 to 45)	
	Not assigned	LOD = 4	4	6-Week weight	6	22 (15 to 26)	
	Not assigned	LOD = 3.3	4	6-Week weight	4	26 (24 to 30)	
	Not assigned	LOD = 6.9	9	6-Week weight	7	25 (23 to 33)	
	Not assigned	LOD = 4.4	5	6-Week weight	9	32 (12 to 50)	
	Not assigned	LOD = 5.7	6	6-Week weight	11	45 (29 to 49)	
	Not assigned	LOD = 4.1	4	6-Week weight	13	59 (29 telomere)	
	Not assigned	LOD = 3	3	6-Week weight	14	0 (0 to 22)	
	Not assigned	LOD = 4.9	7	6-week weight	17	14 (11 to 18)	
	<i>Pfat1</i>	LOD = 5	20	Predicted fat (%)	4	30	
	<i>Pfat2</i>	LOD = 4.9	20	Predicted fat (%)	6	31.8	
	<i>Pfat3</i>	LOD = 5.3	20	Predicted fat (%)	13	20	
	<i>Pfat4</i>	LOD = 8.6	20	Predicted fat (%)	15	43.3	
C57BL/6J × KK-A ^y	<i>Bwq1</i>	LOD = 5.5	15	BW at 50 days of age	4	21.9 (6.3 to 32)	
	<i>Bwq2</i>	LOD = 8.8	26	BW from 40 to 100 days of age; modifier of A ^y	6	35.2 (29 to 47)	
C57BL/6J × KK/ H1Lt	<i>Obq5</i>	LOD = 7	17	Adiposity (females)	9	19	(633)
	<i>Obq6</i>	LOD = 5	15.7	Adiposity (males); except mesenteric	X	16	
	Unassigned	LOD = 4.4 (6.9)			7		
	Unassigned	LOD = 5.9			9		
	Unassigned	LOD = 4.2			7		
C57BL/6J × NZB/ BINJ	<i>Bwefm</i>	LOD = 5.11			5	70	(634)
	<i>Bwem1</i>	LOD = 3.16			5	54	
	<i>Bwem2</i>	LOD = 4.53			13	35	
C57BL/6J-Socs2 ^{hg/hg} × CAST/Ei	<i>Carfhg1</i>	LOD = 2.5	6.2	Fat content	5	38	(635)
	<i>Carfhg2</i>	LOD = 5.8	12.5	Fat content	9		
	<i>wg1</i>			2 to 9 weeks	2	31	

Table 3. (continued)

Cross	QTL	Scores	Variance (%)	Phenotypes	Animal chromosome	QTL peak	Reference
C57BL/6J-Socs2 ^{hg/hg} × CAST/Ei	<i>wg2</i>		10.4	2 to 9 weeks	2	61	
	<i>wg3</i>			2 to 9 weeks	8	45	
	<i>wg4</i>			2 to 9 weeks	11	46	
	<i>Mohg1</i>	$p = 0.004$			2	46 to 50.3	(636)
	<i>Mohg2</i>	$p = 0.021$			X	3	
C57BLKS/J-Cpe ^{fat} × HRS ^{hr/+}	<i>Mohg3</i>	$p = 0.041$			X	37	
	<i>Final</i>	LOD = 6.84	13	Adiposity index	11	40 (30 to 50)	(637)
	<i>Bwt1</i>	LOD = 14.03	24	Interaction with locus on chromosome 18 (47 cM)	14	22.5 (20 to 35)	
CAST/Ei × C57BL/6J	<i>Dob4</i>	LOD = 3.1 to 4.3	14	Fat, mesenteric	4	18.35	(638)
CFLP (P6) × JU/CBA	<i>Bw19</i>	LOD = 24.4	17 to 20	10-Week weight	X	24.64	(639)
Du6 × DuK	<i>Imebt2</i>			BW	14	25 (19 to 32)	(640)
DU6i × DBA/2J	<i>Bw4</i>	$F = 9.52$	4.9		11	55 (36 to 65)	(641,642)
	<i>Bw5</i>	$F = 10.44$	5.4	BW	1	36 (11 to 97)	
	<i>Bw7</i>	$F = 5.34$	2.8		4	59 (34 to 72)	
	<i>Bw9</i>	$F = 3.87$	2.1		12	17 (0 to 50)	
	<i>Bw10</i>	$F = 6.39$	3.4		13	47 (33 to 61)	
	<i>Bw13</i>	$F = 11.7$	6	BW	5	81 (73 to 89)	
	<i>Bw14</i>	$F = 25.9$	12.3	BW	7	28 (23 to 33)	
	<i>Bw15</i>	$F = 7.36$	3.8	BW	13	10 (3 to 16)	
	<i>Bw16</i>	$F = 7.52$	3.9	BW	11	14 (6 to 17)	
	<i>Afpq1</i>	$F = 6.17$	3.2		3	29 (23 to 37)	
	<i>Afpq2</i>	$F = 5.72$	3		4	66 (60 to 72)	
	<i>Afpq4</i>	$F = 4.25$	2.3		13	13 (0 to 46)	
	<i>Afpq5</i>	$F = 5.86$	3.1		11	9 (0 to 19)	
	<i>Afpq6</i>	$F = 8.92$	4.6	Abdominal fat (%)	17	36 (27 to 51)	
	<i>Afpq9</i>	$F = 18.5$	9.1	Abdominal fat (%)	7	22 (13 to 27)	
	<i>Afpq10</i>	$F = 7.48$	3.9	Abdominal fat (%)	12	18 (10 to 26)	
	<i>Afw1</i>	$F = 4.23$	2.3		3	30 (23 to 36)	
	<i>Afw2</i>	$F = 5.72$	3		4	66 (60 to 72)	
	<i>Afw3</i>	$F = 5.67$	3		5	80 (69 to 91)	
	<i>Afw5</i>	$F = 6.48$	3.4		11	12 (2 to 19)	
	<i>Afw6</i>	$F = 6.03$	3.2		13	11 (4 to 18)	
	<i>Afw7</i>	$F = 9$	4.7		17	39 (30 to 52)	
	<i>Afw9</i>	$F = 24.9$	12	Abdominal fat (%)	7	23 (16 to 28)	
	<i>Afw10</i>	$F = 8.56$	4.5	Abdominal fat (%)	12	21 (15 to 27)	
	<i>Afw11</i>	$F = 4.78$	2.5		X	17 (0 to 39)	
	<i>Lepq1</i>	$F = 7.58$	4.4	Leptin	14	28 (21 to 41)	
	<i>Abfp1</i>			Interacts with Abfp2 and Abfp3	17	34	
	<i>Abfp2</i>			Interacts with Abfp1	11	58	
	<i>Abfp3</i>			Interacts with Abfp1	8	16	
	<i>Abfp4</i>			Interacts with Abfp5	3	26	
	<i>Abfp5</i>			Interacts with Abfp4	5	20	
	<i>Abfw1</i>			Interacts with Abfw2, Abfw3, Abfw4	11	60	
	<i>Abfw2</i>			Interacts with Abfw1	4	64	
	<i>Abfw3</i>			Interacts with Abfw1	17	32	
	<i>Abfw4</i>			Interacts with Abfw1	19	43	
	<i>Abfw5</i>			Interacts with Abfw6	5	72	
	<i>Abfw6</i>			Interacts with Abfw5	12	6	
	<i>Bodw1</i>			Interacts with Bodw2	2	18	
	<i>Bodw2</i>			Interacts with Bodw1	11	55	
	<i>Bodw3</i>			Interacts with Bodw4	1	5	
	<i>Bodw4</i>			Interacts with Bodw3	9	34	
DuK × Du6	<i>Afw1</i>	$F = 4.52$	4	Abdominal fat weight	3	51	(643)

Table 3. (continued)

Cross	QTL	Scores	Variance (%)	Phenotypes	Animal chromosome	QTL peak	Reference
F × L	<i>Bw4</i>	$F = 4.79$	23.1	BW	11	42 (36 to 50)	(644)
	<i>Afw2</i>	$F = 4.89$	13.4	Abdominal fat	4	51 (34 to 63)	
	<i>Afpq2</i>	$F = 4.89$	10.2	Abdominal fat	4	55 (31 to 79)	
	<i>Afw5</i>	$F = 4.79$	8.3	Abdominal fat	11	6 (0 to 29)	
	<i>Afpq4</i>	$F = 4.7$	5.3	Abdominal fat	13	0	
	<i>Afpq1</i>	$F = 4.52$	8.3	Abdominal fat (%)	3	46 (24 to 76)	
	<i>Afpq3</i>	$F = 4.13$	4.7		5	51	
	<i>Afw3</i>	$F = 4.13$	3.9		5	61	
	<i>Afw4</i>	$F = 5.13$	4.1		9	29	
	<i>Afw6</i>	$F = 4.7$	7.7		13	0 (0 to 10)	
	<i>Afw7</i>	$F = 4.21$	2.9		17	46	
	<i>Afw8</i>	$F = 3.68$	18.1		19	26	
	<i>Bw5</i>	$F = 4.61$	7.1		1	14	
	<i>Bw6</i>	$F = 5.02$	5.1		2	56	
	<i>Bw7</i>	$F = 4.89$	7		4	55	
	<i>Bw8</i>	$F = 4.13$	3		5	42	
	<i>Bw9</i>	$F = 3.8$	4.3		12	49	
	<i>Bw10</i>	$F = 4.7$	10.1		13	34	
	<i>Bw11</i>	$F = 4.72$	4.2		15	6	
	<i>Bw12</i>	$F = 3.73$	0.2		X	42	
F × L	<i>Fob1</i>	LOD > 3.3	4.9	14-week fat (%)	2	45	(645,646)
	<i>Fob2</i>	LOD = 3.3	19.5	14-week fat (%) (in females)	12	19	
	<i>Fob3</i>	LOD = 11.3	14.4	14-week fat (%)	15	34	
	<i>Fob4</i>	LOD = 3.3	7.3	14-week fat (%)	X	37	
	<i>Fob3</i>	LOD = 11.3	14.4		15	(12 to 78)	
	<i>Fob3a</i>			Subcongenics of Fob3	15	27 (22 to 32)	
F.L. congenic	<i>Fob3b</i>			Subcongenics of Fob3; positional and expression candidate is Sqle (squalene epoxidase)	15	68 (44 to 72)	(645)
	<i>Fob3a</i>	$F = 13.7$	1.6	Fat (%); late-onset	15		
	<i>Fob3b</i>	$F = 11.6$	0.7	Fat (%); early onset	15		
ICR × M16	<i>Mfiq5</i>	LOD = 3.4	1.8		1	57 (34 to 103)	(647)
	<i>Mfe5q1</i>	LOD = 3.7	2.3		8	54 (1 to 81)	
	<i>Mfiq3</i>	LOD = 3.7	2		9	7 (7 to 60)	
	<i>Mfi5q1</i>	LOD = 4.4	2.3		9	60 (7 to 60)	
	<i>Mfi7q1</i>	LOD = 5.3	2.4		11	29 (15 to 74)	
	<i>Mfi8q1</i>	LOD = 4.8	2.2		11	32 (22 to 86)	
	<i>Mfiq1</i>	LOD = 8.1	4.7		11	34 (22 to 68)	
	<i>Mfe5q2</i>	LOD = 3.6	2.2		11	40 (18 to 73)	
	<i>Mfeq1</i>	LOD = 4.9	3		11	50 (18 to 73)	
	<i>Mfiq4</i>	LOD = 3.7	2		12	35 (17 to 63)	
	<i>Mfiq2</i>	LOD = 4.4	2.4		13	54 (26 to 54)	
	<i>Mlepq1</i>	LOD = 7.7	5.7		2	93 (83 to 104)	
	<i>Mlepq2</i>	LOD = 3.4	2.3		17	52 (15 to 68)	
	Not assigned	LOD = 16.8	7		2	84 (82 to 92)	
	Not assigned	LOD = 4	1.2		8	22 (1 to 82)	
	Not assigned	LOD = 5.2	1.8		11	51 (36 to 65)	
	Not assigned	LOD = 4.3	2		17	52 (22 to 58)	
	Not assigned	LOD = 12.1	4.8		2	83 (80 to 89)	
	Not assigned	LOD = 4.6	1.8		4	41 (2 to 62)	
	Not assigned	LOD = 3.6	1.5		11	58 (41 to 75)	
	Not assigned	LOD = 5.1	2		17	38 (18 to 57)	
	Not assigned	LOD = 14.2	6.5		2	85 (83 to 93)	
	Not assigned	LOD = 5.1	1.9		7	28 (8 to 51)	
	Not assigned	LOD = 6	2.6		11	58 (32 to 64)	
	Not assigned	LOD = 3.3	1.9		17	51 (14 to 63)	

Table 3. (continued)

Cross	QTL	Scores	Variance (%)	Phenotypes	Animal chromosome	QTL peak	Reference
ILS inbred, long sleep) × ISS (inbred short sleep)	<i>Wght1</i>	LOD = 4.6		Interacts with <i>Wght2</i>	1	78	(648)
	<i>Wght2</i>	LOD = 4.5		Interacts with <i>Wght1</i>	4	3.2	
	<i>Wght3</i>	LOD = 8.6			4	52.6	
	<i>Wght4</i>	LOD = 3.22			5	73	
	<i>Wght5</i>	LOD = 7.6			11	75	
	<i>Wght6</i>	LOD = 9.4			19	20	
KK/Ta × (BALB/c × KK/Ta)F1	<i>Tgls1</i>	LOD = 2.1		Triglyceride and BW	4	59	(649,650)
	<i>D17Mit218</i>	LOD = 2.9		BW	17	42	
	<i>Azgp1</i>	LOD = 2.3		BW candidate gene	5	78	
LG/J × SM/J	<i>Adip1</i>	LOD = 2.4		Adiposity (males)	1	11	(620,651–654)
	<i>Wta1</i>	LOD = 2.35		Late weight gain	4	6.5	
	<i>Wta2</i>	LOD = 3.49		Late weight gain	6	67	
	<i>Adip2</i>	LOD = 3.71		Adiposity/weight (females)	6	46.3	
	<i>Adip3</i>	LOD = 3.71		Adiposity (males)/weight	7	46.4	
	<i>Adip4</i>	LOD = 2.57		Adiposity	8	32	
	<i>Adip5</i>	LOD = 1.84		Adiposity	9	42	
	<i>Adip6</i>	LOD = 2.69		Adiposity (males)	12	45	
	<i>Adip7</i>	LOD = 1.9		Adiposity (males)	13	1 (0 to 30)	
	<i>Wta3</i>	LOD = 2.7		Weight	14	2.5	
	<i>Wta4</i>	LOD = 2.44			17	17.4	
	<i>Adip8</i>	LOD = 2.84		Adiposity	18	20 (8 to 38)	
	Not assigned				7		
	Not assigned	LOD = 3.7			7	66 (65.6 to 69)	
	Not assigned	LOD = 4.07			17	22.8 (17.7 to 24.2)	
	Not assigned	LOD = 3.68		Also epididymal, retroperitoneal, and mesenteric	1	62 (56.6 to 65)	
	Not assigned	LOD = 3.21	r	Also retroperitoneal, mesenteric, and leptin	8	59 (32 to 59)	
	Not assigned	LOD = 3.58	r	Also Retroperitoneal & mesenteric	10	63 (59 to 70)	
	Not assigned	LOD = 3.5		Also mesenteric and leptin	X	69	
M16i (rapid 3- to 6-week weight gain) × L6 (low 6-week weight)	Not assigned	LOD = 4.99			11	10.9 (1.1 to 17)	(655–658)
	<i>Scfq1</i>	LOD = 7.6	5.9	Fat, subcutaneous	2	84 (81.7 to 88.9)	
	<i>Scfq2</i>	LOD = 4.4	5	Fat, subcutaneous	15	25.2 (8.8 to 39.6)	
	<i>Scfq3</i>	LOD = 4.1	3.8	Fat, subcutaneous	11	24.9 (9.9 to 47.5)	
	<i>Scfq4</i>	LOD = 4.1	3.4	Fat, subcutaneous	17	21.9 (0 to 34)	
	<i>Scfpq1</i>	LOD = 3.9	12	Fat, subcutaneous, adjusted for 10-week weight	10	33.1 (20.5 to 55.3)	
	<i>Epfpq1</i>	LOD = 6	5.3		14	20.3 (0 to 34.5)	
	<i>Epfpq2</i>	LOD = 3.8	1		2	66.8 (52.2 to 72.7)	
	<i>Epfpq3</i>	LOD = 3.6	3.3		15	51.1 (46.3-ter)	
	<i>Epfpq4</i>	LOD = 3.4	3.4		15	33.4 (21.2 to 46.3)	
	<i>Epfq1</i>	LOD = 9.5	6.7		2	84 (79.8 to 87.6)	
	<i>Epfq2</i>	LOD = 4.6	4.3		2	97.6 (95.5 to 102.4)	
	<i>Epfq3</i>	LOD = 3.8	3		17	21.9 (0 to 33.2)	
	<i>Epfq4</i>	LOD = 3.5	2.5		11	17.4 (0 to 34.9)	
	<i>Epfq5</i>	LOD = 3.4	2.6		7	21.5 (0 to 35.9)	
	<i>W10q1</i>	LOD = 29	9.4		2	79.6 (76.8 to 81.7)	
	<i>W10q10</i>	LOD = 8.3	4.8		4	55.3 (41.1 to 61.8)	
	<i>W10q11</i>	LOD = 7.9	8		6	28.5 (20 to 47.2)	
	<i>W10q12</i>	LOD = 6.9	2.1		17	19.2	
	<i>W10q13</i>	LOD = 5.7	7		9	53.9 (44.5 to 61.4)	

Table 3. (continued)

Cross	QTL	Scores	Variance (%)	Phenotypes	Animal chromosome	QTL peak	Reference
MH × C57BL/6J	<i>W10q14</i>	LOD = 5.2			8	26.4 (19.2 to 41.4)	
	<i>W10q15</i>	LOD = 4.8	3.1		17	30.9 (24.5 to 41.1)	
	<i>W10q16</i>		2.8		5	42.8 (29.4 to 61.8)	
	<i>W10q17</i>	LOD = 4.2	1		13	55.4	
	<i>W10w18</i>	LOD = 4.1	1.9		7	18.2	
	<i>W10q2</i>	LOD = 26.4	8.1		11	17.7 (11.3 to 24.1)	
	<i>W10q3</i>	LOD = 15.5	7		3	29.7 (23.7 to 40.5)	
	<i>W10q4</i>	LOD = 14.9	9.5		3	47.7 (40.5 to 54)	
	<i>W10q5</i>	LOD = 10.2	5.3		10	31.7 (24.8 to 42.6)	
	<i>W10q6</i>	LOD = 9.7	3.9		1	56.3 (48.7 to 66.5)	
	<i>W10q7</i>	LOD = 9.1	3		1	72.6 (66.5 to 79)	
	<i>W10q8</i>	LOD = 10.2	5.3		10	31.7 (24.8 to 42.6)	
	<i>W10q9</i>	LOD = 8.6	8.6		2	50.8 (42.4 to 63.3)	
	<i>H1q1</i>	LOD = 5.6	4.7	Heat loss; confirmed in (MH × ML)F2 cross	1	127	(659)
	<i>H1q2</i>	LOD = 3.7	3.1	Heat loss	2	71	
	<i>H1q3</i>	LOD = 3.8	3.1	Heat loss	3	35	
	<i>H1q4</i>	LOD = 4.7	3.9	Heat loss	3	3.9	
	<i>H1q5</i>	LOD = 4.1	3.4	Heat loss	7	61	
	<i>Fatq1</i>	LOD = 7.4 to 8.0	5.4 to 5.9	Gonadal fat	1	62	
	<i>Batq1</i>	LOD = 3.96	3.3	Brown fat	1	102	
	<i>Batq2</i>	LOD = 3.46	2.8	Brown fat	3	55	
	<i>Wt10q1</i>	LOD = 4.25	3.3		1	25	
	<i>Wt10q2</i>	LOD = 4.76	3.8		3	61	
	<i>Wt10q3</i>	LOD = 3.63	2.9		11	32	
	<i>Wt3q1</i>	LOD = 5.13	4.1		1	72	
	<i>Wt3q2</i>	LOD = 10.09	8		1	108	
	<i>Wt3q3</i>	LOD = 6.28	5		17	14	
	<i>Wt6q1</i>	LOD = 4.02	3.3	Confirmed in (MH × ML)F2 cross	1	27	
	<i>Wt6q2</i>	LOD = 3.98	3.2		1	108	
	<i>Wt6q3</i>	LOD = 4.55	3.7		11	36	
Mhi (inbred, high food intake) × Lhi (inbred, low food intake)	Not assigned	$F = 10.47$	4.9		5	73 (66-telomere)	(660)
	Not assigned	$F = 10.48$	4.7		7	49 (35 to 68)	
	Not assigned	$F = 34.28$	14.4*	Non-Mendelian	8	7 (1 to 19)	
	Not assigned	$F = 7.98$	3.6		9	47	
NON/Lt × (NZO/H1Lt × NON/Lt)F1	Not assigned	$F = 7.02$	3.2		18	40	
	<i>Dbsty1</i>	LOD = 9.36		BW	1	21 (8.3 to 43.1)	(661)
	<i>Dbsty2</i>	LOD = 3.86		Adiposity index	5	43	
NZM/B1NJ × SM/J	<i>Dbsty3</i>	LOD = 4.88		Adiposity index	12	48 (45 to 53)	
	<i>Bfq1</i>	LOD = 3.6	36	Body fat	2	81	(662)
NZO × (SJL/x NZO)F1	<i>Nobq1</i>	LOD = 3.8	16.8	BMI (females)	5	32	(663–665)
SM/J × A/J	<i>Bwq3</i>	LOD = 4.6	6	BW at 10 weeks	8	56 (53 to 69)	(666)
	<i>Bwq4</i>	LOD = 4.8	6	BW at 10 weeks	18	28 (20 to 54)	
SM/J × NZO/H1Lt	<i>Obq4</i>	LOD = 6.3		Inguinal fat (%) (males)	17	8.7 (6.1 to 15.5)	(667)
	<i>Obq7</i>	LOD = 6		Mesenteric fat (%) (males)	1	28.7 (25.7 to 42)	
	<i>Obq8</i>	LOD = 6.4		Retroperitoneal fat (%)	1	61.9 (63.7 to 85.1)	
	<i>Obq9</i>	LOD = 6.7		Mesenteric fat (%) (females)	1	88.4 (82.4 to 92.7)	
	<i>Obq10</i>	LOD = 6.4		Gonadal fat (%) (males)	2	58.1 (50.7 to 67.4)	
	<i>Obq11</i>	LOD = 4.1		Gonadal fat (%)	5	10 (3.4 to 16.9)	
	<i>Obq12</i>	LOD = 4.5		Gonadal fat (%)	5	29 (21.9 to 36.1)	
	<i>Obq13</i>	LOD = 9.3		Mesenteric fat (%)	6	26.8 (20.7 to 29.4)	
	<i>Obq14</i>	LOD = 9.2		Mesenteric fat (%)	6	43.5 (39.4 to 46.9)	
	<i>Obq15</i>	LOD = 6.6		Gonadal fat (%) (males)	7	51.4 (44.2 to 52.4)	

Table 3. (continued)

Cross	QTL	Scores	Variance (%)	Phenotypes	Animal chromosome	QTL peak	Reference
(C57BL/6J × TH)F1 × TH and (CAST/Ei × TH)F1 × TH (TallyHo) × various	<i>Tabw</i>	LOD = 3.9			7	27.8 (0 to 44)	(668,669)
	<i>Tafat</i>	LOD = 3.1			4	69.8 (68 to 90)	
	<i>Tabw2</i>				6	38.5 (19.1 to 65.5)	
Sheep							
Texel × Texel	<i>GDF8</i>	p < 0.001	9.9% Less fat	Fat			(670)
Rat							
(OLETF × BN) × OLETF	<i>Dmo9</i>	LOD = 3.5		Adiposity index	11		(671)
	<i>Dmo1</i>	LOD = 8.2 to 14		BW	1		
	<i>Dmo4</i>	LOD = 4.4 to 5.5		Adiposity index	1		
	<i>Dmo7p</i>	LOD = 4.9 to 5.4		Adiposity index	7		
	<i>Dmo6p</i>	LOD = 3.5 to 3.6		Adiposity index	6		
	<i>Dmo5</i>	LOD = 3.5 to 3.6		Adiposity index	3		
	<i>Dmo10</i>	LOD = 3.5 to 3.6		BW	11		
BN × GK/Nidd/gk5	<i>Nidd/gk5 weight</i>	LOD = 4.19	13	Weight	8		(672)
Dahl × MNS	<i>DAHL3</i>	p = 0.00003	13	BW	3		(673)
F344 × OLETF	<i>Olep1</i>	LOD = 5.39	6.5	Leptin	2		(674)
	<i>Olep2</i>	LOD = 4.49	8	Leptin	6		
GK × BN	<i>Nidd/gk6</i>			BW	17		(675)
	<i>Nidd/gk1</i>			Adiposity	1		
	<i>bw/gk1</i>			BW	7		
	<i>Nidd/gk5</i>			BW	8		
GK × F344	<i>Niddm1</i>	LOD = 3.2	23.5	BW	1		(676)
	<i>Weight1</i>	LOD = 6.2			7		
	<i>Niddm3</i>	LOD = 3.0			10		
Lepr(fa)/Lepr(fa) 13M × WKY	<i>Qfa12</i>	LOD = 3	8.3	BMI, female	12		(677)
	<i>Qfa1</i>	LOD = 2.2	6.9	BMI, female	1		
OLETF × BN	<i>Dmo1</i>	LOD = 6	11.6	BW	1		(678)
OLETF × F344	<i>Niddm24</i>	LOD = 3.91		Also known as Nidd6/of	1Distal	D1Rat81-D1Rat90	(679)
	<i>Obs5</i>	LOD = 5.1		Obs5 narrowed to 10-cM interval	14	D14Rat23-D14Wox7	(680)
SHR × BB/OK	<i>SHR4</i>	LOD = 3.1	14	BW (females)	4		(681)
	<i>SHR1</i>	LOD = 3.3	32	BW (males)	1		
SHR × wild	<i>SHR10</i>	LOD = 3.5		BW (males)	10		(682)
WOKW × DA/K	<i>Wokw1/Q1ms5</i>	LOD = 4.5	16	BMI	5		(644,683)
	<i>Wokw1/Q1ms1</i>	LOD = 4.9	31	30-Week BW	1		
Pig							
Berkshire × Yorkshire	<i>SSC4:113</i>	F = 11.8	6	Weight	4		(684)
	<i>SSC7</i>	F = 13.8	6.9	Back fat	7		
	<i>SSC1</i>	F = 11.3	4.8	Back fat	1		
	<i>SSC5</i>	F = 9.5	4.8	Back fat	5		
Duroc × Berkshire	<i>SSC2 0</i>	F = 10.03		Back fat	2		(685)
	<i>SSC2 30</i>	F = 10.61		Fat (%)	2		
	<i>SSC2 37</i>	F = 7.34		Weight	2		
	<i>SSC6 110</i>	F = 7.39		Weight	6		
Duroc, Hampshire, Landrace × Meishan	<i>PigQTL2</i>	F = 7.9		Average back fat	7		(686,687)

Table 3. (continued)

Cross	QTL	Scores	Variance (%)	Phenotypes	Animal chromosome	QTL peak	Reference
Duroc, Hampshire, Landrace × Meishan	<i>HMGA1</i>	$p = 0.01$		Back fat	7		(669)
Large White × European wild boar	<i>FAT1</i>	$p = 0.0001$	9.7	Body fat (%)	4		(688–690)
Landrace × Iberian	<i>FAT1</i>	$F = 11.1$		Back fat depth	4		(691)
Landrace × Iberian	<i>FAT1</i>			Weight			(692)
Landrace × Iberian	<i>SSC6 60-100</i>	$p = 0.001$		Back fat thickness	6		(693)
	<i>SSC6 130-132/LEPR</i>	$p = 0.001$		Back fat thickness, intramuscular fat (%)	6		
Landrace × Iberian	<i>AFABP</i>			Fatness	4		(692)
Meishan × (Dutch Landrace × Large White)	<i>SSC7</i>	$F = 18$		Back fat thickness	7		(694,695)
Meishan × (Dutch Landrace × Large White)	<i>SSC2</i>	$F = 2.7$		Back fat thickness	2		(694,695)
Meishan × Duroc	<i>SSC6 102,7-116.7</i>	$F = 16.16$	4	Weight	6		(696)
	<i>SSC6 102,7-116.7</i>	$F = 12.65$	14	Weight, daily gain	6		
	<i>SSC7 56.2</i>	$F = 11.45$	12	Weight, daily gain	7		
	<i>SSC7 113.3</i>	$F = 13.6$	14	Fat, back fat thickness	7		
	<i>SSCX 74,6</i>	$F = 15.79$	16	Fat, intramuscular	X		
Meishan × Duroc	<i>HMGA2</i>	$p = 0.01$	14	Fat, back fat thickness	7		(669)
Dutch × Meishan	<i>SSC6q</i>	$F = 14.7$	0.1 to 0.2	Intramuscular fat	6q		(465,697)
	<i>SSC7</i>	$F = 49.4$		Back fat thickness	7		
	<i>SSC2</i>	$F = 24.1$		Back fat thickness	2		
	<i>SSC6p</i>	$F = 14.5$		Intramuscular fat	6p		
	<i>SSCX</i>	$F = 12.8$	0.1 to 0.2	Intramuscular fat	X		
Gottingen × Meishan	<i>SSC7</i>	$F = 19.5$	18	Back fat thickness	7		(698)
Large White × Meishan	<i>BFM4</i>			Midback fat depth	4		(699)
Meishan × Large White	<i>SSCX 67</i>	$p = 1.4 \times 10^{-15}$		Fat thickness at the loin	X		(700)
White × Meishan	<i>SSC4:49-84</i>	$F = 14.9$ to 15.3	3 to 4	Back fat thickness	4		(701,702)
	<i>SSC8</i>	$F = 9.5$	1 to 2	Back fat thickness	8		
	<i>SSC7</i>	$F = 10.4$ to 20.5	2 to 5	Back fat depth	7		
	<i>SSC1</i>	$F = 39.4$ to 94.9	1 to 2	Weight	1		
	<i>SSC5</i>	$F = 13.4$ to 15.1	2 to 5	Back fat thickness	5		
	<i>SSC6</i>	$F = 11.9$	1 to 2	Back fat thickness	6		
	<i>SSCX</i>	$F = 37.4$ to 71.8		Back fat depth	X		
White composite × Meishan	<i>SSC7</i>	$F = 14.7$		Back fat thickness	7		(703)
	<i>SSC1</i>	$F = 15.4$		Back fat thickness	1		
	<i>SSCX</i>	$F = 32.3$		Back fat thickness	X		
Hampshire, Landrace × Minghu	<i>PIT1</i>	$F = 3.34$		42-Day weight	13		(704)
Large White × Wild Boar	<i>IGF2q</i>	$F = 7.1$	10.4	Back fat depth	2p		(634,705)

ing of crosses between two strains define only a statistical probability of a polymorphic gene residing in a defined genetic interval. Follow-up studies are necessary to confirm this likelihood. Congenic (and subcongenic) strains have been generated for some of these QTLs, supporting the existence and magnitude of some of these phenotypes. Some cases include the characterization of the *Fob3* QTL (645,646). Congenic strains containing a chromosome 15 region from the lean L strain were introgressed onto the F genetic background. Interestingly the characterization of subcongenic lines suggests that *Fob3* contains two contributory regions: *Fob3a* and *Fob3b*, conferring late and early onset phenotypes, respectively. Expression analysis of genes positioned within the *Fob3b* segment by microarray screening identifies a candidate gene, *Sqle* (squalene epoxidase). This gene is involved in the regulation of cholesterol biosynthesis. Interestingly, the expression of other genes of the cholesterol biosynthesis pathway mapping outside of the *Fob3b* region are also perturbed, suggesting that the changes in activity of this pathway may be responsible for the phenotypic differences between the F parental and the F.L<Chr15> congenic strains. Other murine QTL regions for which candidate genes have been implicated include *Bw19* (*Gpc3*, Glypican 3) (606) and the QTL on chromosome 7 associated with adiposity (*ATP10a*, encodes ATPase, class V, type 10A) (706). A candidate gene for a rat QTL *Niddm24* is *Pnlip* (encodes pancreatic lipase). The continued generation of congenic mouse strains and expression screening and single nucleotide polymorphism genotyping analysis should continue to implicate specific genes with well-characterized QTL regions.

QTLs from Cross-Breeding Experiments Other Than Rodents

Syntenic regions in humans have been picked up directly from the original papers or determined from the U.S. Live-stock Genome Mapping Projects (NAGRP03). Four new chromosomes were targeted according to QTL analysis in chicken, one in pig, and one in sheep (Table 3). In a cross between Landrace and Iberian pig strains, a QTL for fatness was reported on pig chromosome 4 in the region of the *AFABP* gene (692) corresponding to the human fatty acid-binding protein 4 (adipocyte) gene located at 8q24. A QTL for fat was reported in a sheep Texel \times Texel cross at the growth differentiation factor 8 gene (670), which is located at 2q32.2 in humans. The main QTL region detected for fat on chicken chromosome 7 from the cross White Leghorn layer \times commercial broiler (591) corresponded to human chromosome 2q21. The cross of Rhode Island Red layer with itself produced a QTL for BW on chromosome 4 (707) corresponding to human chromosome 17q11.1-q12, whereas the White Plymouth Rock cross produced a QTL for weight on chromosome 5 (594) corresponding to human 22q13.1-q13.31

but also to 12p13-q23 reported in the cross WL \times RIR (593). Finally, a QTL for weight was reported on chicken chromosome 1 (596) that corresponds to human chromosome 21q22.

Associations with Candidate Genes

The evidence for associations between candidate genes and obesity-related phenotypes is summarized in Table 4. A total of 416 studies covering 127 candidate genes have reported significant associations. Of these, 57 studies (40 candidate genes) were published during the past year. This year's update includes 14 new candidate gene entries.

Associations with BW, BMI, Overweight, and Obesity

BW, BMI, overweight, and obesity were associated with DNA sequence variation in *ACE* (710,711), *ADIPOQ* (718–720), *ADRB2* (744), *BDNF* (814), *COMT* (822), *CYP11B2* (824), *DRD4* (836), *ENPP1* (839), *ESR1* (841), *ESR2* (841), *FOXC2* (850,851), *GAD2* (855), *GHRHR* (859), *HTR2C* (884), *LIPC* (951), *MC4R* (971), *MCHR1* (876,877), *NPY* (981), *NTRK2* (998), *NPY2R* (984), *PLIN* (1112), *PPARG* (1012,1021,1027), *PPARGC1A* (1042), *PYY* (984,1046), *RETN* (1051), *SERPINE1* (1055), *UCP1* (1084), and *VDR* (1110).

Associations with Body Composition and Fat Distribution Phenotypes

Body composition-related phenotypes (fat mass, fat-free mass, percentage body fat, sum of skinfolds) showed associations with markers in *ACE* (712), *UCP1* (1079), *LEPR* (937), *LIPC* (951), *PLIN* (1113), *PPARG* (1021), *GFPT1* (858), *AR* (809), *DIO1* (830), *IGF2* (899), *FOXC2* (850), and *COMT* (822). Phenotypes reflecting body fat distribution [abdominal visceral and subcutaneous fat, waist-to-hip ratio (WHR), waist circumference, sagittal diameter] were associated with *ACE* (710), *ADIPOQ* (719), *ADRB2* (744), *APOA2* (792), *FABP2* (847), *LTA* (964), *MTTP* (976), *PLIN* (1113), *PPARG* (1021), and *UCP1* (1079).

Associations with Changes in BW and Body Composition

Eight studies reported associations between seven candidate genes and changes in BW and body composition. The *ADRB1* (736), *NMB* (978), and *PPARG* (1016) loci showed associations with spontaneous changes in BW and adiposity over time. Markers in the *PPARG* (1032) gene were reported to be associated with exercise training-induced weight loss, whereas sequence variation in the *APOA5* (797) and *MC4R* (972) loci modified weight loss in response to a low-fat diet and bariatric surgery, respectively. The *ADIPOQ* (721) and *LEPR* (949) loci were reported to be associated with changes in BW during a 3-year diabetes prevention trial with acarbose.

Table 4. Evidence for association between markers of candidate genes with obesity-related phenotypes

Gene	Location	Subjects	Phenotype	<i>p</i>	Reference
ABCC8	11p15.1	232 Cases	Obesity, morbid	0.02	(708)
ABCG5	2p21	262 Cases	BMI	0.05	(709)
ACE	17q24.1	1009 Men	BMI	0.012	(710)
		964 Men	Waist circumference	0.0023	(710)
		956 Subjects	Overweight (Blacks from U.S.)	0.03	(711)
		1059 Subjects	Overweight (Blacks from Nigeria)	0.04	(711)
		956 Subjects	Obesity (Blacks from U.S.)	0.02	(711)
		1059 Subjects	Obesity (Blacks from Nigeria)	0.04	(711)
		922 Subjects	Body fat (%) (physically active Health ABC subjects)	0.05	(712)
		959 Cases	Overweight	0.014	(713)
		186 Cases	BMI	0.04	(714)
ACPI	2p25	75 Cases	BMI (in children)	0.02	(715)
		265 Cases	BMI (in type 2 diabetic subjects)	0.002	(716)
ADA	20q13.12	273 Cases	BMI (in type 2 diabetic subjects)	0.0004	(717)
ADIPOQ	3q27	194 Subjects	BMI	0.017	(718)
		811 Subjects, 45 families	BMI (Hispanic families from IRAS)	0.004	(719)
		811 Subjects, 45 families	Waist circumference (Hispanic families from IRAS)	0.001	(719)
		811 Subjects, 45 families	Abdominal visceral fat (Hispanic families from IRAS)	0.01	(719)
		100 Subjects, 100 women	BMI (women with polycystic ovarian syndrome)	0.01	(720)
		770 Subjects	Weight change during acarbose trial (STOP-NIDDM trial cohort)	0.043	(721)
		4479 Cases	3-year increase in BMI	0.033	(722)
		4479 Cases	3-year increase in waist-to-hip ratio	0.01	(722)
		103 Cases	BMI	0.03	(723)
		995 Cases	Obesity	0.047	(724)
		413 Cases	Body weight, waist circumference (in Japanese, in whites)	0.03	(725)
		371 Cases	BMI	0.02	(726)
		95 Cases	BMI (in obese women)	0.014	(727)
		95 Cases	Sagittal abdominal diameter (in obese women)	0.032	(727)
		245 Cases	BMI	0.05	(728)
ADRA2A	10q24-q26	213 Cases	Skinfolds, trunk-to-extremity ratio (in Blacks)	0.04	(729)
		72 Cases	Skinfolds, trunk-to-extremity ratio (in women)	0.002	(730)
		476 Cases	Abdominal total fat	0.003	(731)
		476 Cases	Abdominal subcutaneous fat	0.012	(731)
		93 Cases, 49 men, 44 women	Weight change (Chinese schizophrenic under anti-psychotic)	0.023	(732)
ADRA2B	2q11.2	166 Cases	Basal metabolic rate (in obese non-diabetics)	0.01	(733)
		126 Cases	Body weight, change, 5-year (in non-diabetics)	0.04	(734)
ADRB1	10q24-q26	931 Cases	BMI, body weight, fat mass	0.05	(735)
		760 Subjects	BMI increase during 15-year follow-up	0.018	(736)
ADRB2	5q31-q32	239 Cases	Waist-to-hip ratio	0.05	(737)
		180 Cases	BMI	0.003	(738)
		494 Cases	Body weight, increase (in men)	0.01	(739)
		141 Cases	Catecholamine-induced lipolysis in adipocytes	0.01	(740)
		247 Cases	BMI, change (in women)	0.04	(741)
		247 Cases	Fat mass, change (in women)	0.0008	(741)
		247 Cases	Body fat (%) change (in women)	0.0003	(741)
		230 Cases	Skinfolds, sum of eight (in men)	0.03	(741)
		236 Cases	Lipolysis	0.02	(742)
		508 Cases	BMI (in Japanese)	0.001	(743)
		272 Subjects	BMI (African Americans from IRAS)	0.001	(744)
		992 Subjects	BMI (whole IRAS cohort)	0.045	(744)
		992 Subjects	Waist-to-hip ratio (whole IRAS cohort)	0.0001	(744)
		948 Subjects	Abdominal visceral fat (whole IRAS cohort)	0.0001	(744)
		140 Cases	BMI, fat mass, fat cell volume	0.001	(745)
		826 Cases	BMI, obesity, waist-to-hip ratio, waist circumference, hip circumference	0.05	(746)
		366 Cases	BMI (in women)	0.01	(747)
		836 Cases	BMI, body weight, waist-to-hip ratio, waist circumference, hip circumference (in French men)	0.002	(748)

Table 4. (continued)

Gene	Location	Subjects	Phenotype	<i>p</i>	Reference
ADRB3	8p12-p11.2	63 Cases	BMI, fat mass	0.05	(749)
		277 Cases	BMI (in Japanese men)	0.004	(750)
		1576 Cases	BMI	0.02	(751)
		284 Cases	Leptin	0.03	(752)
		224 Cases	BMI (in men)	0.01	(731)
		24 Cases	Leptin, body weight, increase, skinfolds, sum of eight	0.03	(753)
		286 Cases	Body weight, increase	0.04	(754)
		574 Cases	BMI (in Japanese)	0.009	(755)
		185 Cases	Body weight, increase over 20 years, weight, current	0.007	(756)
		313 Cases	Obesity (in those 20 to 35 years old)	0.05	(757)
		476 Cases	BMI (in men)	0.05	(758)
		553 Cases	Obesity (in Japanese children)	0.02	(759)
		179 Cases	BMI	0.006	(760)
		295 Cases	BMI	0.05	(761)
		695 Cases	BMI	0.001	(762)
		83 Cases	BMI (in coronary artery disease patients)	0.05	(763)
		211 Cases	Obesity, moderate	0.02	(764)
		53 Cases	Obesity	0.05	(765)
		350 Cases	BMI	0.009	(766)
		398 Cases	BMI, abdominal subcutaneous fat, abdominal visceral fat	0.02	(767)
		154 Cases	Obesity (in sedentary individuals)	0.05	(768)
		46 Cases	5-year weight gain	0.05	(769)
		586 Cases	BMI, hip circumference (in women)	0.03	(770)
		56 Cases	BMI, fat mass, waist circumference	0.05	(771)
		128 Cases	Body weight, increase over 25 years	0.01	(772)
		63 Cases	BMI	0.001	(773)
		63 Cases	Abdominal visceral fat	0.001	(773)
		63 Cases	Abdominal subcutaneous fat	0.001	(773)
		1675 Cases	BMI, obesity, body fat (%)	0.05	(774)
		254 Cases	Obesity, early onset	0.002	(775)
		76 Cases	Fat mass (in Thai men)	0.05	(776)
		131 Cases	Fat mass, abdominal visceral fat	0.01	(777)
		261 Cases	BMI	0.05	(778)
		979 Cases	Waist-to-hip ratio, overweight (in men >53 years old)	0.05	(779)
		802 Cases	BMI	0.02	(780)
		224 Cases	BMI (in men)	0.02	(731)
		49 Cases	BMI	0.03	(781)
		335 Cases	Waist-to-hip ratio (in women)	0.02	(782)
AGRP	16q22	47 Cases	BW (in obese children)	0.05	(783)
		183 Cases	BMI, body fat (%), fat mass (in whites)	0.003	(784)
		253 Cases	BMI	0.015	(785)
		212 Cases	Fat mass	0.028	(785)
		212 Cases	Body fat (%)	0.013	(785)
		874 Cases	Body weight	0.02	(786)
		874 Cases	BMI	0.01	(786)
		874 Cases	Fat-free mass	0.002	(786)
AGT	1q42.2	874 Cases	Fat mass	0.04	(786)
		135 Cases	Body weight, change	0.006	(787)
		316 Cases	Waist-to-hip ratio	0.007	(788)
		57 Cases	Adipocyte size	0.01	(789)
		106 Cases	Adipocyte size	0.02	(789)
APOA1	11q23.3	94 Cases	Fat mass (in women >42 years old)	0.008	(790)
		482 Cases	BMI (in type 2 diabetics)	0.048	(791)
APOA2	1q23.1	482 Cases	Waist-to-height ratio (in type 2 diabetics)	0.023	(791)
		122 Women	Abdominal visceral fat (white women)	0.05	(792)
APOA4	11q23.3	624 Cases	Waist circumference	0.03	(793)
		369 Cases	BMI	0.003	(794)
		375 Cases	BMI, waist-to-hip ratio (in young men)	0.004	(795)
APOA5	11q23	613 Cases	BMI, body fat (%)	0.004	(796)
		606 Subjects, 606 women	Weight loss on a 3-month low-fat diet	0.0021	(797)

Table 4. (continued)

Gene	Location	Subjects	Phenotype	<i>p</i>	Reference
APOB	2p24.2	56 Cases	Body fat (%), abdominal fat	0.04	(798)
		232 Cases	BMI	0.005	(799)
		181 Cases	BMI	0.05	(800)
APOC3	11q23.1-q23.2	270 Cases	Obesity	0.05	(801)
APOD	3q26.2-qter	114 Cases	BMI	0.006	(802)
APOE	19q13.32	1775 Cases	BMI	0.01	(803)
		405 Cases	Fat mass	0.002	(804)
		405 Cases	Body fat (%)	0.003	(804)
		405 Cases	Lean mass	0.004	(804)
		164 Cases	Waist circumference (in women with a family history of diabetes)	0.05	(805)
AR	Xq11.2-q12	64 Cases	Body fat (%), leptin (in women)	0.02	(806)
		131 Cases	BMI	0.043	(807)
		113 Cases	Waist circumference (in women)	0.002	(808)
		294 Men	Fat-free mass	0.027	(809)
		112 Men	Fat-free mass	0.049	(809)
ATP1A2	1q23.1	106 Cases	Body fat (%)	0.01	(810)
		122 Cases	Body fat (%), respiratory quotient	0.05	(811)
		156 Cases	Respiratory quotient (in young adults)	0.0001	(812)
		12 Cases	Fat mass	0.01	(813)
BDNF	11p13	12 Cases	Body weight	0.05	(813)
CAPN10	2q37.3	249 Subjects	Minimum lifetime BMI (PO trios with restricting AN)	0.019	(814)
		148 Cases	adrb3 activity in adipocytes (in overweight individuals)	0.004	(815)
CART	5q13.2	286 Cases	BMI	0.003	(816)
		612 Cases	Waist-to-hip ratio (in men)	0.002	(817)
CBFA2T1	8q21.3	528 Cases	BMI, obesity	0.008	(818)
		281 Cases	BMI, body fat (%), waist circumference, hip circumference	0.0002	(819)
CCKAR	4p15.2-p15.1	1296 Cases	Leptin, body fat (%)	0.003	(820)
CNTFR	9p13.2	465 Cases	Fat-free mass	0.011	(821)
COMT	22q11.21	246 Subjects	Height (pre-/early pubertal girls)	0.001	(822)
		246 Subjects	Body weight (pre-/early pubertal girls)	0.009	(822)
		246 Subjects	Total lean mass (pre-/early pubertal girls)	0.004	(822)
CRHR1	17q12-q22	83 Cases	Exercise training-induced percentage body fat loss	0.05	(823)
		503 Cases	BMI	0.0083	(14)
CYP11B2	8q24.3	190 Subjects	BMI (normotensive highlanders from India)	0.002	(824)
		100 Subjects	BMI (hypertensive highlanders from India)	0.004	(824)
CYP19A1	15q21.1	125 Cases	Sagittal abdominal diameter (in women)	0.049	(825)
		300 Cases	BMI	0.01	(826)
		83 Cases	Exercise training-induced body fat loss	0.01	(823)
CYP2D6	22q13.1	83 Cases	Exercise training-induced percentage body fat loss	0.01	(823)
		11 Cases, 11 men	BMI change percentage (white schizophrenic under anti-psychotic)	0.01	(827)
CYP7A1	8q12.1	1102 Cases	BMI	0.05	(828)
DF	19p13.3	24 Cases	Abdominal fat (in monozygotic twins)	0.05	(829)
DIO1	1p32	350 Subjects	Fat-free mass	0.03	(830)
DRD2	11q23.2	392 Cases	Body weight	0.002	(831)
		176 Cases	Obesity	0.002	(832)
		320 Cases	Energy expenditure, 24-hour, sleeping metabolic rate	0.03	(833)
		383 Cases	Skinfolds, iliac, skinfolds, triceps	0.002	(834)
		990 Cases	Obesity	0.03	(835)
DRD4	11p15.5	128 Subjects, 128 women	Maximal lifetime BMI (women with seasonal affective disorder)	0.001	(836)
ENPP1	6q23.1	103 Cases	Maximal lifetime BMI	0.007	(837)
		293 Cases	Leptin	0.01	(838)
		1225 Cases, 1205 controls	Obesity (obese children and controls)	1×10^{-5}	(839)
ESR1	6q25.1	184 Families	Obesity	0.01	(839)
		108 Cases	BMI (in post-menopausal women)	0.04	(840)
		295 Subjects	BMI	0.024	(841)
		551 Cases	BMI (in middle-aged women)	0.05	(842)

Table 4. (continued)

Gene	Location	Subjects	Phenotype	<i>p</i>	Reference
ESR2	14q23.2	551 Cases	Body fat (%) (in middle-aged women)	0.05	(842)
		551 Cases	Waist circumference (in middle-aged women)	0.05	(842)
		216 Cases	Obesity, android type	0.0002	(843)
FABP1	2p11	295 Subjects	BMI	0.02	(841)
FABP2	4q27	130 Cases	BMI	0.05	(844)
		130 Cases	Waist circumference	0.005	(844)
		714 Cases	BMI	0.042	(845)
FASN	17q25	507 Cases	BMI, body fat (%)	0.01	(846)
		120 Subjects, 120 women	Total abdominal fat (white women)	0.004	(847)
		120 Subjects, 120 women	Subcutaneous abdominal fat (white women)	0.03	(847)
		395 Cases	Abdominal fat	0.008	(848)
		214 Cases	Body fat (%)	0.002	(849)
FOXC2	16q22-q24	174 Cases	Respiratory quotient, 24-hour	0.04	(849)
		174 Cases	24-hour carbohydrate oxidation	0.03	(849)
		127 Cases, 127 controls	Obesity	0.027	(850)
		223 Cases, 231 controls	Obesity	0.043	(850)
		388 Subjects	BMI (Swedish type 2 diabetics)	0.03	(851)
GAD2	10p11.23	388 Subjects	Fat mass (Swedish type 2 diabetics)	0.04	(851)
		644 Cases	BMI	0.03	(852)
		215 Cases	Body fat (%)	0.02	(852)
		724 Cases	Waist-to-hip ratio	0.04	(853)
		575 Cases	Morbid obesity, eating behavior	0.0049	(854)
GCGR	17pter	575 Cases	Morbid obesity, eating behavior	0.014	(854)
		477 Cases, 614 controls	Obesity (obese children and controls)	0.043	(855)
		559 Subjects	Birth weight (obese children)	0.009	(855)
		950 Cases	Waist-to-hip ratio, waist girth, sagittal abdominal diameter	0.001	(856)
		58 Cases	Body weight at birth (in men)	0.002	(857)
GKPT1	7p15.3-p15.1	164 Subjects, 164 men	Fat (%)	0.009	(858)
GHRHR	7p14	1095 Subjects, 178 families	Obesity	0.025	(859)
GHRL	3p26-p25	1418 Subjects	Obesity (MONICA Augsburg cohort)	0.002	(859)
		300 Cases	Obesity age of onset	0.003	(860)
		65 Cases	BMI (in tall obese children)	0.001	(861)
		192 Cases	Obesity (in women)	0.05	(862)
		737 Cases	Obesity (in men)	0.01	(863)
GNB3	12p13.31	294 Cases	Weight gain during pregnancy	0.006	(864)
		230 Cases	BMI (in primiparous women)	0.01	(865)
		20 Cases	Lipolysis	0.01	(866)
		111 Cases	Weight loss with sibutramine	0.0013	(867)
		213 Cases	BMI, waist circumference, hip circumference, skinfolds (in Nunavut Inuit)	0.05	(868)
		181 Cases	Body weight at birth	0.02	(869)
		130 Cases	BMI	0.001	(870)
		250 Cases	Fat mass, change, body fat, change (%)	0.006	(871)
		114 Cases	Lipolysis (subcutaneous, adrenoceptor-mediated)	0.004	(872)
		197 Cases	BMI (in hypertensives)	0.02	(873)
		1950 Cases	BMI, body weight (in men, white, Chinese, and African)	0.001	(874)
		774 Cases	BMI	0.03	(875)
		774 Cases	Body fat (%)	0.02	(875)
		134 Cases, 80 men, 54 women	Weight change (Chinese schizophrenic under anti-psychotic)	0.003	(732)
GPR24	22q13.2	469 Cases, 1127 controls	Obesity (French obese children)	0.006	(876)
		719 Cases, 326 controls	Obesity	0.0016	(877)
GYS1	19q13.33	130 Cases	Obesity	0.03	(878)
HSD11B1	1q32-q41	263 Cases	BMI (in children)	0.005	(879)
		263 Cases	Waist circumference (in children)	0.05	(879)
		263 Cases	Waist-to-hip ratio (in children)	0.05	(879)
HSD3B1	1p11.2	132 Cases	Skinfolds, sum of six, 12-year change	0.04	(811)
HSPA1B	6p21.31	517 Cases	Obesity	0.0002	(880)
HTR1B	6q14.1	98 Cases	BMI (in women with bulimia nervosa)	0.001	(881)

Table 4. (continued)

Gene	Location	Subjects	Phenotype	<i>p</i>	Reference
HTR2A	13q14.11	276 Cases	Dietary energy, carbohydrate and alcohol intake (in obese subjects)	0.028	(882)
		264 Cases	BMI, waist-to-hip ratio, sagittal abdominal diameter	0.015	(883)
HTR2C	Xq24	293 Cases, 481 controls	Obesity	0.0001	(884)
		224 Cases	Obesity	0.008	(885)
		117 Cases	Body weight, gain, anti-psychotic-induced	0.0003	(886)
		148 Cases	Body weight, loss (in teenage women)	0.0001	(887)
		589 Cases	BMI	0.009	(888)
		73 Cases, 45 men, 28 women	Weight change (from 58 white/22 African-American schizophrenic under anti-psychotic)	0.05	(889)
		42 Cases, 34 men, eight women	BMI change 10% (white schizophrenic under anti-psychotic)	0.004	(890)
		41 Cases, 26 men, 15 women	BMI change (%) (5 white/35 African American/1 Hispanic schizophrenic under anti-psychotic)	0.05	(891)
		41 Cases, 26 men, 15 women	BMI change 7% (5 white/35 African American/1 Hispanic schizophrenic under anti-psychotic)	0.003	(891)
		58	BMI change 9 months; white schizophrenic under anti-psychotic	0.03	(892)
		117	BMI change; Chinese schizophrenic under anti-psychotic	0.0003	(886)
		32	BMI change; Chinese schizophrenic under anti-psychotic	0.02	(893)
IDE	10q23-q25	724 Cases	BMI	0.0067	(894)
IGF1	12q23.3	502 Cases	Body fat (%), fat-free mass, fat mass, change	0.05	(895)
IGF2	11p15.5	2734 Cases	Body weight	0.01	(896)
		1474 Cases	BMI	0.02	(897)
		427 Cases	Fat mass	0.05	(898)
		206 Women	Fat-free mass	0.05	(899)
IL6	7p21	271 Cases	BMI (in men)	0.007	(900)
		271 Cases	Waist circumference (in men)	0.01	(900)
		124 Cases	Fasting energy expenditure	0.012	(901)
		124 Cases	Energy expenditure during hyperinsulinemic clamp	0.007	(901)
		3376 Cases	BMI	0.027	(902)
		3376 Cases	Body weight change during a 3.5-year follow-up	0.03	(902)
		242 Cases	Fat-free mass	0.02	(903)
		571 Cases	BMI	0.009	(904)
		485 Cases	BMI	0.003	(905)
		74 Cases	BMI	0.03	(905)
IL6R	1q22	184 Cases	Obesity (in women)	0.05	(906)
		700 Cases	BMI	0.003	(907)
		700 Cases	BMI	0.001	(907)
		700 Cases	BMI	0.004	(907)
		700 Cases	BMI	0.02	(907)
		700 Cases	BMI	0.02	(907)
INS	11p15.5	758 Cases	Body weight	0.009	(908)
		2734 Cases	Body weight	0.001	(896)
		431 Cases	BMI	0.043	(909)
		431 Cases	Waist circumference	0.015	(909)
		238 Cases	Obesity	0.05	(910)
		1152 Cases	BMI	0.0002	(911)
		1207 Cases	Body weight	0.02	(912)
		1207 Cases	BMI	0.03	(912)
		1207 Cases	Waist circumference	0.03	(912)
		52 Cases	Waist-to-hip ratio (in obese women)	0.005	(913)
INSR	19p13.3-p13.2	75 Cases	Obesity (in hypertensives)	0.05	(914)
IRS1	2q36.3	304 Cases	BMI	0.001	(915)
		304 Cases	Waist-to-hip ratio	0.001	(915)
		156 Cases	Leptin (in obese subjects)	0.03	(916)
		1748 Cases	BMI (in African Americans)	0.04	(917)
IRS2	13q34	233 Cases	BMI	0.02	(918)
		233 Cases	Body fat (%)	0.01	(918)
		233 Cases	Waist circumference	0.004	(918)

Table 4. (continued)

Gene	Location	Subjects	Phenotype	<i>p</i>	Reference
LDLR	19p13.2	83 Cases	BMI (in normotensives)	0.008	(919)
		131 Cases	BMI, skinfolds, subscapular, skinfolds, triceps, arm fat index	0.001	(920)
		270 Cases	Obesity	0.02	(921)
		84 Cases	BMI (in hypertensives)	0.004	(922)
		112 Cases	BMI (in hypertensives)	0.04	(923)
LEP	7q31.3	103 Cases	BMI, body weight	0.005	(924)
		395 Cases	Leptin	0.02	(925)
		39 Cases	Leptin secretion	0.05	(926)
		738 Cases	Obesity	0.011	(927)
		738 Cases	BMI	0.028	(927)
		233 Cases	Leptin (in obese women)	0.02	(928)
		211 Cases	Obesity (in women)	0.05	(929)
		117 Cases	Leptin	0.04	(930)
		168 Cases	Body weight, decrease	0.006	(931)
		84 Cases	Body weight	0.05	(932)
		73 Cases, 55 men, 18 women	BMI change 9 months (white schizophrenic under anti-psychotic)	0.03	(892)
		128 Cases, 38 controls, 61 men, 67 women	BMI change (Chinese schizophrenic under anti-psychotic)	0.003	(933)
		128 Cases, 38 controls, 61 men, 67 women	Abdominal subcutaneous fat change (Chinese schizophrenic under anti-psychotic)	0.009	(933)
LEPR	1p31	502 Cases	BMI, fat mass	0.005	(934)
		308 Cases	Fat-free mass	0.03	(935)
		335 Cases	BMI, body weight, fat mass (in women)	0.01	(936)
		103 Subjects	Body fat (%)	0.02	(937)
		405 Cases	Fat mass	0.015	(938)
		405 Cases	Lean mass	0.002	(938)
		179 Cases	BMI, fat mass, body weight, loss (in overweight women)	0.006	(939)
		336 Cases	Overweight/obesity	0.009	(940)
		220 Cases	Leptin, BMI, fat mass (in post-menopausal women)	0.0001	(941)
		267 Cases	BMI, sagittal abdominal diameter	0.04	(942)
		600 Cases	BMI > 25	0.007	(943)
		130 Cases	Obesity, extreme (in children)	0.02	(944)
		268 Cases	Energy expenditure, 24-hour	0.02	(945)
		184 Cases	Adipocyte size, subcutaneous abdominal	0.02	(945)
		20 Cases	Body fat (%)	0.003	(946)
		62 Cases	Abdominal total fat, abdominal subcutaneous fat	0.03	(947)
		118 Cases	BMI	0.01	(948)
		770 Subjects	BMI change during 3-year follow-up (STOP-NIDDM trial cohort)	0.009	(949)
		770 Subjects	Waist circumference change during 3-year follow-up (STOP-NIDDM trial cohort)	0.006	(949)
LIPC	15q21–23	230 Cases	BMI	0.002	(950)
		234 Cases	Waist circumference	0.002	(950)
		231 Cases	Abdominal visceral fat	0.03	(950)
		1070 Subjects	BMI	0.02	(951)
		1070 Subjects	Body fat (%)	0.03	(951)
LIPE	19q13.2	257 Cases	BMI, body fat (%), fat mass, skinfolds, sum of eight (in white and black women)	0.005	(952)
		117 Cases	Waist-to-hip ratio, lipolysis	0.02	(953)
		405 Cases	Obesity (in women)	0.05	(954)
		405 Cases	Body fat (%) (in women)	0.05	(954)
		380 Cases	Obesity	0.002	(955)
		110 Cases	BMI (in women)	0.012	(956)
LMNA	1q23.1	48 Cases	Leptin, lipodystrophy, leptin-to-BMI ratio	0.05	(957)
		306 Cases	Leptin, BMI, waist-to-hip ratio (in Canadian Oji-Cree)	0.05	(958)
		47 Cases	Familial partial lipodystrophy	0.0001	(959)
		186 Cases	BMI, body weight, waist circumference, skinfolds, subscapular	0.002	(960)

Table 4. (continued)

Gene	Location	Subjects	Phenotype	p	Reference
LPL	8p21.3	587 Cases	BMI (in women)	0.02	(758)
		249 Cases	Body fat (%), fat mass, BMI, change (in white women)	0.01	(961)
		236 Cases	BMI	0.05	(962)
LRPAP1	4p16.3	235 Cases	Abdominal obesity	0.045	(963)
LTA	6p21.3	5630 Subjects	Waist circumference	0.009	(964)
MACS2	16p12.3	1976 Cases	BMI	0.009	(965)
		1976 Cases	Waist-to-hip ratio	0.0011	(965)
MAOA	Xp11.4-p11.3	50 Cases	BMI > 35	0.005	(966)
MC3R	20q13.2-q13.3	314 Cases	BMI, body fat (%), fat-free mass, fat mass, respiratory quotient (in normal-weight and overweight individuals)	0.0005	(967)
		244 Cases	Leptin (in morbidly obese subjects)	0.05	(968)
		156 Cases	BMI, body fat (%), fat-free mass, fat mass (in women)	0.003	(969)
		520 Cases	Obesity	0.017	(970)
		1135 Cases	Obesity (in children and adolescents)	0.006	(33)
		332 Subjects	BMI (offspring of the Quebec Family Study)	0.002	(971)
		426 Cases	Severe obesity	0.04	(38)
		174 Subjects	Weight loss after bariatric surgery (severely obese patients undergoing bariatric surgery)	0.003	(972)
		268 Cases	BMI, waist-to-hip ratio	0.023	(973)
		229 Cases	Resting energy expenditure	0.007	(974)
MC5R	18q22	156 Cases	BMI, body fat (%), fat-free mass, fat mass (in women)	0.002	(969)
MED12	Xq13.1	68 Cases	Obesity	0.001	(975)
MTTP	4q24	258 Subjects	Abdominal visceral fat	0.005	(976)
NCOA3	20q13.13	301 Cases	BMI (in post-menopausal women with breast cancer)	0.01	(977)
NMB	15q25	291 Subjects	6-year change in BMI	0.037	(978)
		291 Subjects	6-year change in waist circumference	0.018	(978)
		291 Subjects	6-year change in percentage body fat	0.017	(978)
NPR3	5p14-p13	787 Cases	BMI	0.048	(979)
		787 Cases	Waist-to-hip ratio	0.022	(979)
NPY	7p15.1	595 Cases	BMI, waist-to-hip ratio	0.03	(980)
		907 Subjects	BMI (non-obese Swedish subjects)	0.005	(981)
		369 Cases	Body weight at birth	0.03	(982)
NPY2R	4q31	952 Cases	BMI	0.017	(983)
		952 Cases	Waist-to-hip ratio	0.013	(983)
		100 Cases, 67 controls, 167 men	Severe obesity (male Pima Indians)	0.002	(984)
NPY5R	4q31-q32	74 Cases	Obesity (in Pima Indians)	0.05	(985)
NR0B2	1p35.3	294 Cases	Body weight at birth	0.05	(986)
		809 Cases	BMI (in 7-year-olds)	0.05	(986)
		809 Cases	Waist circumference (in 7-year-olds)	0.01	(986)
		305 Cases	BMI (in women)	0.05	(986)
		217 Cases	Obesity, early onset	0.009	(987)
NR3C1	5q31	51 Cases	Abdominal visceral fat (in lean subjects)	0.003	(988)
		279 Cases	BMI (in obese subjects)	0.04	(989)
		135 Cases	Waist-to-hip ratio (in men)	0.01	(990)
		262 Cases	Leptin, BMI, waist-to-hip ratio, waist circumference	0.001	(991)
		369 Cases	Overweight (in type 2 diabetics)	0.003	(992)
		83 Cases	Skinfolds, sum of (in girls)	0.01	(993)
		480 Cases	Abdominal visceral fat	0.001	(994)
		12 Cases	Body weight, gain	0.01	(995)
		1963 Cases	BMI	0.002	(996)
		1963 Cases	Waist-to-hip ratio	0.02	(996)
		370 Cases	BMI	0.05	(996)
		337 Cases	Lean mass	0.02	(997)
NTRK2		164 Subjects	Minimum lifetime BMI (Spanish eating disorder patients)	0.001	(998)
PGR	11q22.2	301 Cases	BMI (in post-menopausal women with breast cancer)	0.005	(977)
PLIN	15q26	117 Cases	Lipolysis in adipocytes (in obese women)	0.0008	(999)
		1538 Cases	BMI	0.004	(1000)
		1538 Cases	BMI	0.004	(1000)
		351 Subjects, 351 women	Body fat (%)	0.016	(1113)

Table 4. (continued)

Gene	Location	Subjects	Phenotype	<i>p</i>	Reference
PNMT POMC	17q21.2 2p22-p21	351 Subjects, 351 women	Body fat (%)	0.014	(1113)
		351 Subjects, 351 women	Waist circumference	0.02	(1113)
		351 Subjects, 351 women	Waist circumference	0.045	(1113)
		123 Cases, 623 controls	Obesity (Malays from Singapore)	0.05	(1112)
		77 Cases, 521 controls	Obesity (Indians from Singapore)	0.05	(1112)
		149 Cases	Weight loss (in women)	0.006	(1002)
		75 Cases	Leptin (in obese children)	0.03	(860)
		337 Cases	Leptin (in Mexican Americans)	0.001	(1003)
		118 Cases	Leptin (in lean subjects)	0.003	(1004)
		114 Cases	BMI	0.045	(1005)
PON1 PON2	7q21.3 7q21.3	100 Cases	Body weight at birth (in Trinidadian neonates and South Asians)	0.05	(1006)
PPARA	22q13.31	698 Cases	BMI	0.023	(1007)
		570 Cases	Body fat (%)	0.028	(1007)
		154 Cases	BMI (in type 2 diabetics)	0.02	(1008)
PPARD	6p21.2-p21.1	178 Cases	BMI	0.03	(1009)
		179 Cases	BMI	0.04	(1009)
PPARG	3p25	414 Cases	BMI	0.039	(1010)
		921 Cases	Leptin, BMI, waist circumference (in Mexican Americans)	0.02	(1011)
		203 Subjects	BMI (Javanese non-diabetics)	0.0016	(1012)
		333 Cases	BMI (in the middle-aged)	0.03	(1013)
		973 Cases	BMI (in the elderly)	0.02	(1013)
		422 Cases	BMI	0.03	(1014)
		752 Cases	BMI, change (in obese men)	0.002	(1015)
		869 Cases	BMI, change (in lean men)	0.008	(1015)
		1954 Subjects	BMI over 15 years (whites of the CARDIA study)	0.01	(1016)
		1954 Subjects	Waist circumference over 15 years (whites of the CARDIA study)	0.01	(1016)
		1844 Subjects	BMI over 15 years (Blacks of the CARDIA study)	0.05	(1016)
		464 Cases	BMI, obesity	0.01	(1017)
		619 Cases	BMI	0.04	(1018)
		41 Cases	BMI	0.02	(1019)
		41 Cases	Fat mass	0.02	(1019)
		451 Cases	BMI (in overweight Blacks)	0.02	(1020)
		451 Cases	Waist-to-hip ratio (in overweight Blacks)	0.01	(1020)
		451 Cases	Waist circumference (in overweight Blacks)	0.004	(1020)
		1051 Subjects	BMI	0.012	(1021)
		1051 Subjects	Waist-to-hip ratio	0.001	(1021)
		1051 Subjects	Fat mass	0.003	(1021)
		1051 Subjects	Body fat (%)	0.025	(1021)
		228 Cases	Obesity, morbid	0.02	(1022)
		119 Cases	Weight, increase, 10-year	0.009	(1023)
		225 Cases	Weight, decrease, 3-year	0.04	(1024)
		140 Cases	BMI	0.05	(1025)
		838 Cases	BMI, body weight, waist circumference, height	0.002	(1026)
		1133 Subjects	BMI	0.036	(1027)
		820 Cases	Leptin (in obese subjects)	0.001	(1028)
		183 Cases	Lipid oxidation, 24-hour	0.03	(1029)
		183 Cases	Lipid balance, 24-hour	0.02	(1029)
		70 Cases	Weight, increase	0.01	(1030)
		100 Cases	BMI	0.0012	(1031)
		29 Subjects	Endurance training-induced weight loss (healthy offspring of type 2 diabetics)	0.05	(1032)
		311 Cases	Ponderal index at birth	0.007	(1033)
		311 Cases	Body weight gain	0.001	(1033)
		121 Cases	BMI	0.03	(1034)
		714 Cases	BMI	0.04	(1035)
		596 Cases	Fat mass	0.009	(1035)
		685 Cases	Waist circumference	0.03	(1035)

Table 4. (continued)

Gene	Location	Subjects	Phenotype	<i>p</i>	Reference
PPARGC1A	4p15.31	501 Cases	Abdominal visceral fat	0.01	(1035)
		501 Cases	Abdominal subcutaneous fat	0.001	(1035)
		268 Cases	BMI	0.022	(1036)
		3080 Cases	BMI	0.037	(1037)
		3080 Cases	BMI	0.036	(1037)
		375 Cases	Obesity, severe, with early onset	0.05	(1038)
		141 Cases	BMI, body weight, fat mass, waist circumference, lean body mass, hip circumference	0.002	(1039)
		467 Cases	Fat mass (in Austrian women)	0.005	(1040)
		467 Cases	BMI (in Austrian women)	0.006	(1040)
		467 Cases	Waist circumference (in Austrian women)	0.01	(1040)
		467 Cases	Hip circumference (in Austrian women)	0.03	(1040)
		201 Cases	Adipocyte size (in Pima Indians)	0.04	(1041)
		165 Cases	Lipid oxidation, 24-hour (in Pima Indians)	0.03	(1041)
		165 Cases	Lipid balance, 24-hour (in Pima Indians)	0.004	(1041)
		156 Subjects	BMI	0.031	(1042)
PTPN1	20q13.1-q13.2	1553 Cases	BMI	0.0146	(1043)
		1553 Cases	BMI	0.018	(1043)
PTPRF	1p34	257 Cases	BMI	0.03	(1044)
		589 Cases	BMI	0.03	(1045)
PYY	17q21.1	589 Cases	Waist circumference	0.01	(1045)
		100 Cases, 67 controls, 167 men	Severe obesity (male Pima Indians)	0.001	(984)
RETN	19p13.2	6022 Subjects	Overweight	0.018	(1046)
		777 Cases	Body weight	0.005	(1047)
		777 Cases	Waist circumference	0.001	(1047)
		777 Cases	BMI	0.019	(1047)
		773 Cases	Waist circumference	0.026	(1047)
		411 Cases	BMI, obesity	0.0097	(1048)
		814 Cases	BMI	0.01	(1049)
		814 Cases	Waist circumference	0.048	(1049)
		12 Cases	Overfeeding-induced increase in abdominal visceral fat	0.033	(1050)
		320 Subjects, 320 women	BMI (women with polycystic ovary syndrome)	0.02	(1051)
SAH	16p13.11	4059 Cases	BMI	0.0066	(1052)
SCARB1	12q24.31	288 Cases	BMI (in healthy lean women)	0.004	(1053)
		228 Cases	Obesity, morbid	0.002	(1022)
SERPINE1	7q21.3-q22	1098 Cases	Abdominal subcutaneous fat	0.0265	(1054)
		472 Women	BMI (women from the Quebec Family Study cohort)	0.009	(1055)
		505 Cases	Obesity	0.002	(1056)
SGK	6q23	263 Cases	BMI	0.008	(1057)
SLC6A14	Xq23-q24	1267 Cases	Obesity	0.0001	(1058)
		1267 Cases	Obesity, eating behavior	0.013	(1058)
		299 Cases	Obesity	0.0002	(1059)
		1805 Cases	Obesity	0.003	(1059)
		90 Cases	Obesity (in black smokers)	0.006	(1060)
SORBS1	10q24.1	770 Cases	Obesity	0.05	(1061)
		114 Cases	BMI	0.008	(1005)
SREBF1	17p11.2	807 Cases	Obesity	0.038	(1062)
		807 Cases	Obesity	0.006	(1062)
TCF1	12q24.31	203 Cases	BMI (in young early onset diabetics)	0.0024	(1063)
TGFB1	19q13.31	405 Cases	Lean mass	0.002	(804)
		284 Cases	BMI (in Swedish men)	0.05	(1064)
		284 Cases	Sagittal abdominal diameter (in Swedish men)	0.05	(1064)
TH	11p15.5	2734 Cases	Body weight	0.0014	(896)
TNF	6p21.3	176 Cases	BMI	0.01	(1065)
		159 Cases	BMI	0.01	(1066)
		159 Cases	Body fat (%)	0.05	(1066)
		159 Cases	Waist circumference	0.05	(1066)
		136 Cases	Waist circumference (in women)	0.04	(1067)
		38 Cases	Body fat (%)	0.02	(1068)

Table 4. (continued)

Gene	Location	Subjects	Phenotype	p	Reference
TNFRSF1B	1p36.21	1351 Cases	BMI	0.004	(1069)
		378 Cases	BMI, body fat (%) (in women)	0.02	(1070)
		1047 Cases	Obesity	0.04	(1071)
		363 Cases	BMI	0.01	(1072)
		110 Cases	Obesity	0.02	(1073)
		217 Cases	Leptin, BMI	0.05	(1074)
		396 Cases	Fat mass	0.026	(1075)
		396 Cases	Body fat (%)	0.001	(1075)
		396 Cases	Waist-to-hip ratio	0.034	(1075)
		163 Cases	Body weight, decrease, BMI, decrease	0.05	(1076)
UCP1	4q28-q31	526 Cases	BMI (in overweight women)	0.02	(1077)
		162 Cases	Waist-to-hip ratio	0.003	(1078)
		387 Subjects	Waist-to-hip ratio	0.008	(1079)
		387 Subjects	Body fat (%)	0.014	(1079)
		113 Cases	Body weight (in Japanese women)	0.001	(1080)
		99 Cases	Body weight, change (in pre-menopausal women)	0.048	(1081)
		22 Cases	High-fat meal-induced thermogenesis	0.01	(1082)
		123 Cases	Fat, increase (in high-weight gainers)	0.05	(1083)
		172 Subjects, 172 women	Obesity	0.002	(1084)
		24 Cases	Body weight, resting metabolic rate	0.05	(1085)
UCP2	11q13.3	60 Cases	Energy expenditure, 24-hour, spontaneous physical activity, 24-hour, sleeping spontaneous physical activity, respiratory quotient, 24-hour non-protein, fat oxidation, 24-hour	0.005	(1086)
		220 Cases	BMI (in South Indian women)	0.02	(1087)
		791 Cases	BMI	0.03	(1088)
		596 Cases	Obesity	0.007	(1088)
		813 Cases	Obesity	0.002	(1089)
		949 Cases	Obesity	0.006	(1090)
		147 Cases	Resting energy expenditure	0.05	(1091)
		147 Cases	Glucose oxidation rate at rest	0.02	(1091)
		147 Cases	Lipid oxidation rate at rest	0.02	(1091)
		307 Cases	Obesity	0.01	(1092)
UCP3	11q13	41 Cases	Body weight, increase, fat mass, increase (in peritoneal dialysis patients)	0.05	(1093)
		82 Cases	BMI, metabolic rate, 24-hour sleeping (in those >45 years old)	0.007	(1094)
		63 Cases	BMI	0.028	(1095)
		105 Cases	BMI, body fat (%), body weight, fat mass, overweight (%), skinfolds, sum of four	0.001	(1096)
		120 Cases	BMI, respiratory quotient, lean body mass, respiratory quotient, non-protein, fat oxidation (in African Americans)	0.008	(1097)
		116 Cases	Waist-to-hip ratio (in South Indian women, in European women)	0.03	(1098)
		722 Cases	Fat mass	0.004	(1099)
		722 Cases	Lean mass	0.013	(1099)
		722 Cases	BMI	0.023	(1099)
		722 Cases	Body fat (%)	0.049	(1099)
VDR	12q13.11	419 Cases	BMI	0.004	(1100)
		73 Cases	Resting energy expenditure (in Black women)	0.01	(1101)
		734 Cases	Leptin, BMI, body fat (%), fat mass, skinfolds, sum of six	0.0005	(1102)
		393 Cases	Skinfolds, sum of eight	0.01	(1103)
		434 Cases	BMI	0.01	(1104)
		401 Cases	BMI (in morbidly obese subjects)	0.0037	(1105)
		382 Cases	Body weight, BMI, current, BMI, maximum	0.02	(1106)
		24 Cases	Body weight, resting metabolic rate	0.01	(1085)
		64 Cases	Leptin, body fat (%) (in women)	0.03	(806)
		153 Cases	Fat mass	0.05	(1107)
		153 Cases	Body weight	0.05	(1107)

Table 4. (continued)

Gene	Location	Subjects	Phenotype	<i>p</i>	Reference
		588 Cases	BMI	0.009	(1108)
		302 Cases	BMI	0.01	(1109)
		302 Cases	Fat-free mass	0.002	(1109)
		260 Women	BMI	0.042	(1110)
		309 Cases	BMI (in early onset type 2 diabetics)	0.0058	(1111)

Negative Associations with Obesity-Related Phenotypes

In addition to the positive studies summarized above, we identified 92 studies dealing with 58 genes in which there was no evidence of associations between DNA sequence variations and obesity-related phenotypes. Among these studies, the most frequent ones were those pertaining to markers of *PPARG* (1012,1114–1126) (14 studies), *ADIPOQ* (1127–1130), *ADRB3* (1084,1121,1131,1132), *IL6* (1129,1133–1135) (four studies each), and *ESR1* (1136–1138) (three studies). Other markers yielding negative findings were those related to *ACE* (710,1032), *ACTN* (1139), *ADIPOR1* (1140), *ADIPOR2* (1140), *ADRB1* (1132), *ADRB2* (1132), *AGER* (1141), *AHSG* (1142), *APOA4* (1143), *APOE* (1144,1145), *AR* (1146), *BDNF* (1147), *CASQ1* (1148), *COL1A1* (1134), *CRP* (1149), *ENPP1* (1150), *FABP2* (1151), *GNAS* (1152), *GNB3* (1152,1153), *GPR40* (1154), *H6PD* (1155), *HSD11B1* (1155,1156), *ICAM1* (1157), *IGF1* (1158), *IL6R* (1159), *INS* (1160,1161), *KCNJ11* (1120), *KL* (1146), *LEP* (1129), *LEPR* (1162), *LIPC* (1163), *LPL* (1164), *LTA* (964), *MKKS* (1165), *MT-DLOOP* (1166), *MTHFR* (1167), *MTTP* (1168), *NOS3* (1169,1170), *NPY* (1171), *NR0B2* (1172), *PARD6A* (1173), *PLIN* (1174), *PPARGC1A* (1115,1175), *PRDM2* (1176), *PTPNI* (1177), *SCD* (1178), *SELE* (1179), *TAS2R38* (1180), *TNF* (1181), *UCP1* (1084), *UCP2* (1182,1183), *UCP3* (1182,1184), and *VDR* (1134,1185).

Drug-Induced BW Gain and Obesity

Unintentional weight gain and weight loss are potential side effects associated with several pharmacological therapies. In previous editions of the human obesity gene map, these studies were summarized within the association studies section. However, because the number of reports addressing the contribution of DNA sequence variation in specific candidate genes to the drug-induced weight changes has increased, these studies will be reviewed in a specific section from now on.

Drug-induced weight gain and obesity have been observed after insulin therapy in patients with type 1 or 2 diabetes; in psychiatric therapy using anti-psychotics, anti-depressants, or mood stabilizers; in neurological treatments with anti-epileptic drugs; and in hypertension or steroid hormone therapies (for review, see 1186). Drug-induced weight changes could range from a loss of weight to a gain

of >50 kg in patients on anti-epileptic, anti-depressant, or anti-psychotic medication (1186). Because modest weight losses of 5% to 10% of initial BW are clinically significant (1187), it is clear that even modest weight gain is an undesirable side-effect of drugs.

Response to anti-psychotic treatment is considered to be a complex trait in which many genes, each with a small effect, are expected to play a role (1188). Few genes have yet to be studied in relation to BW gain under anti-psychotics (Table 4). The functional $-759C>T$ variant (1189) in the serotonin receptor 2C gene (*HTR2C*) was studied in Chinese anti-psychotic-naïve schizophrenic patients. Carriers of the $-759T$ variant showed three times lower anti-psychotic-induced weight gain than those not carrying the *T* allele (886). This result was confirmed in anti-psychotic-naïve Chinese men (893) but not in a third sample of anti-psychotic-resistant Chinese (1190). However, in a group of anti-psychotic-resistant African-American, white, and Hispanic individuals, the association of the $-759T$ variant with a smaller weight gain was confirmed recently (891), as in anti-psychotic-naïve whites (892). In contrast, Basile et al. (889) reported that carriers of the $-759T$ allele gained more weight than non-carriers in a mixed population of anti-psychotic-resistant white and African-American patients. A Cys23Ser variant of the *HTR2C* locus showed no association with BW gain in clozapine-treated anti-psychotic-naïve or resistant schizophrenics of white or African American descent (1191–1193).

A significant effect of the cytochrome P450, subfamily IID, polypeptide 6 (CYP2D6) genotypes on the percentage change of BMI was reported in white men taking olanzapine and carrying the poor *4 and intermediate *1/*3 metabolizer genotypes (827,1194). On the other hand, no association with BW changes in African Americans and whites taking clozapine was observed with a dinucleotide repeat polymorphism of the cytochrome P450 subfamily I, polypeptide 2 (CYP1A2) gene (1191). Chinese anti-psychotic-naïve schizophrenic homozygotes for the *A* allele of the $-2548A>G$ polymorphism of the *LEP* gene showed higher changes in BW than patients carrying *A/G* and *G/G* genotypes (933). An opposite result was observed in anti-psychotic-naïve whites showing a higher BMI change in homozygotes for the *G* allele (892). In two recent studies on Chinese schizophrenic patients treated with clozapine, the

G/G homozygotes of the $-1291C>G$ variant of the adrenergic $\alpha 2A$ receptor (*ADRA2A*) locus showed a 3 times greater weight gain than the C/C genotype (732). Furthermore, a 2 to 3 times greater weight gain was reported in the TT genotype of the *GNB3* 825C>T variant in contrast to carriers of the C allele (1195). Negative results were reported previously for these two genes (1191,1196). Finally, 12 genes showed negative results with anti-psychotic-induced weight changes. Those were the tumor necrosis factor α (1191), the serotonin 1A and 2A receptors, the histamine H1 and H2 receptors, the $\beta 3$ and $\alpha 1a$ -adrenergic receptors (1191,1192,1196,1197), the serotonin transporter and the serotonin receptor 6 (1192), the dopamine receptor 4 (1198), the cytochrome P450 1A2 (*CYP1A2*), which is different from the *CYP2D6* that had shown some association, and the 25-kDa synaptosomal-associated protein (1199).

Treatment with lithium has long been recognized to be associated with adverse metabolic effects, notably weight gain (1200). No evidence for an association has been observed between two polymorphisms ($+35A>G$ in intron 3 and $+7T>G$ in intron 10) in the α subunit of the olfactory G-protein G_{olf} gene and weight gain in response to lithium treatment (1201). The combination of glitazones with insulin may favor weight gain due to enhanced adipogenesis. Patients with the *PPARG* Pro12Ala genotype show a better response to rosiglitazone treatment than those with the Pro12Pro genotype do, with no difference in weight or BMI (1202).

Human QTLs

Linkage Studies

Linkage studies with obesity-related phenotypes are summarized in Table 5. During the past year, 11 linkage studies were published: nine genome scans, one bivariate linkage analysis of metabolic syndrome phenotypes with markers on chromosome 7q (1203), and a meta-analysis of genome-wide linkage studies for BMI (1204).

Two genome scans for eating-related phenotypes were reported last year. The first was a genome scan for total caloric and macronutrient intakes assessed from a food frequency questionnaire in 816 subjects from the San Antonio Family Heart Study (1235). Evidence of linkage was found on chromosome 2p22-p21 near marker D2S1346 for total caloric intake and intakes of fat, saturated fat, and protein (LOD scores ranging from 2.09 to 2.62). The second was a genome scan of eating behaviors assessed from the Three-Factor Eating questionnaire in 660 subjects from the Quebec Family Study (978). Evidence of linkage was found on chromosomes 15q21-q23 (*LIPC*), 15q24-q25 (*D15S206*), and 17q22-q24 (*D17S1306*, *D17S1290*, *D17S1351*) for susceptibility to hunger and on chromosome 19p13 (*D19S215*) for disinhibition.

A genome-wide linkage analysis of obesity associated with the use of anti-psychotics in patients treated for psy-

choses was performed in 508 subjects from 21 multigenerational kindreds (1258). Obesity diagnosed from medical files was found to be 2.5 times more prevalent in patients treated with anti-psychotics than in untreated family members. Linkage with obesity and a set of 470 microsatellite markers was tested only in pedigrees with at least two occurrences of obesity. Evidence of linkage with obesity was found on chromosomes 6p23 (*D6S260*; LOD = 1.72), 8q22-q23 (*D8S1136*; LOD = 1.93), 9q34 (*D9S282*; LOD = 1.71), and 12q23.1-q24.23 (*D12S1279*-*D12S366*; LOD = 2.74).

Four genome scans reporting linkages with BMI and body fatness phenotype were published during the past year. In a study performed in West African families with type 2 diabetes (1236), linkage analysis of BMI and body composition assessed by bioelectric impedance revealed evidence of three QTLs affecting body fatness chromosomes 2p16-p13.3 (*D2S2739*-*D2S441*), 4q24 (*D4S1647*-*D4S2623*), and 5q14.3 (*D5S1725*). All linkages with BMI showed LOD scores below 1.7 (1236). A second genome scan for loci linked to BMI and percentage body fat assessed from bioelectric impedance was conducted in 3383 subjects from 1124 hypertensive African-American and white families (1227). Linkage to BMI and percentage body fat was tested separately in men and women and also in the combined sample. In the combined sample, evidence of linkage was found on chromosome 3q13.33 for BMI (LOD = 2.8) and on chromosome 12q24.3 for percentage body fat (LOD = 3.3). QTLs influencing both BMI and percentage body fat were found over a broad region [102 to 200 centimorgans (cM)] on chromosome 3 in men (3p12.2, 3q13.33, 3q26.33, and 3q27.3). Evidence of linkage with percentage body fat was also found on chromosomes 7q36.1 (LOD = 1.8), 15q25.3 (LOD = 3.0), and 18p11.22-p11.23 (LOD = 1.7) in men. In women, QTLs affecting percentage body fat were found on chromosomes 2p24.2 (LOD = 1.8), 12q24-q24.32 (LOD = 3.8), and 21q21.2 (LOD = 1.8), whereas linkage with BMI was found on chromosome 11p13 (LOD = 1.8). The third study was undertaken in a European-American sample of 1297 subjects from 260 families with the aim of detecting imprinted genetic loci influencing obesity-related traits (1224). Parent-specific linkage analyses of overweight (BMI ≥ 27), obesity (BMI ≥ 30), and obesity-related quantitative traits [BMI, percentage body fat, and waist circumference (WC)] were performed with 391 microsatellite markers. Several QTLs influencing obesity were uncovered: a paternal effect for BMI and WC on 2p25.1, a maternal effect for percentage body fat on 3p24, a paternal effect for BMI on 3q12.3, a maternal effect for obesity on 9q22.33, a maternal effect for overweight on 10p12.2, a paternal effect for percentage body fat on 11q12 and 11q13.3, a maternal effect for BMI and WC on 12q24.21, a maternal effect for overweight on 13q13.3, and a paternal effect for BMI and WC on 13q31.3. The fourth scan was

Table 5. Evidence for the presence of linkage with obesity-related phenotypes

Gene/marker	Location	Population	Phenotypes	Score	Reference
D1S468	1p36.32	1249 sibpairs, >10,000 relative pairs	BMI	LOD = 2.75	(1205)
		758 subjects, 53 pedigrees	BMI	LOD = 2.32	(1206)
		994 subjects, 37 pedigrees	BMI	LOD = 2.5	(1207)
D1S508	1p36.23-p36.22	994 subjects, 37 pedigrees	BMI	LOD = 2.2	(1207)
PGD	1p36.22	>168 pairs	Skinfolds, suprailiac	$p = 0.03$	(1208)
D1S552	1p36.13	893 sibpairs	BMI (in whites)	LOD = 2.03	(1209)
ATCT051	1p36	320 subjects, 154 families	BMI	MLS = 2.14	(1210)
D1S3721	1p34.1	157 subjects, 7 families	BMI (in whites)	$p = 0.0099$	(1211)
D1S193	1p34.1	202 to 251 pairs, 137 sibships of adult brothers and sisters	BMI	$p = 0.03$	(1212)
D1S197	1p33	202 to 251 pairs, 137 sibships of adult brothers and sisters	Insulin level, fasting	$p = 0.05$	(1212)
D1S200	1p32.2	202 to 251 pairs, 137 sibships of adult brothers and sisters	Fat mass	$p = 0.009$	(1212)
			BMI	$p = 0.04$	
D1S476	1p32.2	202 to 251 pairs, 137 sibships of adult brothers and sisters	Insulin level, integrated, after oral glucose tolerance test	$p = 0.02$	(1212)
			BMI	$p = 0.05$	
			Fat mass	$p = 0.02$	
			Skinfolds, sum of six	$p = 0.02$	
LEPR-IVS16CTTT	1p31.2	268 to 324 pairs	Fat-free mass	$p = 0.007$	(935)
			Fat mass	$p = 0.03$	
LEPR-IVS3CA	1p31.2	268 to 324 pairs	BMI	$p = 0.04$	(935)
			Fat mass	$p = 0.04$	
			Skinfolds, sum of six	$p = 0.02$	
			Fat-free mass	$p = 0.05$	
LEPR-Q223R	1p31.2	268 to 324 pairs	Fat mass	$p = 0.005$	(935)
			BMI	$p = 0.02$	
			Skinfolds, sum of six	$p = 0.04$	
			Fat-free mass	$p = 0.05$	
D1S1665	1p31.1	198 subjects, 18 pedigrees	Leptin	LOD = 3.4	(1213)
D1S550	1p31.1	236 pairs	Respiratory quotient, 24-hour (in Pima Indians)	LOD = 2.8	(1214)
D1S2737	1p31.1	342 families	Trends in BMI from childhood to adulthood	LOD = 2.2	(1215)
LEPR	1p31	302 subjects, 57 families, 545 sibpairs	Blood glucose, fasting (in Mexican Americans)	$p = 0.018$	(1216)
			Blood pressure, diastolic (in Mexican Americans)	$p = 0.003$	
D1S1631	1p21.2	514 subjects, 99 families, 347 sibships	Total energy intake	$p = 0.0002$	(1217)
			Carbohydrate intake	$p = 0.0026$	
			Fat intake	$p = 2^{-05}$	
AMPD1	1p13.2	514 subjects, 99 families, 347 sibships	Total energy intake	$p = 0.0005$	(1217)
			Fat intake	$p = 6^{-05}$	
D1S2726	1p12	342 families	Trends in BMI from childhood to adulthood	LOD = 2.5	(1215)
D1S534	1p11.2	769 subjects, 182 families	BMI (in Africans)	LOD = 2.24	(1218)
		514 subjects, 99 families, 347 sibships	Total energy intake	$p = 0.0008$	(1217)
			Fat intake	$p = 0.00038$	
		521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 2.3	(1219)
S100A1	1q21	514 subjects, 99 families, 347 sibships	Fat intake	$p = 0.001$	(1217)
D1S1679	1q21-1q22	3027 subjects, 401 families, 317 sibships	BMI (National Heart, Lung, and Blood Institute Family Heart Study)	LOD = 1.8	(1220)
D1S394	1q21.1	514 subjects, 99 families, 347 sibships	Fat intake	$p = 0.00081$	(1217)
ATP1A2	1q23.1	582 subjects, 171 families, 289 pairs	Respiratory quotient	$p = 0.02$	(812)
		295 subjects, 164 families	Adipocyte size	LOD = 1.7	(1221)
D1S194-D1S196	1q23.1-q23.2	897 subjects, 179 families, 2127 relative pairs	Waist circumference	MLS = 3.71	(1222)
ATP1B1	1q23.3	94 pairs	Respiratory quotient	$p = 0.04$	(811)

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
D1S222	1q31.1	514 subjects, 99 families, 347 sibships	Fat intake	$p = 0.0002$	(1217)
D1S456	1q32.1	313 subjects, 126 families, 99 sibships	Protein intake (%) (in Blacks)	$p = 0.0021$	(1217)
D1S517	1q43	313 subjects, 126 families, 99 sibships	Sucrose intake (in Blacks)	$p = 0.0018$	(1217)
D1S204	1q44	313 subjects, 126 families, 99 sibships	Sucrose intake (in Blacks)	$p = 0.0054$	(1217)
D2S2976	2p25.3	2086 subjects, 330 pedigrees	Waist circumference	LOD = 2.06	(1223)
D2S2952	2p25.1	1297 subjects, 260 families	Waist circumference, paternal	LOD = 2	(1224)
D2S1400	2p25.1	1297 subjects, 260 families	BMI, paternal	LOD = 2.45	(1224)
ACPI	2p25	300 pairs	BMI	$p = 0.004$	(1225)
		>168 pairs	Skinfolds, triceps	$p = 0.02$	(1208)
D2S1360	2p24.2	1297 subjects, 260 families	BMI	LOD = 1.7	(1226)
		3383 subjects, 1124 families	Body fat (%) (women)	LOD = 1.8	(1227)
D2S2337	2p24.1	264 sibpairs	Leptin (in French whites)	LOD = 2	(1228)
D2S165	2p23.3	1100 subjects, 170 families	Adiponectin (in Northern Europeans)	LOD = 2.7	(1229)
		264 pairs	Leptin	LOD = 2.4	(1230)
D2S367	2p23.1	1100 subjects, 170 families	Adiponectin (in Northern Europeans)	LOD = 2.7	(1229)
		264 pairs	Leptin	LOD = 2.7	(1230)
D2S1788	2p22.3	5000 relative pairs	Leptin	LOD = 4.9	(1231)
			Fat mass	LOD = 2.8	
		337 subjects	Leptin	LOD = 7.5	(1003)
		1778 sibships	BMI	$p = 0.0006$	(1232)
		349 subjects, 66 pedigrees	BMI (in whites)	LOD = 3.08	(1233)
		720 subjects, 230 families	Leptin	$p = 0.008$	(1234)
			BMI	$p = 0.008$	
D2S1346	2p22-p21	816 subjects, 42 families	Total energy intake	LOD = 2	(1235)
			Protein intake	LOD = 2.22	
			Fat intake	LOD = 2.09	
			Saturated fat intake	LOD = 2.62	
D2S1356	2p22-p21	1778 sibships	BMI	$p = 0.0004$	(1232)
D2S1352	2p16.3	1778 sibships	BMI	$p = 0.0004$	(1232)
D2S2739	2p16	321 relative pairs	Body fat (%)	LOD = 3.3	(1236)
D2S2739-D2S441	2p16-2p13.3	321 relative pairs	Fat mass	LOD = 2.56	(1236)
D2S441	2p13.3	453 subjects, 99 families	Abdominal subcutaneous fat	LOD = 1.88	(1237)
IGKC	2p11.2	>168 pairs	Skinfolds, triceps	$p = 0.03$	(1208)
D2S293-D2S383	2q12.2-2q14.3	430 subjects, 27 sibpairs, 27 pedigrees	BMI	LOD = 2.9	(1238)
D2S160	2q13	1249 sibpairs, >10,000 relative pairs	BMI	LOD = 2.56	(1205)
D2S410	2q14.1	2086 subjects, 330 pedigrees	Waist circumference	LOD = 2	(1223)
D2S347	2q14.3	1249 sibpairs, >10,000 relative pairs	BMI	LOD = 4.04	(1205)
			Body fat (%)	LOD = 1.91	
			Fat mass	LOD = 2.03	
		758 subjects, 53 pedigrees	BMI	LOD = 3.42	(1206)
D2S1334	2q21.3	453 subjects, 99 families	Abdominal visceral fat	LOD = 1.97	(1237)
D2S1399	2q23.3	453 subjects, 99 families	Abdominal visceral fat	LOD = 2.3	(1237)
D2S112-D2S396	2q33.2-2q36.3	506 subjects, 115 pedigrees	BMI > 99th percentile	LOD = 2.73	(1239)
			BMI > 97th percentile	LOD = 2.08	
D2S434	2q35	453 subjects, 99 families	Abdominal visceral fat	LOD = 2.5	(1237)
D2S1363-D2S1279	2q35-2q36.3	2467 subjects, 387 families	BMI	LOD = 2.4	(1240)
			Waist-to-hip ratio	LOD = 1.72	
			Subscapular skinfold	LOD = 2.55	
D3S2387	3p26.3	320 subjects, 154 families	BMI	MLS = 3.67	(1210)
		215 subjects, 105 families	Abdominal subcutaneous fat	LOD = 2.16	(1237)
D3S1259	3p25.2	1055 pairs	BMI	LOD = 2	(1241)
D3S3608	3p25.2	624 subjects, 28 families	Eating behavior, restraint (in Old Order Amish)	LOD = 2.5	(1242)
D3S2403	3p24	1297 subjects, 260 families	Body fat (%), maternal	LOD = 2.2	(1224)

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
D3S3038	3p24.3	893 sibpairs	BMI, paternal effect (in whites)	$p = 0.0065$, LOD = 1.77	(1209)
D3S2432	3p22.3	377 pairs	Body fat (%) (in Pima Indians)	LOD = 2	(1243)
D3S1768	3p22.2	580 families	BMI	LOD = 3.4	(1244)
Chr3p-region	3p14	1848 subjects, 279 pedigrees	BMI	LOD = 1.9	(1245)
			BMI and systolic blood pressure	LOD = 2.13	
			BMI and diastolic blood pressure	LOD = 2.36	
D3S2406	3p12.2	3383 subjects, 1124 families	BMI (men)	LOD = 2	(1227)
D3S3045	3q12.3	1297 subjects, 260 families	BMI, paternal	LOD = 3.66	(1224)
		1297 subjects, 260 families	BMI ≥ 30	NPL = 1.88	(1246)
		1297 subjects, 260 families	BMI ≥ 30	NPL = 1.88	(1226)
Chr3q-region	3q13.3	1848 subjects, 279 pedigrees	BMI and systolic blood pressure and diastolic blood pressure (trivariate)	LOD = 2.59	(1245)
ATA28H11	3q13.33	3383 subjects, 1124 families	BMI (men)	LOD = 2.3	(1227)
			Body fat (%) (men)	LOD = 2.6	
			BMI (men and women)	LOD = 2.8	
D3S1764	3q22.1	596 subjects, 158 families	Factor central obesity	MLS = 2.61	(1247)
		1055 pairs	BMI	LOD = 3.4	(1241)
D3S1744	3q23	1778 sibships	BMI	$p = 0.0009$	(1232)
D3S3053	3q26	1778 sibships	BMI	$p = 0.0015$	(1232)
D3S2427	3q26.33	2209 subjects, 507 families	BMI	LOD = 3.3	(1248)
			Waist circumference	LOD = 2.4	
		3383 subjects, 1124 families	BMI (men)	LOD = 1.7	(1227)
		545 subjects, 128 families	BMI (in African Americans)	LOD = 4.3	(1249)
		1055 pairs	BMI	LOD = 3.4	(1241)
		618 subjects, 202 families	BMI	LOD = 1.8	(1250)
D3S3676	3q26.33	545 subjects, 128 families	BMI (in African Americans)	LOD = 4.3	(1249)
D3S1262	3q27.3	3383 subjects, 1124 families	Body fat (%) (men)	LOD = 2.4	(1227)
D3S1311	3q29	215 subjects, 105 families	Abdominal subcutaneous fat	LOD = 2.5	(1237)
D4S912	4p16.1	430 subjects, 27 sibpairs, 27 pedigrees	BMI	LOD = 4.5	(1238)
D4S2639	4p15.32	994 subjects, 37 pedigrees	BMI	LOD = 2.2	(1207)
D4S2289	4p15.31	994 subjects, 37 pedigrees	BMI	LOD = 2.6	(1207)
D4S2397	4p15.2	521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 2.4	(1219)
D4S3350	4p15.1	994 subjects, 37 pedigrees	BMI	LOD = 9.2	(1207)
D4S2632	4p15.1	994 subjects, 37 pedigrees	BMI	MLS = 6.1	(1207)
D4S1627	4p13	994 subjects, 37 pedigrees	BMI	LOD = 3.4	(1207)
D4S3019	4q12	994 subjects, 37 pedigrees	BMI	LOD = 2.1	(1207)
D4S1592	4q12	1249 sibpairs, >10,000 relative pairs	BMI	LOD = 2.29	(1205)
D4S3248	4q13.1	994 subjects, 37 pedigrees	BMI	LOD = 2	(1207)
D4S1647	4q24	59 pedigrees, 277 sibships	BMI	LOD = 2.63	(1251)
D4S1647-D4S2623	4q24-4q25	321 sibpairs	Body fat (%)	LOD = 2.39	(1236)
D4S1644	4q28.3	1297 subjects, 260 families	BMI	LOD = 1.71	(1226)
		1297 subjects, 260 families	BMI	LOD = 1.71	(1246)
D4S2417	4q31.1	893 sibpairs	BMI, paternal effect (in whites)	$p = 0.005$, LOD = 1.84	(1209)
		521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 1.8	(1219)
GYPA	4q31.1	160 pairs	Skinfolds, trunk-to-extremity ratio	$p = 0.02$	(1252)
D4S1629	4q32.1	893 sibpairs	BMI, maternal effect (in whites)	$p = 0.005$, LOD = 1.89	(1209)
D4S406	4q34.1	447 subjects, 109 pedigrees	BMI > 35	LOD = 2.55	(1253)
D4S2431	4q34.1	215 subjects, 105 families	Abdominal subcutaneous fat	LOD = 2.3	(1237)
D5S817	5p15.2	1100 subjects, 170 families	Adiponectin (in Northern Europeans)	LOD = 4.1	(1229)
		618 subjects, 202 families	BMI	LOD = 1.9	(1250)
D5S426	5p13.3	264 pairs	Leptin	LOD = 2.9	(1230)
D5S2489	5p13.2	1526 pairs	BMI (in Pima Indians)	LOD = 1.7	(1254)
ISL1	5q11.2	226 pairs	Obesity	$p = 0.03$	(1255)
			Leptin	$p = 0.0004$	
			BMI	$p = 0.0004$	

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
D5S407	5q11.2	1249 sibpairs, >10,000 relative pairs	Fat-free mass	LOD = 1.59	(1205)
D5S2500	5q12.1	1526 pairs	BMI (in Pima Indians)	LOD = 1.7	(1254)
D5S1725	5q14.3	321 sibpairs	Fat mass	LOD = 2.25	(1236)
			Body fat (%)	LOD = 2.56	
D5S1463	5q14.3	447 subjects, 109 pedigrees	BMI > 27	LOD = 2.68	(1253)
D5S1453	5q21.3	342 families	Trends in BMI from childhood to adulthood	LOD = 2	(1215)
D5S1505	5q23.1	342 families	Long-term burden in BMI	LOD = 2.2	(1215)
D5S658	5q31.3	453 subjects, 99 families	Abdominal subcutaneous fat	LOD = 2.06	(1237)
			Abdominal total fat	LOD = 1.84	
NR3C1	5q31	88 pairs	BMI	$p = 0.009$	(1256)
D5S1480	5q32	453 subjects, 99 families	Abdominal total fat	LOD = 2.1	(1237)
D5S820-D5S1456	5q33.2-5q35.1	729 subjects, 275 families	Abdominal subcutaneous fat	MLS = 2.64	(1210)
D5S1471	5q35.1	893 sibpairs	BMI (in whites)	$p = 0.0006$, LOD = 2.48	(1209)
D5S211	5q35.2	3027 subjects, 401 families, 317 sibships	BMI (National Heart, Lung, and Blood Institute Family Heart Study)	LOD = 1.8	(1220)
		2072 subjects, 407 families	Factor central obesity	MLS = 1.87	(1247)
D5S408	5q35.3	157 subjects, 7 families	BMI (in whites)	$p = 0.0039$	(1211)
SE30	6p25.1	803 subjects, 192 families	BMI	LOD = 2.13	(1257)
		596 subjects, 158 families	Factor central obesity	MLS = 2.07	(1247)
D6S2434	6p23	596 subjects, 158 families	Factor central obesity	MLS = 1.94	(1247)
D6S260	6p23	508 subjects, 21 families	Obesity under anti-psychotics	LOD = 1.72	(1258)
D6S1959	6p22.3-p22.2	618 subjects, 202 families	Body fat (%)	LOD = 2.7	(1250)
D6S276	6p22.1	624 subjects, 28 families	Eating behavior, restraint (in Old Order Amish)	LOD = 2.3	(1242)
BF	6p21.31	>168 pairs	Skinfolds, subscapular	$p = 0.01$	(1208)
			Skinfolds, triceps	$p = 0.01$	
			Skinfolds, suprailiac	$p = 0.01$	
GLO1	6p21.3-p21.1	>168 pairs	Body weight	$p = 0.004$	(1259)
			Skinfolds, suprailiac	$p = 0.004$	
TNF	6p21.3	>255 pairs, 304 sibpairs	Body fat (%) (in Pima Indians)	$p = 0.002$	(1072)
D6S271	6p21.1	1199 pairs	Leptin	LOD = 2.1	(1260)
D6S462	6q22.31	447 subjects, 109 pedigrees	BMI > 35	LOD = 2.49	(1253)
D6S462-D6S441	6q22.31-6q23.2	506 subjects, 115 pedigrees	BMI > 97th percentile	LOD = 3.27	(1239)
			BMI > 95th percentile	LOD = 3.13	
D6S1009	6q23.3	2086 subjects, 330 pedigrees	Waist circumference	LOD = 3.3	(1223)
		330 pedigrees, 1702 sibships	BMI	LOD = 2.79	(1261)
D6S403	6q23.3	261 subjects, 27 pedigrees	(BMI, leptin, fasting specific insulin) (in Mexican Americans)	LOD = 4.2	(1262)
D6S1003	6q24.1	261 subjects, 27 pedigrees	(BMI, leptin, fasting specific insulin) (in Mexican Americans)	LOD = 4.2	(1262)
D6S264	6q27	261 subjects, 27 pedigrees	(Systolic blood pressure, diastolic blood pressure) (in Mexican Americans)	LOD = 4.9	(1262)
D6S281	6q27	1249 sibpairs, >10,000 relative pairs	BMI	LOD = 1.77	(1205)
			Fat mass	LOD = 2.02	
D7S2477	7p22.3	349 subjects, 66 pedigrees	BMI (in whites)	LOD = 2.53	(1233)
D7S1819	7p22.2	349 subjects, 66 pedigrees	BMI (in whites)	LOD = 2.53	(1233)
D7S2557	7p21.2	342 families	Long-term burden in BMI	LOD = 2.9	(1215)
D7S3051	7p21.1	1055 pairs	BMI	LOD = 2.7	(1241)
D7S1802	7p15.3	803 subjects, 192 families	BMI	LOD = 2.4	(1257)
NPY	7p15.1	302 subjects, 57 families, 545 sibpairs	Obesity (in Mexican Americans)	$p = 0.042$	(1216)
			Body weight (in Mexican Americans)	$p = 0.02$	
			Abdominal circumference (in Mexican Americans)	$p = 0.031$	
			Hip circumference (in Mexican Americans)	$p = 0.012$	
			Diastolic blood pressure (in Mexican Americans)	$p = 0.005$	
			Body mass, body size (in Mexican Americans)	$p = 0.048$	

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
D7S1808	7p15.1	336 sibpairs, 609 relative pairs	Fat-free mass	LOD = 2.72	(1263)
D7S817	7p14.3	769 subjects, 182 families	BMI (in Africans)	LOD = 3.83	(1218)
D7S484	7p14.2	342 families	Long-term burden in BMI	LOD = 2.4	(1215)
D7S1818	7p12.3	342 families	Trends in BMI from childhood to adulthood	LOD = 2.2	(1215)
D7S506-D7S653	7p11.2-7q11.22	430 subjects, 27 sibpairs, 27 pedigrees	BMI	LOD = 1.9	(1238)
D7S3046	7q11.22	514 subjects, 99 families, 347 sibships	Protein intake (%)	$p = 0.0012$	(1217)
D7S653	7q11.22	440 subjects, 27 families	Bivariate BMI: high-density lipoprotein	MLS = 3.86	(1203)
			Bivariate BMI: triglycerides	MLS = 4.21	
			Bivariate waist circumference: high-density lipoprotein	MLS = 3.47	
			Bivariate waist circumference: triglycerides	MLS = 3.74	
			Bivariate BMI: insulin	MLS = 2.44	
			Bivariate waist circumference: insulin	MLS = 1.86	
			Bivariate BMI: waist circumference	MLS = 2.98	
D7S653-D7S479	7q11.22-7q22.1	440 subjects, 27 families	BMI	MLS = 2.4	(1203)
			Waist circumference	MLS = 2	
D7S821	7q21.3	1297 subjects, 260 families	BMI ≥ 35	NPL = 1.93	(1226)
D7S479	7q22.1	261 subjects, 27 pedigrees	High-density lipoprotein, in triglycerides (in Mexican Americans)	LOD = 3.2	(1262)
D7S1799	7q22.1	1297 subjects, 260 families	BMI ≥ 35	NPL = 2.25	(1226)
		1297 subjects, 260 families	BMI > 27	NPL = 2.52	(1246)
			BMI > 30	NPL = 2.04	
			BMI > 35	NPL = 2.25	
D7S692	7q22.3	1020 subjects, 200 families	BMI (in whites)	$p = 0.0002$, LOD = 2.75	(1264)
D7S523	7q31.1	1020 subjects, 200 families	BMI (in whites)	$p = 0.0009$, LOD = 2.11	(1264)
D7S471	7q31.1	261 subjects, 27 pedigrees	High-density lipoprotein, in triglycerides (in Mexican Americans)	LOD = 3.2	(1262)
LEP	7q31.3	302 subjects, 57 families, 545 sibpairs	Waist-to-hip ratio (in Mexican Americans)	$p = 0.01$	(1216)
			Cholesterol, total (in Mexican Americans)	$p = 0.03$	
			Cholesterol, high-density lipoprotein (in Mexican Americans)	$p = 0.026$	
		47 pairs, 47 healthy female/female dizygotic twins	body fat	$p = 0.008$	(1265)
D7S2847	7q31.31	1055 pairs	BMI	LOD = 2.4	(1241)
D7S680	7q32.2	60 pairs	BMI	$p = 0.002$	(1266)
D7S514	7q32.2	60 pairs	BMI	$p = 0.002$	(1266)
		545 pairs	BMI (in Mexican Americans)	$p = 0.0001$	(1267)
			Skinfolds, extremity (in Mexican Americans)	$p = 0.0001$	
			Waist circumference (in Mexican Americans)	$p = 0.0001$	
			Fat mass (in Mexican Americans)	$p = 0.0001$	
D7S504	7q32.2	46 pairs, 103 affected sibpairs	BMI (in African Americans)	$p = 0.001$	(1268)
		78 families, 59 pairs	BMI	$p = 0.04$	(1269)
D7S1875	7q32.2	302 subjects, 57 families, 545 sibpairs	Waist-to-hip ratio (in Mexican Americans)	$p = 0.009$	(1216)
		521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 2	(1219)
		88 trios (index probands and both parents)	BMI (in German children and adolescents)	$p = 0.04$	(1270)
D7S530	7q32.3	60 pairs	BMI	$p = 0.002$	(1266)
D7S1804	7q32.3	3027 subjects, 401 families, 317 sibships	BMI (National Heart, Lung, and Blood Institute Family Heart Study)	MLS = 4.9, $p < 0.00001$	(1220)
D7S640	7q33	672 subjects, 28 pedigrees	Leptin (in Old Order Amish)	LOD = 1.9 adjusted for BMI	(1271)
D7S495	7q34	545 pairs	BMI (in Mexican Americans)	$p = 0.0001$	(1267)
			Skinfolds, extremity (in Mexican Americans)	$p = 0.0001$	

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
			Fat mass (in Mexican Americans)	$p = 0.0001$	
			Waist circumference (in Mexican Americans)	$p = 0.0001$	
D7S1824	7q34	157 subjects, 7 families	BMI (in whites)	$p = 0.0008$	(1211)
KEL	7q35	160 pairs	BMI	$p = 0.0001$	(1252)
			Skinfolds, sum of six	$p = 0.0001$	
D7S2195	7q35	157 subjects, 7 families	BMI (in whites)	$p = 0.001$	(1211)
D7S3068	7q35	157 subjects, 7 families	BMI (in whites)	$p = 0.004$	(1211)
D7S636	7q36.1	672 subjects, 28 pedigrees	Leptin (in Old Order Amish)	LOD = 1.9 adjusted for BMI	(1271)
D7S3070	7q36.1	3383 subjects, 1124 families	Body fat (%) (men)	LOD = 1.8	(1227)
		215 subjects, 105 families	Abdominal total fat training response	LOD = 2.5	(1237)
Chromosome 8 region	8pter-p23.3	2814 subjects, 505 families	BMI	$p = 4.6 \times 10^{-5}$	(1204)
D8S264	8p23.3	2072 subjects, 407 families	Factor central obesity	MLS = 1.92	(1247)
D8S277	8p23.1	893 sibpairs	BMI, paternal effect (in whites)	$p = 0.003$, LOD = 1.98	(1209)
GATA151F02	8p22	769 subjects, 182 families	BMI (in Africans)	LOD = 2.34	(1218)
D8S549	8p22	1249 sibpairs, >10,000 relative pairs	Fat mass	LOD = 1.95	(1205)
D8S282	8p21.3	994 subjects, 37 pedigrees	BMI	LOD = 2	(1207)
D8S1121	8p11.23	470 subjects, 10 families	BMI	$p = 0.0001$, MLS = 3.21	(1272)
D8S1110	8q11.22	5000 sibpairs	Leptin	LOD = 2.2	(1231)
D8S1110-D8S1113	8q11.22-8q12.1	729 subjects, 275 families	Abdominal subcutaneous fat	MLS = 2.24	(1210)
D8S1113	8q12.1	893 sibpairs	BMI (in whites)	$p = 0.0013$, LOD = 2.05	(1209)
D8S2324	8q13.3	1297 subjects, 260 families	BMI ≥ 35	NPL = 1.9	(1226)
		1297 subjects, 260 families	BMI ≥ 35	NPL = 1.9	(1246)
GATA8B01	8q21.3	59 pedigrees, 277 sibships	BMI	LOD = 2.56	(1251)
D8S1136	8q22.3	508 subjects, 21 families	Obesity	MLS = 1.93	(1258)
D8S556	8q23.1	522 subjects, 99 families, 364 sibpairs	BMI (in whites)	LOD = 2	(1273)
D8S1132	8q23.1	157 subjects, 7 families	BMI (in whites)	$p = 0.005$	(1211)
D8S1179	8q24.11	729 subjects, 275 families	Waist-to-hip ratio	MLS = 2.06	(1210)
D9S910	9q22.33	1297 subjects, 260 families	BMI ≥ 30 , maternal	LOD = 2.28	(1224)
		1297 subjects, 260 families	BMI > 30	NPL = 2.09	(1246)
		1297 subjects, 260 families	BMI ≥ 30	NPL = 2.09	(1226)
D9S1122	9q21-q22	521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 2.4	(1219)
D9S257	9q22.1	521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 2.4	(1219)
D9S299-D9S930	9q31-9q31	430 subjects, 27 sibpairs, 27 pedigrees	BMI	LOD = 2.1	(1238)
ORM1	9q33.1	>168 pairs	Skinfolds, suprailiac	$p = 0.03$	(1208)
D9S282	9q34	508 subjects, 21 families	Obesity under anti-psychotics	LOD = 1.71	(1258)
AK1	9q34.13	>168 pairs	Skinfolds, suprailiac	$p = 0.01$	(1208)
D9S158	9q34.3	522 subjects, 99 families, 364 sibpairs	BMI (in whites)	LOD = 2.3	(1273)
D10S1435	10p15.3	522 subjects, 99 families, 364 sibpairs	BMI (in whites)	LOD = 2.7	(1273)
		1526 pairs	Fat mass (in whites)	LOD = 2.7	
			BMI (in Pima Indians)	LOD = 1.7	(1254)
D10S189	10p15.1	522 subjects, 99 families, 364 sibpairs	BMI (in whites)	MLS = 2.7, SEGPAT	(1273)
		1526 pairs	Fat mass (in whites)	MLS = 1	
			BMI (in Pima Indians)	LOD = 1.7	(1254)
D10S1423	10p12.33	893 sibpairs	BMI, paternal effect (in whites)	$p = 0.005$, LOD = 1.89	(1209)
D10S582	10p12.31	667 subjects, 244 families	Obesity (in whites and African Americans)	NPL = 2.68	(1274)
		862 subjects, 170 families	Obesity (in African Americans, in European Americans)	$p = 0.0005$	(1275)

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
D10S197	10p12.2	1297 subjects, 260 families 264 pairs	BMI \geq 27, maternal Obesity	LOD = 2.71 LOD = 4.9	(1224) (1230)
D10S204	10p12.1	369 subjects, 89 families	Obesity (in white children and adolescents)	LOD = 2.24	(1276)
D10S193	10p12.1	386 subjects, 93 families	Obesity	LOD = 2.5	(1277)
D10S208	10p11.23	667 subjects, 244 families 862 subjects, 170 families	Obesity (in whites and African Americans) Obesity (in African Americans, in European Americans)	NPL = 2.68 $p = 0.0005$	(1274) (1275)
D10S1781	10p11.2	386 subjects, 93 families	Obesity	LOD = 2.5	(1277)
SHGC-31480	10p11.23	386 subjects, 93 families	Obesity	LOD = 2.5	(1277)
D10S220	10q21.1	672 subjects, 28 pedigrees	Leptin (in Old Order Amish)	LOD = 2.7	(1271)
D10S107	10q21.1	862 subjects, 170 families	Obesity (in African Americans, in European Americans)	$p = 0.0005$	(1275)
D10S1646	10q22.1	667 subjects, 244 families	Waist circumference (in whites and African Americans)	LOD = 2.5	(1274)
D10S535	10q22.3	667 subjects, 244 families	BMI (in whites and African Americans) Waist circumference (in whites and African Americans)	NPL = 2.24 LOD = 2.5	(1274)
D10S1267	10q24.32	447 subjects, 109 pedigrees	BMI (in whites and African Americans) BMI $>$ 27	NPL = 2.24 LOD = 2.47	(1253)
D10S1679	10q26.13	667 subjects, 244 families	Waist-to-hip ratio (in whites and African Americans)	NPL = 2.22	(1274)
D10S1656	10q26.2	667 subjects, 244 families	Obesity (in whites and African Americans) Waist-to-hip ratio (in whites and African Americans)	NPL = 2.25 NPL = 2.22	(1274)
Chr10q-region	10q26.3	1848 subjects, 279 pedigrees	Obesity (in whites and African Americans) BMI	NPL = 2.25 LOD = 1.98	(1245)
D10S212	10q26.3	59 pedigrees, 277 sibships	BMI and systolic blood pressure	LOD = 2.55	(1213)
D11S984-D11S988	11p15.5- 11p15.5	198 subjects, 18 pedigrees 430 subjects, 27 sibpairs, 27 pedigrees	BMI and diastolic blood pressure BMI	LOD = 3.2 LOD = 4.09	(1238)
CCKBR	11p15.4	226 pairs	BMI and systolic blood pressure and diastolic blood pressure	LOD = 2.08	(1251)
C11P15_3	11p15.2	215 subjects, 105 families	BMI	LOD = 3.3	(1213)
D11S419	11p15.2	67 pairs	BMI	LOD = 2.5	(1238)
ATA34E08	11p13	3383 subjects, 1124 families	Leptin	$p = 0.01$	(1255)
D11S1993	11q12	215 subjects, 105 families	Abdominal subcutaneous fat	LOD = 1.85	(1237)
D11S1313	11q12.1	1297 subjects, 260 families	BMI (in French whites)	$p = 0.003$	(708)
D11S2006-D11S2371	11q12.13- 11q13.3	729 subjects, 275 families	BMI (Women)	LOD = 1.8	(1227)
D11S916	11q13.3	640 subjects, 240 relative pairs, 155 pedigrees	Abdominal subcutaneous fat	LOD = 1.75	(1237)
D11S2371	11q13.3	1297 subjects, 260 families	Body fat (%), paternal	LOD = 2.21	(1224)
D11S1321	11q13.3	640 subjects, 240 relative pairs, 155 pedigrees	Obesity (in white children and adolescents)	LOD = 1.65	(1276)
D11S911	11q13.4	640 subjects, 240 relative pairs, 155 pedigrees	Abdominal visceral fat	MLS = 2.36	(1210)
D11S2002	11q13.3	1510 subjects, 509 families	Resting metabolic rate	$p = 0.006$	(1278)
D11S940-D11S2000	11q22-11q22.3	562 subjects, 178 families	Body fat (%), paternal	LOD = 2	(1224)
D11S2000	11q22.3	769 subjects, 182 families	Resting metabolic rate	$p = 0.02$	(1278)
D11S2366	11q23.1	277 siblings	Body fat (%)	$p = 0.04$	(1211)
D11S1998	11q23.3	1526 pairs	Fat mass	$p = 0.02$	(1243)
			Resting metabolic rate	$p = 2^{-06}$	(1278)
			Factor central obesity	MLS = 2.19	(1247)
			BMI	LOD = 2.5	(1279)
			BMI (in Africans)	LOD = 3.35	(1218)
			Body fat (%) (in Pima Indians)	$p = 0.0028$	(1243)
			BMI (in whites)	$p = 0.0079$	(1211)
			Body fat (%) (in Pima Indians)	$p = 0.0009$	(1243)
			BMI (in Pima Indians)	LOD = 2.7	(1254)

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
D11S976	11q23.3	236 pairs	Energy expenditure, 24-hour (in Pima Indians)	LOD = 2	(1214)
D11S4464	11q24.1	430 subjects, 27 sibpairs, 27 pedigrees	BMI	LOD = 2.3	(1238)
		1526 pairs	BMI (in Pima Indians)	LOD = 2.7	(1254)
		1778 sibships	BMI	$p = 0.0023$	(1232)
		994 subjects, 37 pedigrees	BMI	LOD = 2.8	(1207)
D11S934	11q24.2	994 subjects, 37 pedigrees	BMI	LOD = 2.6	(1207)
D11S912	11q24.3	264 families, 1766 pairs, 966 siblings	BMI	LOD = 3.6	(1280)
		1778 sibships	BMI	$p = 0.0003$	(1232)
		994 subjects, 37 pedigrees	BMI	LOD = 2.7	(1207)
D11S2359	11q25	1778 sibships	BMI	$p = 0.0012$	(1232)
GATA49D12N (D3S2395)	12p13.31	1297 subjects, 260 families	BMI ≥ 27	NPL = 2.12	(1226)
		893 sibpairs	BMI, paternal effect (in whites)	$p = 0.006$, LOD = 1.83	(1209)
		1297 subjects, 260 families	BMI ≥ 27	NPL = 2.12	(1246)
D12S391	12p13.2	342 families	Trends in BMI from childhood to adulthood	LOD = 2.9	(1215)
D12S1042	12p12.1	522 subjects, 99 families, 364 sibpairs	BMI (in whites)	MLS = 2.1	(1273)
			Fat mass (in whites)	MLS = 1.2	
D12S297-D12S1294	12q13.13-12q15	729 subjects, 275 families	Waist-to-hip ratio	MLS = 2.67	(1210)
D12S83	12q13.3	1249 sibpairs, >10,000 relative pairs	Fat-free mass	LOD = 1.79	(1205)
D12S1691	12q14.1	514 subjects, 99 families, 347 sibships	Fat intake	$p = 0.0013$	(1217)
D12S1052	12q21	729 subjects, 275 families	Waist-to-hip ratio	MLS = 2.6	(1210)
		349 subjects, 66 pedigrees	BMI (in whites)	LOD = 3.41	(1233)
D12S1052-D12S1064	12q21-12q21.33	729 subjects, 275 families	Waist-to-hip ratio	MLS = 2.91	(1210)
D12S1064	12q21.33	342 families	Trends in BMI from childhood to adulthood	LOD = 2.1	(1215)
		349 subjects, 66 pedigrees	BMI (in whites)	LOD = 3.41	(1233)
PAH	12q22-q24.2	1297 subjects, 260 families	BMI > 30	NPL = 1.92	(1246)
PAH-D12S2070	12q22-q24.2-12q24.21	729 subjects, 275 families	Waist-to-hip ratio	MLS = 2.48	(1210)
D12PAH	12q23.1	342 families	Trends in BMI from childhood to adulthood	LOD = 2.3	(1215)
			Long-term burden in BMI	LOD = 3	
		1297 subjects, 260 families	BMI ≥ 30	NPL = 1.92	(1226)
D12S79-D12S1366	12q23.1-12q24.23	508 subjects, 21 families	Obesity under anti-psychotics	MLS = 2.74	(1258)
IGF1	12q23.3	521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 1.9	(1219)
		502 subjects, 99 families, 352 pairs, 190 parents, 312 offspring	Abdominal visceral fat (in whites)	$p = 0.02$	(895)
			Fat-free mass (in whites)	$p = 0.0002$	
D12S1339	12q24.2	1297 subjects, 260 families	Body fat (%)	LOD = 4.08	(1246)
D12S2070	12q24.21	514 subjects, 99 families, 347 sibships	Fat intake (%)	$p = 0.002$	(1217)
		1297 subjects, 260 families	BMI, maternal	MLS = 4.01	(1224)
			Waist circumference, maternal	MLS = 3.69	
		1297 subjects, 260 families	Body fat (%)	LOD = 3.79	(1226)
		1297 subjects, 260 families	BMI	LOD = 3.57	(1246)
			Waist circumference	LOD = 3.05	
D12S395-D12S2078	12q24-12q24.32	3383 subjects, 1124 families	Body fat (%) (women)	LOD = 3.8	(1227)
D12S2078	12q24.32	521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 2.9	(1219)
D12S2078-D12S1045	12q24.32-12q24.33	3383 subjects, 1124 families	Body fat (%) (men and women)	LOD = 3.3	(1227)
D12S1045	12q24.33	521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 2.9	(1219)
D12S1638	12q24.33	59 pedigrees, 277 sibships	BMI	LOD = 1.94	(1251)
D13S175	13q12.11	580 families	BMI	LOD = 3.3	(1244)

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
D13S221	13q12.13	580 families	BMI	LOD = 3.3	(1244)
D13S1493	13q13.2	1297 subjects, 260 families	BMI \geq 40	NPL = 2.03	(1226)
		3383 subjects, 1124 families	BMI and blood pressure response to postural change	LOD = 3.2	(1281)
D13S894	13q13.3	1297 subjects, 260 families	BMI \geq 27, maternal	LOD = 2.34	(1224)
		1297 subjects, 260 families	BMI \geq 40	NPL = 2.63	(1226)
		1297 subjects, 260 families	BMI \geq 27	NPL = 1.88	(1246)
ESD	13q14.11	160 pairs	Body fat (%)	$p = 0.04$	(1252)
			Skinfolds, sum of six	$p = 0.04$	
D13S257	13q14.2	3027 subjects, 401 families, 317 sibships	BMI (National Heart, Lung, and Blood Institute Family Heart Study)	MLS = 3.2 $p = 0.00006$	(1220)
D13S1807	13q21.1	1297 subjects, 260 families	BMI	LOD = 2.67	(1226)
D13S800	13q21.32	342 families	Trends in BMI from childhood to adulthood	LOD = 2	(1215)
		1297 subjects, 260 families	BMI	LOD = 2.7	(1226)
		1297 subjects, 260 families	BMI	LOD = 2.7	(1246)
D13S793	13q31.3	1297 subjects, 260 families	BMI, paternal	LOD = 4.79	(1224)
			Waist circumference, paternal	LOD = 3.11	
		1297 subjects, 260 families	BMI	LOD = 2.78	(1226)
D13S779	13q32.2	1312 subjects, 696 families	Factor central obesity	MLS = 2.17	(1247)
		1297 subjects, 260 families	BMI	LOD = 2.82	(1226)
		1312 subjects, 696 families	Factor central obesity	MLS = 2.67	(1247)
		1297 subjects, 260 families	BMI	LOD = 2.82	(1246)
			Waist circumference	LOD = 1.8	
D13S285	13q34	330 pedigrees, 1702 sibships	Obesity before age 35	$p = 0.001$	(1282)
		521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 1.9	(1219)
D14S742	14q11.2	522 subjects, 99 families, 364 sibpairs	Fat mass (in whites)	MLS = 1.7	(1273)
		893 sibpairs	BMI (in whites)	MLS = 2.2	
			BMI (in whites)	$p = 0.002$, LOD = 1.95	(1209)
D14S283	14q11.2	522 subjects, 99 families, 364 sibpairs	Fat mass (in whites)	$p = 0.0006$	(1273)
			Leptin (in whites)	$p = 0.003$	
			Fat mass (in whites)	MLS = 2	
			BMI (in whites)	MLS = 1.8	
D14S1280	14q11.2	522 subjects, 99 families, 364 sibpairs	Fat-free mass (in whites)	MLS = 1.1	(1273)
			BMI (in whites)	MLS = 2.4	
D14S608	14q12	1100 subjects, 170 families	Adiponectin (in Northern Europeans)	LOD = 3.2	(1229)
D14S599	14q13.1	1100 subjects, 170 families	Adiponectin (in Northern Europeans)	LOD = 3.2	(1229)
D14S276	14q22.2	672 subjects, 28 pedigrees	Waist circumference (in Old Order Amish)	LOD = 1.8	(1271)
D14S588	14q24.1	215 subjects, 105 families	Abdominal subcutaneous fat	LOD = 2.4	(1237)
D14S74	14q24.3	672 subjects, 28 pedigrees	Leptin (in Old Order Amish)	LOD = 2.5	(1271)
D14S280	14q32.12	672 subjects, 28 pedigrees	Leptin (in Old Order Amish)	LOD = 2.5	(1271)
D14S617	14q32.12	1055 pairs	BMI	LOD = 2.2	(1241)
D15S128-D15S513	15q12-15q15.1	506 subjects, 115 pedigrees	Age adiposity rebound	LOD = 2.53	(1239)
D15S1232	15q13.3	3027 subjects, 401 families, 317 sibships	BMI (National Heart, Lung, and Blood Institute Family Heart Study)	LOD = 1.7	(1220)
D15S641	15q15.2	478 subjects, 10 families	asp levels	LOD = 2.1	(1283)
			asp and high-density lipoprotein 2a-cholesterol	LOD = 3.2	
LIPC	15q21-23	660 subjects, 202 families, 315 sibpairs, 274 men, 386 women	Eating behavior, hunger	LOD = 1.76	(978)
D15S206	15q24-q25	660 subjects, 202 families, 315 sibpairs, 274 men, 386 women	Eating behavior, hunger	LOD = 3	(978)
D15S655	15q25.3	3383 subjects, 1124 families	Body fat (%) (men)	LOD = 3	(1227)
D15S652	15q26.1	336 sibpairs, 609 relative pairs	Fat-free mass	LOD = 3.56	(1263)
D15S657	15q26.2	336 sibpairs, 609 relative pairs	Fat-free mass	LOD = 2	(1263)
D16S510	16p13.3	672 subjects, 28 pedigrees	Leptin (in Old Order Amish)	LOD = 1.7	(1271)
			BMI (in Old Order Amish)	LOD = 1.7	
D16S404	16p13.2	893 sibpairs	BMI (in whites)	$p = 0.00025$, LOD = 3.12	(1209)

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
D16S764	16p13.12	893 sibpairs	BMI (in whites)	$p = 0.0006$, LOD = 2.45	(1209)
D16S3253	16q12.2	330 pedigrees, 1702 sibships	BMI	LOD = 3.21	(1261)
D16S415-D16S420	16q12.2- 16q24.1	506 subjects, 115 pedigrees	Age adiposity rebound	LOD = 2.54	(1239)
D16S2620	16q21	1055 pairs	BMI	LOD = 2.6	(1241)
D16S265	16q21	1199 pairs	Leptin	LOD = 2	(1260)
D16S422	16q23.3	995 subjects, 153 families	Resting energy expenditure	LOD = 2.96	(1284)
D17S849-D17S799	17p13-17p13	506 subjects, 115 pedigrees	BMI > 95th percentile	LOD = 2.25	(1239)
D17S1308	17p13.3	729 subjects, 275 families	Abdominal subcutaneous fat	MLS = 2.06	(1210)
D17S1303	17p13.1	478 subjects, 10 families	asp levels	LOD = 2.7	(1283)
D17S947	17p12	1100 subjects, 170 families	Adiponectin (in Northern Europeans)	LOD = 1.7	(1229)
		2209 subjects, 507 families	Leptin	LOD = 5	(1248)
		1055 pairs	BMI	LOD = 2.5	(1241)
D17S1293	17q11.2	470 subjects, 10 families	BMI	$p = 0.001$	(1272)
D17S2180	17q21.32	521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 2.2	(1219)
D17S1306	17q22	660 subjects, 202 families, 315 sibpairs, 274 men, 386 women	Eating behavior, hunger	MLS = 2.06	(978)
D17S1290	17q23.2	660 subjects, 202 families, 315 sibpairs, 274 men, 386 women	Eating behavior, hunger	MLS = 2.45	(978)
		729 subjects, 275 families	BMI	MLS = 2.76	(1210)
		521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 2.2	(1219)
D17S944	17q23.3-q25.1	447 subjects, 109 pedigrees	BMI > 35	LOD = 3.16	(1253)
D17S1351	17q23-q24	660 subjects, 202 families, 315 sibpairs, 274 men, 386 women	Eating behavior, hunger	LOD = 1.75	(978)
D17S1301	17q25.2	521 subjects, 156 families	Abdominal subcutaneous fat	LOD = 2.2	(1219)
D18S481	18p11.3	342 families	Trends in BMI from childhood to adulthood	LOD = 2	(1215)
D18S843-D18S53	18p11.22- 18p11.23	3383 subjects, 1124 families	Body fat (%) (men)	LOD = 1.7	(1227)
MC5R	18p11.21	289 pairs	BMI	$p = 0.001$	(969)
			Skinfolds, sum of six	$p = 0.005$	
			Fat mass	$p = 0.001$	
			Body fat (%)	$p = 0.02$	
			Fat-free mass	$p = 0.008$	
			Resting metabolic rate	$p = 0.002$	
D18S877	18q12.1	336 sibpairs, 609 relative pairs	Fat-free mass	LOD = 3.6	(1263)
		236 pairs	Body fat (%) (in Pima Indians)	LOD = 2.3	(1214)
D18S535	18q12.3	336 sibpairs, 609 relative pairs	Fat-free mass	LOD = 3.58	(1263)
D18S858	18q21.31	3383 subjects, 1124 families	BMI and blood pressure response to postural change	LOD = 2.6	(1281)
D18S1155	18q21.32	367 subjects, 166 families, 193 pairs	Obesity (in Finns)	LOD = 2.4	(1285)
MC4R	18q22	289 pairs	Respiratory quotient	$p = 0.04$	(969)
D19S714	19p13.3	404 subjects	Resistin mRNA levels in adipose tissue (adult baboons)	LOD = 3.84	(1286)
LDLR	19p13.2	522 subjects, 99 families, 364 sibpairs	Skinfolds, sum of eight (in whites)	$p = 0.002$	(1273)
			Leptin (in whites)	$p = 0.0009$	
			Body fat (%) (in whites)	$p = 0.009$	
D19S221-D19S414	19p13-19q13.11	506 subjects, 115 pedigrees	Age adiposity rebound	LOD = 2.13	(1239)
D19S215	19p13	660 subjects, 202 families, 315 sibpairs, 274 men, 386 women	Eating behavior, disinhibition	LOD = 1.8	(978)
D19S418	19q13.3-q13.43	447 subjects, 109 pedigrees	BMI > 35	LOD = 3.21	(1253)
D19S414	19q13.11	369 subjects, 89 families	Obesity (in white children and adolescents)	LOD = 1.97	(1276)
D19S254	19q13.43	330 pedigrees, 1702 sibships	Obesity before age 35	$p = 0.001$	(1282)
D20S482	20p13	893 sibpairs	BMI (in whites)	$p = 0.00016$, LOD = 3.55	(1209)
D20S851	20p12.2	893 sibpairs	BMI (in whites)	$p = 4.6 \times 10^{-5}$, LOD = 4.08	(1209)
		1724 subjects, 1202 families	Factor central obesity	MLS = 1.97	(1247)
D20S604	20p12.1	1724 subjects, 1202 families	Factor central obesity	MLS = 2.46	(1247)

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
D20S601	20q11.22-q11.23	236 pairs	Respiratory quotient, 24-hour (in Pima Indians)	LOD = 3	(1214)
D20S478	20q12	994 subjects, 37 pedigrees	BMI	LOD = 2	(1207)
D20S438	20q12	1711 subjects, 103 pedigrees	BMI (in Utah pedigrees)	LOD = 3.5	(1287)
		994 subjects, 37 pedigrees	BMI	LOD = 2	(1207)
D20S465	20q12	994 subjects, 37 pedigrees	BMI	LOD = 2	(1207)
D20S107	20q12	513 subjects, 92 families, 423 pairs	BMI	LOD = 3.2	(1288)
			Body fat (%)	LOD = 3.2	
D20S476	20q13	513 subjects, 92 families, 423 pairs	BMI	LOD = 3.06	(1288)
ADA	20q13.12	160 pairs	BMI	$p = 0.001$	(1252)
			skinfolts, sum of six	$p = 0.001$	
D20S481	20q13.12	994 subjects, 37 pedigrees	BMI	LOD = 2.2	(1207)
D20S17	20q13.12	650 subjects, 258 pairs, 152 pedigrees	Body fat (%)	$p = 0.0078$	(662)
D20S178	20q13.13	667 subjects, 244 families	Body fat (%) (in whites and African Americans)	NPL = 2.57	(1274)
D20S887	20q13.13	514 subjects, 99 families, 347 sibships	Fat intake	$p = 0.0093$	(1217)
D20S869	20q13.13	514 subjects, 99 families, 347 sibships	Carbohydrate intake	$p = 0.0023$	(1217)
			Fat intake	$p = 0.0005$	
			Protein intake	$p = 9 \times 10^{-5}$	
D20S857	20q13.13	514 subjects, 99 families, 347 sibships	Total energy intake	$p = 7 \times 10^{-5}$	(1217)
			Carbohydrate intake	$p = 0.0008$	
			Fat intake	$p = 0.0006$	
			Protein intake	$p = 0.00022$	
D20S839	20q13.13	514 subjects, 99 families, 347 sibships	Total energy intake	$p = 0.00014$	(1217)
			Carbohydrate intake	$p = 0.0009$	
			Fat intake	$p = 0.0019$	
			Protein intake	$p = 0.00041$	
D20S480	20q13.13	514 subjects, 99 families, 347 sibships	Total energy intake	$p = 0.0003$	(1217)
			Carbohydrate intake	$p = 0.0006$	
			Fat intake	$p = 0.0016$	
			Protein intake	$p = 0.0009$	
D20S211	20q13.2	513 subjects, 92 families, 423 pairs	BMI	LOD = 3.2	(1288)
		513 subjects, 92 families, 423 pairs	Body fat (%)	LOD = 3.2	(1288)
D20S876	20q13.13	514 subjects, 99 families, 347 sibships	Total energy intake	$p = 0.00012$	(1217)
			Carbohydrate intake	$p = 0.001$	
			Protein intake	$p = 0.00085$	
D20S120	20q13.2	650 subjects, 258 pairs, 152 pedigrees	Body fat (%)	$p = 0.004$	(662)
D20S149	20q13.31-qter	667 subjects, 244 families	Body fat (%) (in whites and African Americans)	NPL = 2.57	(1274)
		513 subjects, 92 families, 423 pairs	BMI	LOD = 3.2	(1288)
			Body fat (%)	LOD = 3.2	
D21S1442	21q21.2	3383 subjects, 1124 families	Body fat (%) (women)	LOD = 1.8	(1227)
D21S2052	21q21.3	1510 subjects, 509 families	Factor central obesity	MLS = 2.13	(1247)
D21S1440	21q22.12	1510 Subjects, 509 families	Factor central obesity	MLS = 2.13	(1247)
D21S1446	21q22.3	1297 subjects, 260 families	Body fat (%)	LOD = 4.21	(1226)
		1297 subjects, 260 families	Body fat (%)	LOD = 4.27	(1246)
D22S264	22q11.21	453 subjects, 99 families	Abdominal subcutaneous fat	LOD = 1.96	(1237)
D22S1685 (D20S608)	22q11.21	318 subjects, 10 families	Leptin	$p = 0.001$	(1289)
A4GALT	22q13.31	>168 pairs	Body weight	$p = 0.03$	(1208)
DXS8099	Xp22.13	994 subjects, 37 pedigrees	BMI	LOD = 2.6	(1207)

Table 5. (continued)

Gene/marker	Location	Population	Phenotypes	Score	Reference
DXS997	Xp21.3	1148 subjects, 133 families, 190 European-American families (940 members); 43 African-American families (208 members)	Waist-to hip ratio (in European Americans and African Americans)	LOD = 2.7	(1290)
DXS1003	Xp11.3	1148 subjects, 133 families, 190 European-American families (940 members); 43 African-American families (208 members)	Waist-to-hip ratio (in European Americans and African Americans)	LOD = 2.7	(1290)
DXS1059	Xq23	994 subjects, 37 pedigrees	BMI	LOD = 2	(1207)
DXS6804	Xq23	367 subjects, 166 families, 193 pairs	Obesity (in Finns)	LOD = 3.1	(1285)
DXS1220	Xq24	184 families, 218 sibships	Obesity	MLS = 1.93	(1059)
AGTR25747C/T	Xq24	184 families, 218 sibships	Obesity	MLS = 2.3	(1059)

LOD, logarithm of odds; MLS, maximum LOD score; NPL, non-parametric linkage.

undertaken in the same sample of European American families with the aim of detecting epistatic interactions among QTLs (1226). QTLs influencing BMI were found on chromosomes 2p24.2 and 4q28.3 and over a broad region of chromosome 13q21.1-q32.2, whereas QTLs influencing percentage body fat were found on chromosomes 12q24.21 and 21q22.3. Linkages with different obesity affection status (BMI \geq 27, 30, 35, and 40) were found on chromosomes 3q12.3, 7q21.3, 7q22.1, 8q13.3, 9q22.33, 12p13.31, 12q23.1, 13q13.2, and 13q13.3. Significant evidence of interactions was found between loci on chromosome regions 2p25-p24 and 13q13-q21 (1226).

A search for genes influencing BMI, WHR, and abdominal fat assessed by computed tomography scan was undertaken in 330 subjects from 154 African-American families and in 729 subjects from 275 Hispanic-American families (1210). In the African-American families, significant linkage to BMI was found on chromosomes 1p36 (LOD = 2.14) and 3p26.3 (LOD = 3.67). In the Hispanic-American families, a QTL for BMI was found on chromosome 17q23.2 and QTLs for WHR were found on chromosomes 8q24.11, 12q13.13-q15, 12q21-q21.33, and 12q22-q24.21. QTLs for abdominal fat were found on chromosomes 5q33.2-q35.1, 8q11.22-q12.1, and 17p13.3 for abdominal subcutaneous fat and on chromosome 11q12.13-q13.3 for abdominal visceral fat. The last genome scan study was a genome-wide linkage analysis of four factors related to the metabolic syndrome derived from a factor analysis of 10 risk factors (1247). Factor analysis yielded four different metabolic syndrome factors (obesity-insulin, blood pressure, lipids-insulin, and central obesity) that were tested for linkage with 400 microsatellite markers in four different ethnic groups (blacks, whites, Hispanics, Asians). Only results with the central obesity factor are reported in Table 5. Evidence of linkage

was found on chromosomes 13q31.3, 13q32.2, 20p12.2, and 20p12.1 in blacks, on chromosomes 11q13.3, 21q21.3, and 21q22.12 in whites, and on chromosomes 3q22.1, 5q35.2, 6p25.1, 6p23, and 8p23.3 in Asians. No evidence of linkage was found in Hispanics (1247).

A bivariate linkage analysis of metabolic syndrome phenotypes (BMI, WC, lipids, and insulin) with 19 markers located on chromosome 7q11.22-q22.1 performed in 440 subjects from 27 Mexican-American families revealed evidence of univariate linkage for BMI (LOD = 2.4) and WC (LOD = 2.0) between markers D7S653 and D7S479 and linkages (LOD scores ranging from 1.86 to 4.21) for most of the bivariate traits (BMI-lipids, BMI-insulin, WC-lipids, WC-insulin, BMI-WC) to a 6-cM region near marker D7S653 (1203).

Finally, a meta-analysis of genome scans that used BMI as their primary obesity phenotype and were published before July 2003 was undertaken to identify QTLs influencing obesity (1204). A total of 29 genome scans were identified from the literature; of these studies, 13 analyzed BMI as a quantitative trait. Access to detailed results was requested from the authors of the 13 studies, and information was obtained in only 5 of the 13 studies. The results from these five studies, which included a total of 2814 individuals from 505 families, were jointly analyzed using a variance component approach. For the purpose of the analysis, the genome was divided into 121 30-cM regions called bins in such a way that the first bin on chromosome 1 (1.1) includes the results of markers tested between locations 0 and 30 cM, the second bin (1.2) encompasses the 30- to 60-cM region of chromosome 1, and so on for all chromosomes. For each scan, the bins were then sorted according to the maximum LOD score in that bin, and ranks were assigned with the lowest rank assigned to the bin with the

Table 6. Evolution in the status of the Human Obesity Gene Map

	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005
Single-gene mutations*				2	6	6	6	6	6	6/7	10	11
KO and Tg									38	55	166	244
Mendelian disorders with map location	8	12	13	16	16	20	24	25	33	41	49	50
Animal QTLs	7	9	24	55	67	98	115	165	168	183	221	408
Human QTLs from genome scans				3	8	14	21	33	68	139	204	317
Candidate genes with positive findings	9	10	13	21	29	40	48	58	71	90	113	127

* Number of genes, not number of mutations.

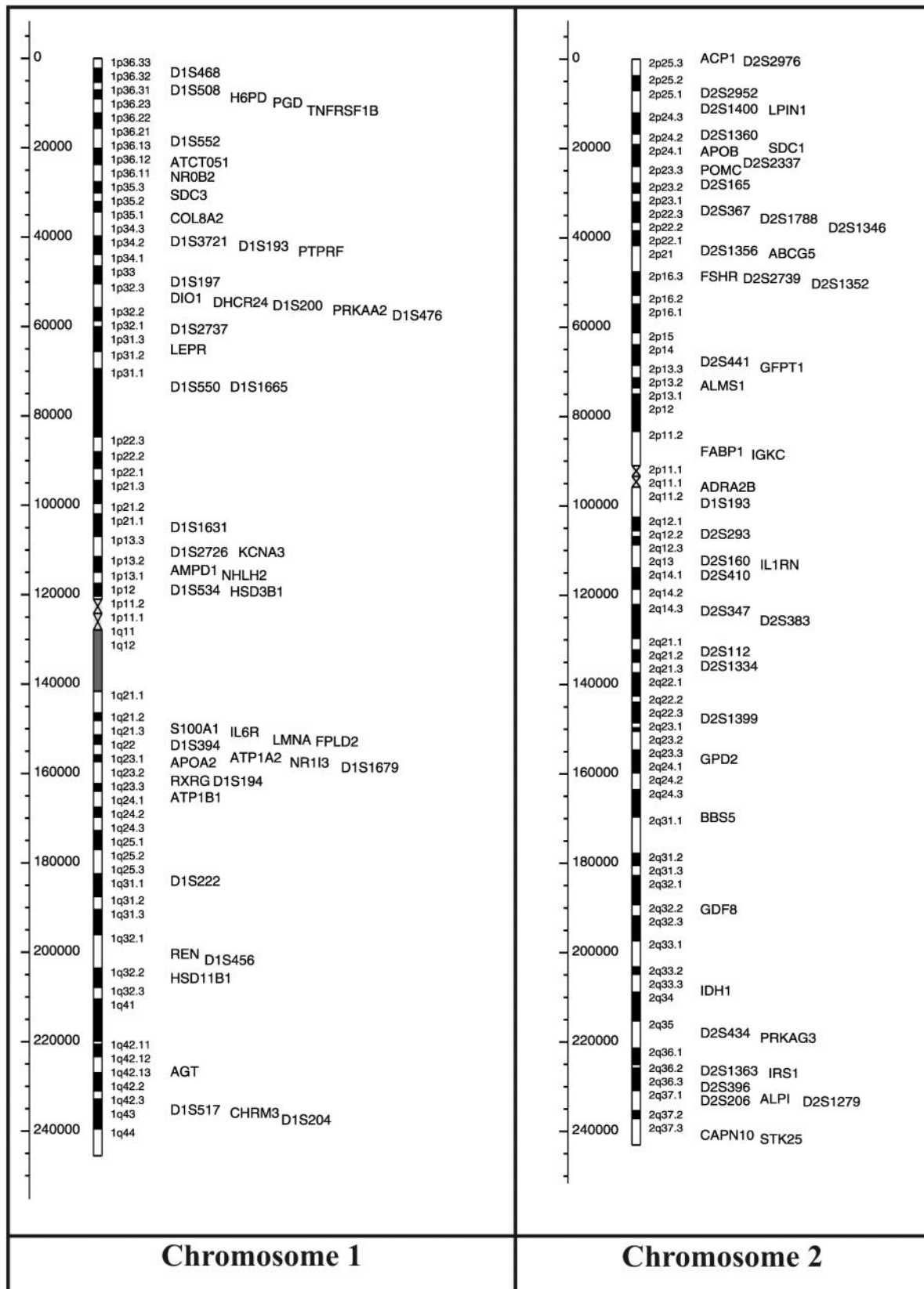
highest LOD score. Within each study, the ranks were weighted according to the number of genotyped individuals in the sample, and the weighted average rank was then calculated for each bin across the five studies. The bin with the lowest weighted average rank for all studies corresponded to the region of the genome showing the most evidence of linkage across all studies retained in the meta-analysis. The results of the analysis revealed that the lowest weighted average rank was found in bin 8.1, suggesting that the best evidence of linkage to BMI across all five studies is found at the location 0 to 30 cM on chromosome 8 (8pter-p23.3). Based on permutation testing, this was the only region showing significant ($p = 0.0005$) evidence of linkage to BMI. Interestingly, only two of the five studies retained in the meta-analysis showed suggestive evidence of linkage to BMI in that region of chromosome 8.

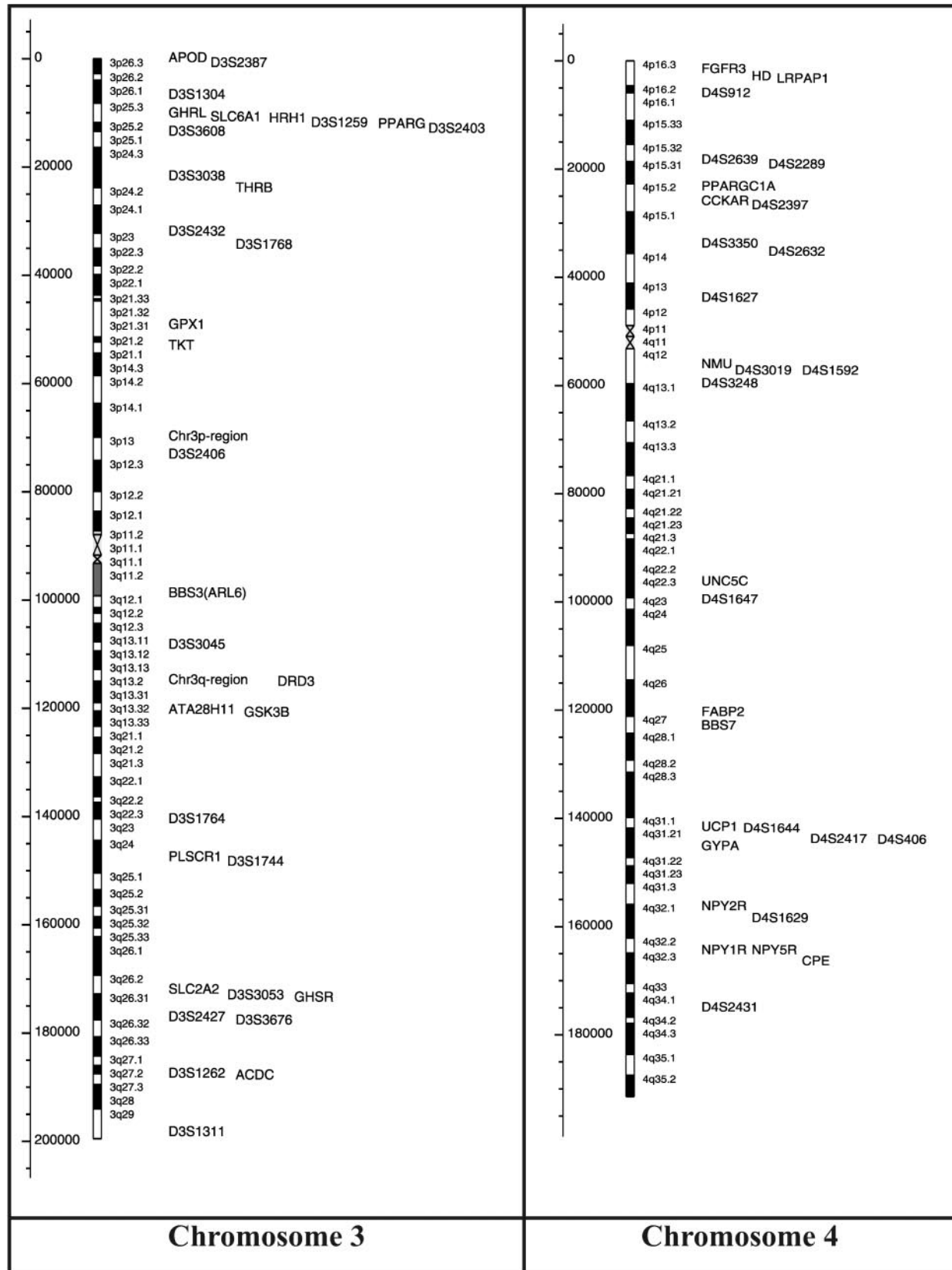
Conclusion

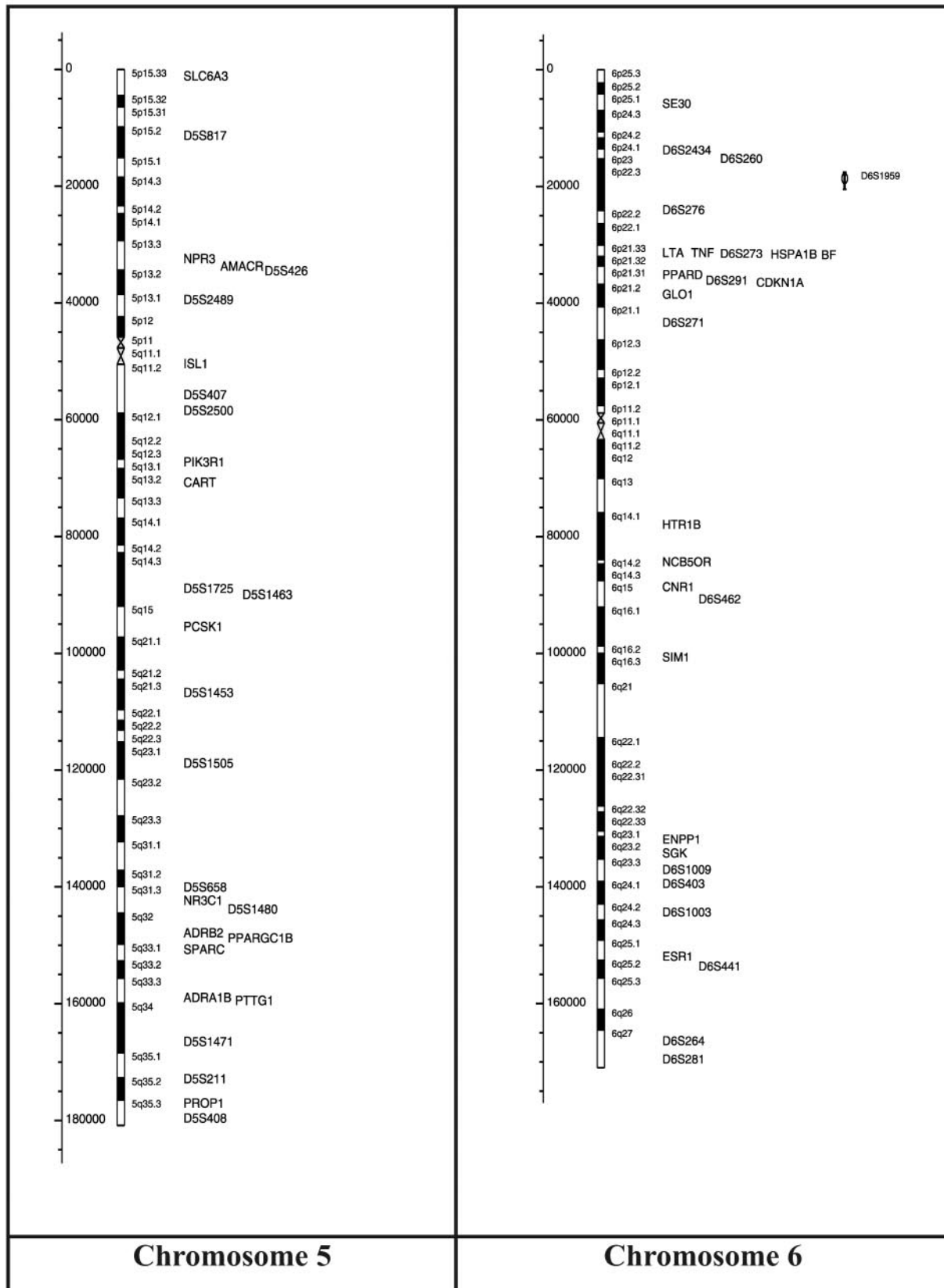
The 2005 human obesity gene map is depicted in Figure 1. The map includes >600 loci from single-gene mutations in mouse models of obesity, non-syndromic human obesity cases due to single-gene mutations, obesity-related Mendelian disorders that have been mapped, transgenic and KO mice models, QTLs from cross-breeding experiments and genome-wide scans, and genes or markers that have been shown to be associated or linked with an obesity phenotype. The map reveals that putative loci affecting obesity-related phenotypes are found on all chromosomes except Y. The number of genes and other markers associated or linked with human obesity phenotypes continues to increase, as indicated by the numbers collated in Table 6. Based on the various lines of evidence reviewed in the different sections of this report, there are now 135 different candidate genes that have been associated and/or linked with obesity-related phenotypes. The majority of the 127 candidate genes associated with obesity have been identified in association studies (Table 4). With the growing number of genes and loci indexed in the map, several genes and QTLs identified from association and genome scan studies have been replicated. We can now identify 22 different genes that have shown

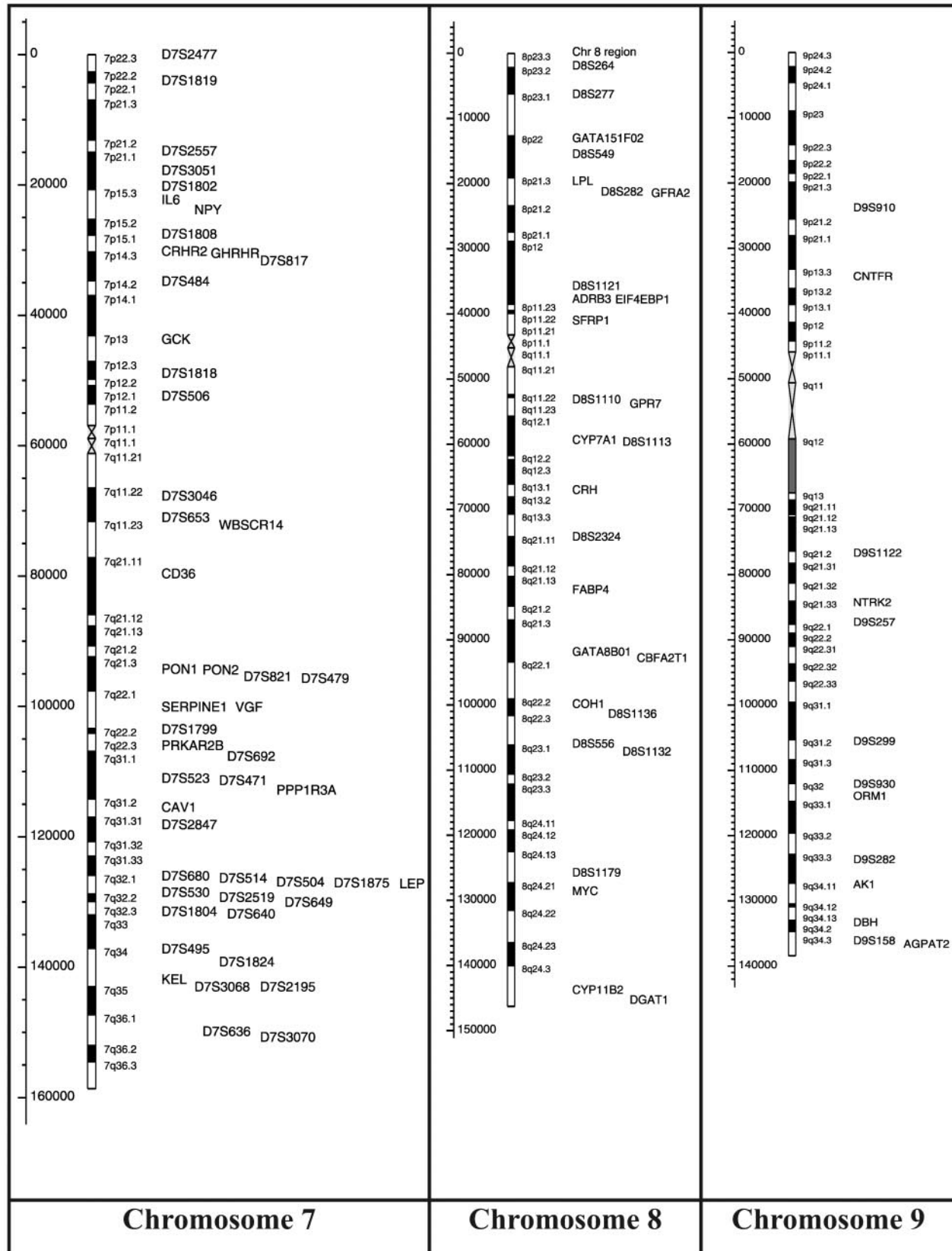
associations with obesity-related phenotypes in at least five studies. Among them, those showing replications in 10 studies and more include *PPARG* (30 studies), *ADRB3* (29), *ADRB2* (20), *LEPR* (16), *GNB3* (14), *UCP3* (12), *ADIPOQ* (11), *LEP* (11), *UCP2* (11), *HTR2C* (10), *NR3C1* (10), and *UCP1* (10). The number of obesity QTLs identified from genome scans now reaches 253, which include 15 QTLs that have been replicated in at least three studies. The large number of genes and loci depicted in the obesity gene map is a good indication of the complexity of the task of identifying genes associated with the susceptibility to obesity. Although several of the genes listed in this report may be false positives, it is also clear that some genes are more important than others based on the numbers of replications from independent studies. A recent meta-analysis of genetic association studies concluded that, although false positive associations are abundant in the literature, 20% to 30% of genetic associations are real and have modest effects on risk of common diseases (1291). This would suggest that perhaps as many as 20 to 30 of the obesity candidate genes identified in this report might contribute to the risk of obesity in humans. Of course, the goal remains to identify the right combination of genes and mutations that are associated with this increased risk and to determine how envi-

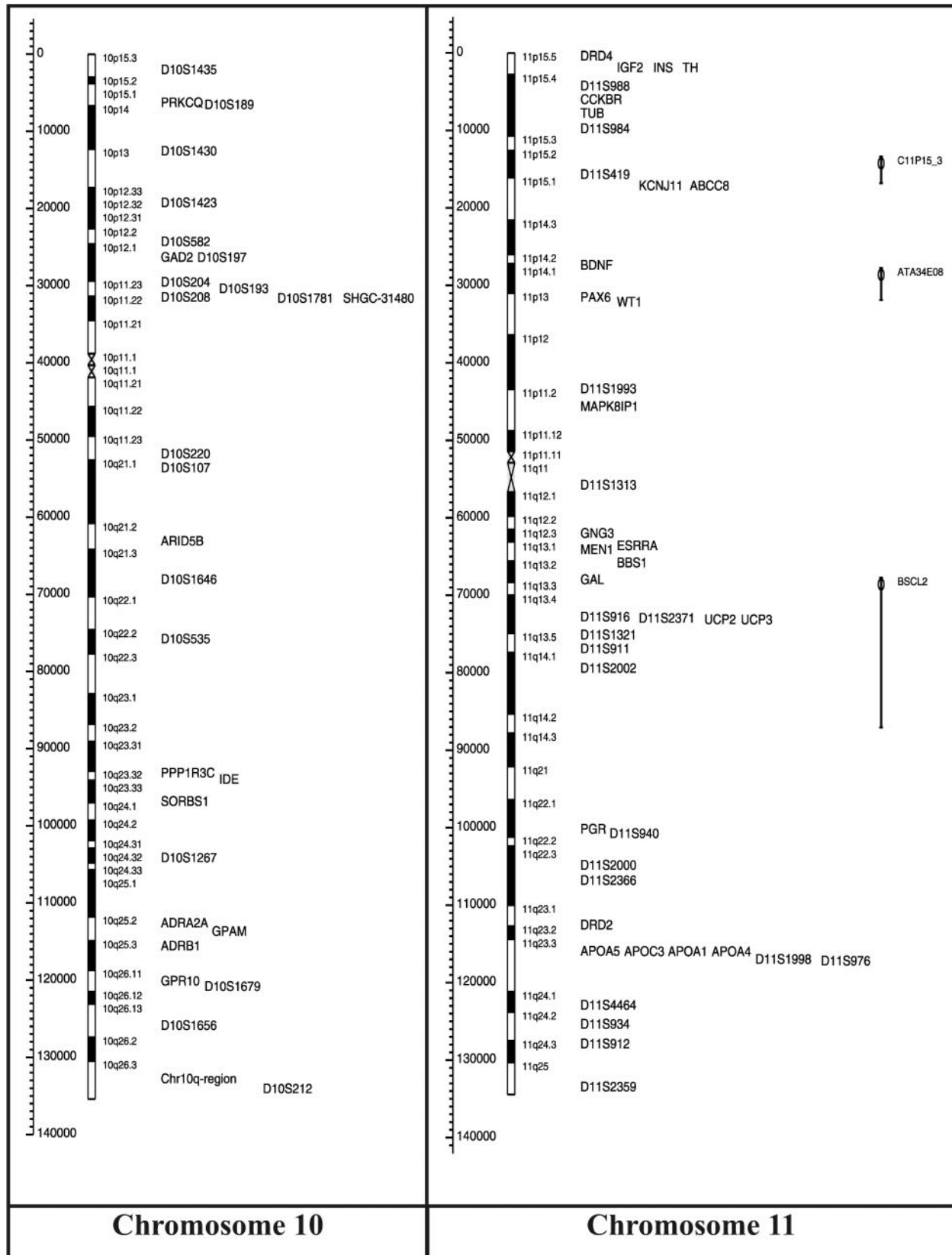
Figure 1: The 2005 human obesity gene map. The map includes all obesity-related genes and QTLs identified from the various lines of evidence reviewed in this article. This year's map consists of a 862-band-resolution cytogenetic map overlaid with build 35.1 of the human genome sequence available from National Center for Biotechnology Information (<http://www.ncbi.nlm.nih.gov>). This allows the human genes (as abbreviated in the tables and appendix and located to the right of each chromosome in this figure) to be placed at precise positions on both the sequence and the cytogenetic map. For all loci, we used the name preferred by UniSTS or Entrez Gene. The ruler to the left of each figure represents kilobasepairs. Chromosomes are drawn to scale only within a given page and not on the last page. These maps, along with information from this report, can be browsed and searched interactively at the Obesity Gene Map web site (<http://obesitygene.pbrc.edu>).

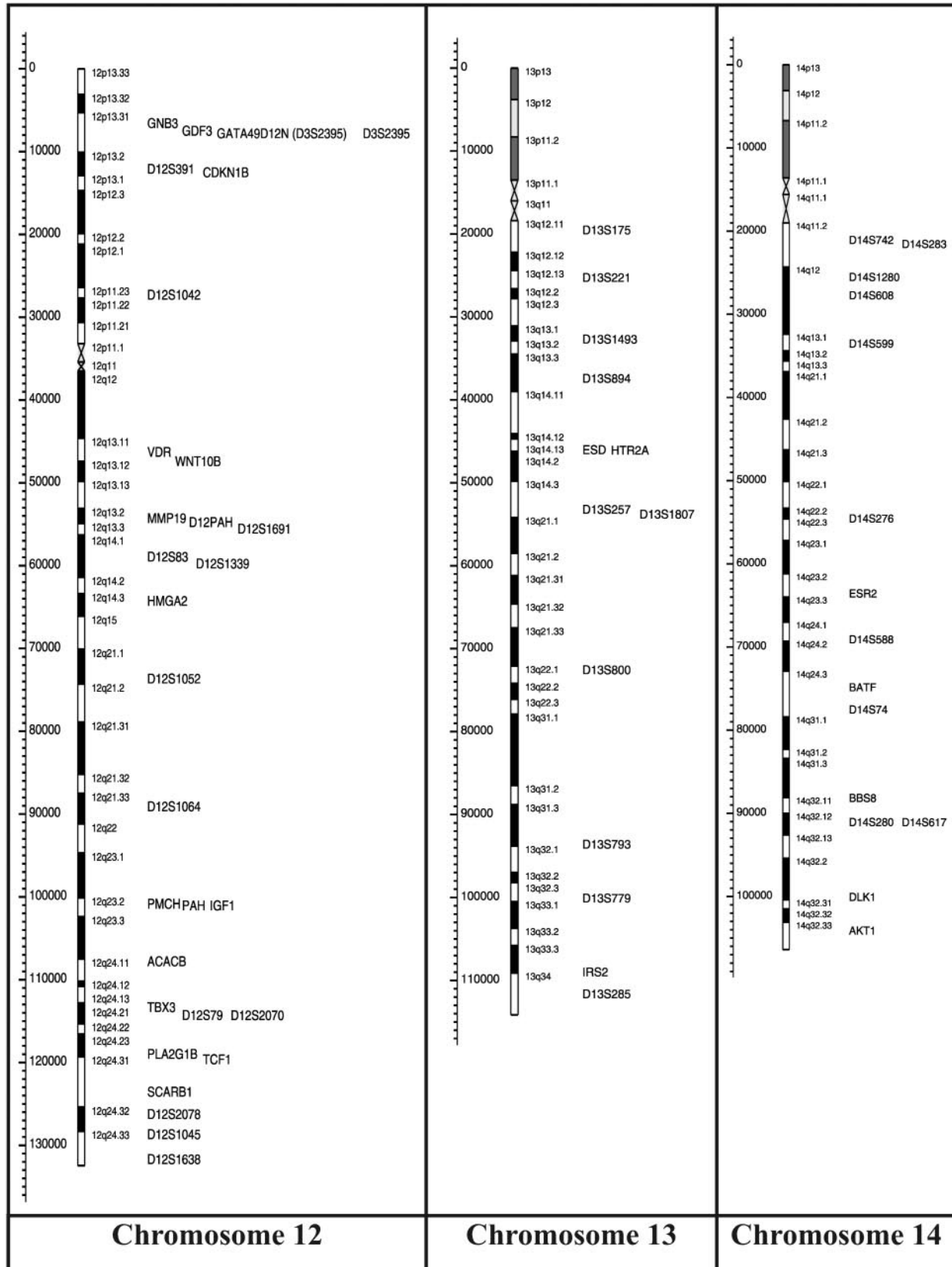


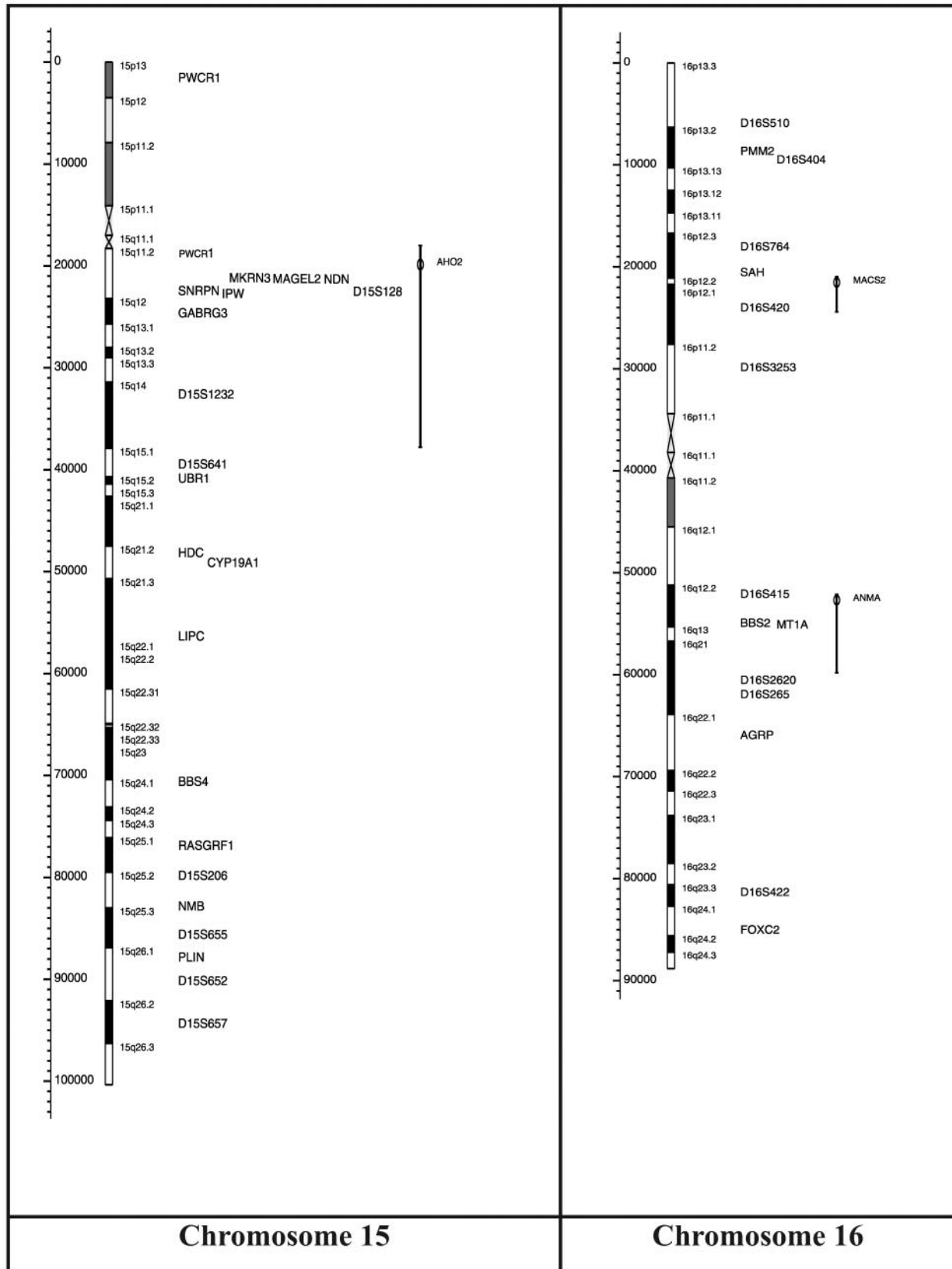


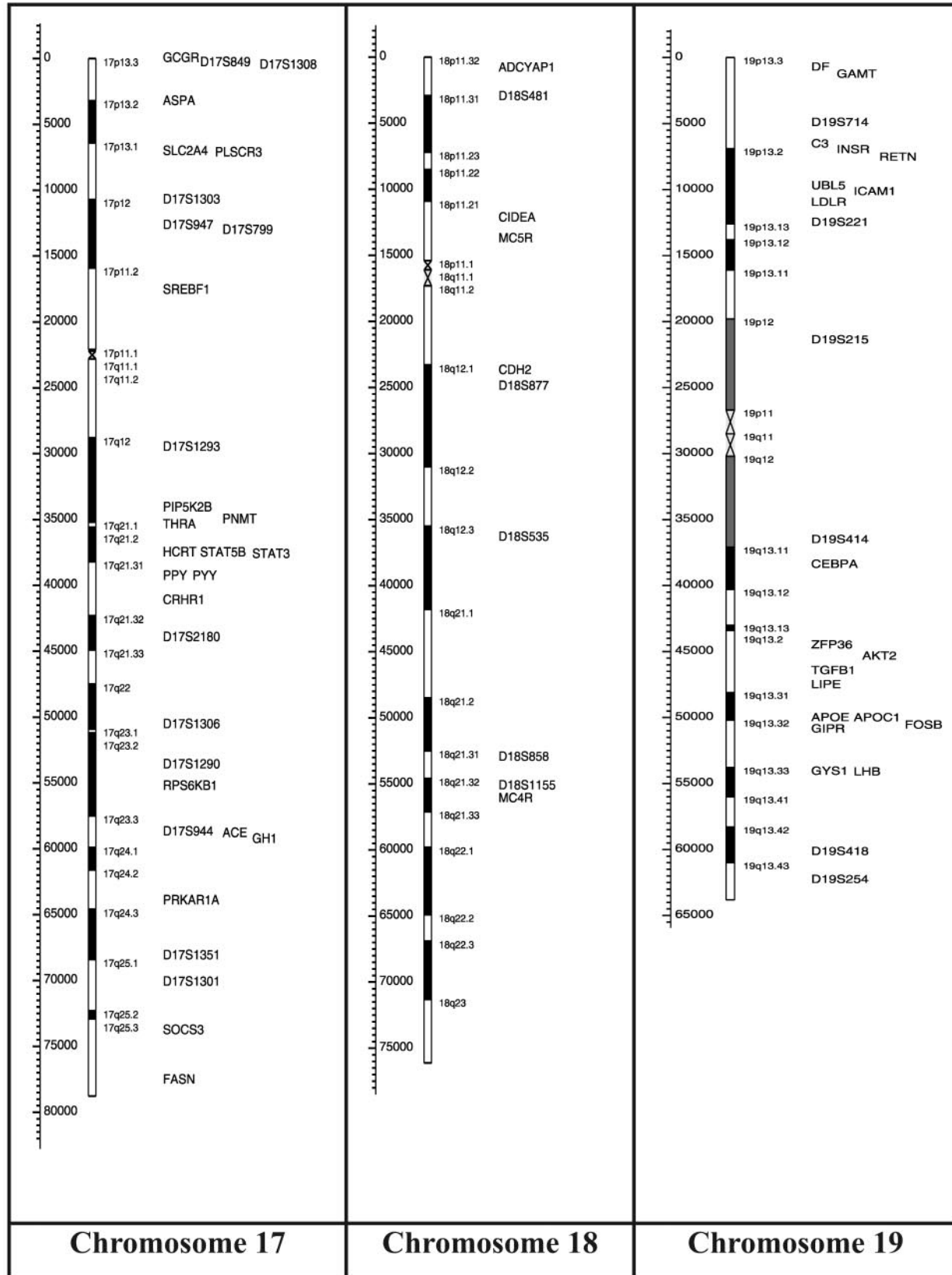


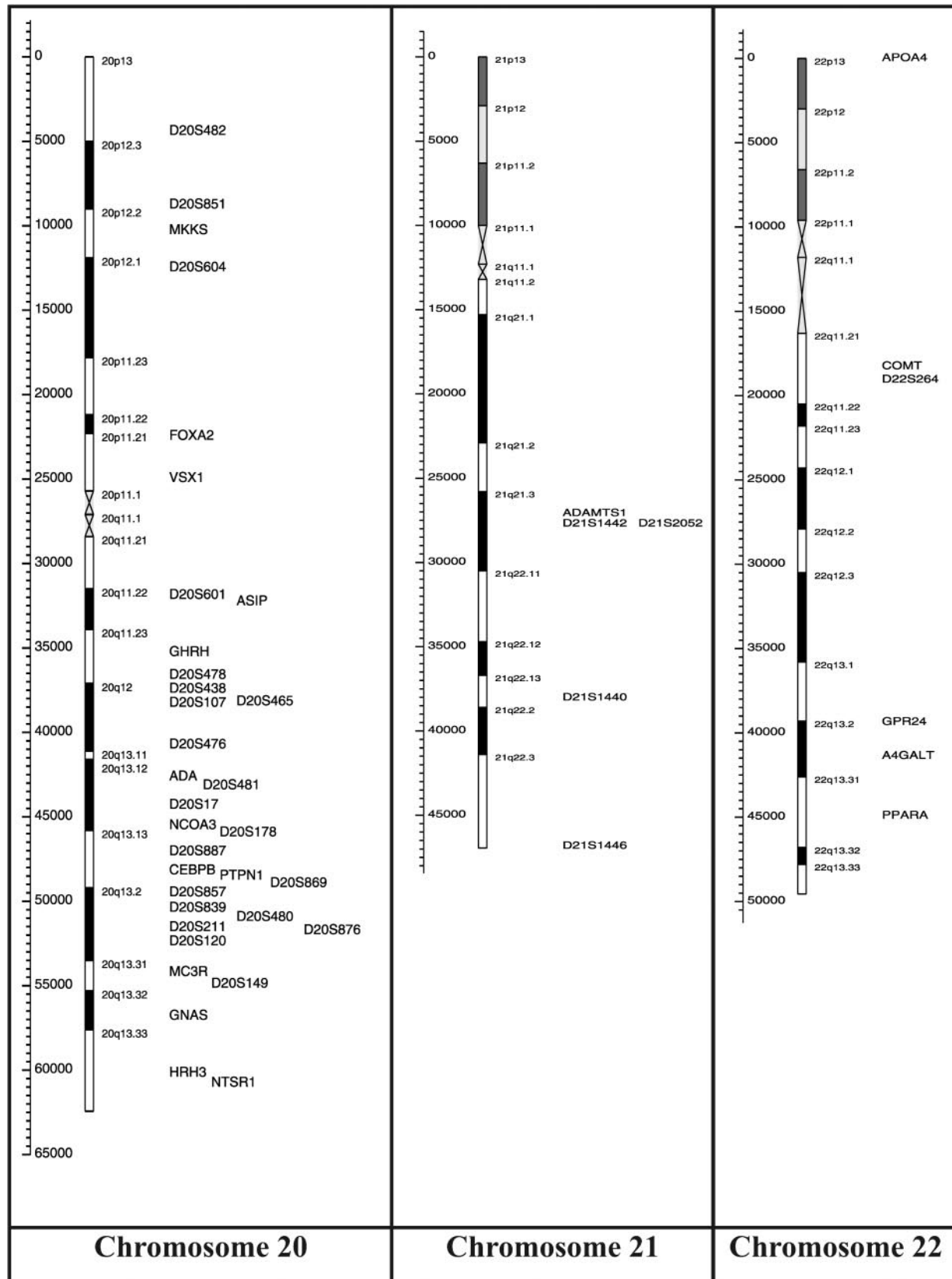


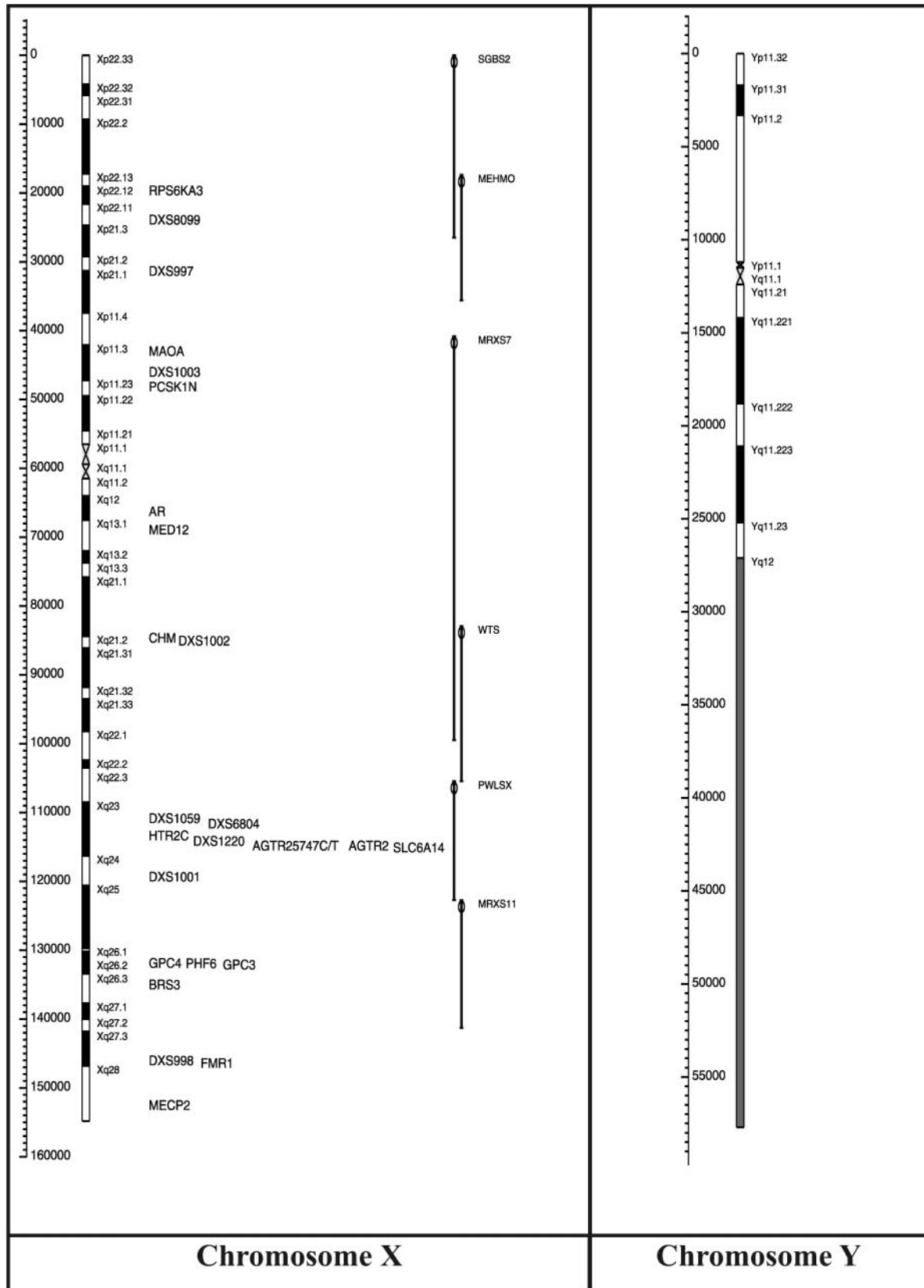












ronmental factors interact with these genes and mutations to determine the risk. We hope that the information provided in this publication will contribute in the years ahead to the resolution of this enormous challenge.

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Appendix. Symbols, full names, and cytogenetic location of genes and loci of the 2005 human obesity gene map

Gene or locus	Name	Location (NCBI)
<i>A4GALT</i>	α 1,4-Galactosyltransferase (P1 blood group)	22q13.31
<i>ABCA7</i>	ATP-binding cassette, subfamily A, member 7	19p13.3
<i>ABCC8</i>	ATP-binding cassette, subfamily C (CFTR/MRP), member 8 (sulfonylurea receptor)	11p15.1
<i>ABCG5</i>	ATP-binding cassette, subfamily G (WHITE), member 5 (sterolin 1)	2p21
<i>ACACB</i>	Acetyl-coenzyme A carboxylase β	12q24.1
<i>ACADVL</i>	Acyl-coenzyme A dehydrogenase, very long chain	17p13-p11
<i>ACE</i>	Angiotensin I-converting enzyme (peptidyl-dipeptidase A) 1	17q24.1
<i>ACP1</i>	Acid phosphatase 1, soluble	2p25
<i>ADA</i>	Adenosine deaminase	20q13.12
<i>ADAM12</i>	ADAM metalloproteinase domain 12 (meltrin α)	10q26.3
<i>ADAMTS1</i>	A disintegrin-like and metalloprotease (repolylin type) with thrombospondin type 1 motif, 1	21q21.2
<i>ADCYAP1</i>	Adenylate cyclase-activating polypeptide 1 (pituitary)	18p11
<i>ADIPOQ</i>	Adiponectin, C1Q, and collagen domain containing	3q27
<i>ADRA1B</i>	Adrenergic, α -1B-, receptor	5q23-q32
<i>ADRA2A</i>	Adrenergic, α -2A-, receptor	10q24-q26
<i>ADRA2B</i>	Adrenergic, α -2B-, receptor	2q11.2
<i>ADRB1</i>	Adrenergic, β -1-, receptor	10q24-q26
<i>ADRB2</i>	Adrenergic, β -2-, receptor, surface	5q31-q32
<i>ADRB3</i>	Adrenergic, β -3-, receptor	8p12-p11.2
<i>AEBP1</i>	AE-binding protein 1	7p13
<i>AGPAT2</i>	1-Acylglycerol-3-phosphate <i>O</i> -acyltransferase 2 (lysophosphatidic acid acyltransferase, β) (Bernardinelli-Seip congenital lipodystrophy 1)	9q34.3
<i>AGRP</i>	Agouti-related protein homolog (mouse)	16q22
<i>AGT</i>	Angiotensinogen (serine or cysteine) proteinase inhibitor, clade A (α -1 anti-proteinase, anti-trypsin; member 8)	1q42.2
<i>AGTR2</i>	Angiotensin II receptor, type 2	Xq22-23
<i>AHO2</i>	AHO 2	15q11-q13
<i>AHSG</i>	α -2-HS-glycoprotein	3q27
<i>AK1</i>	Adenylate kinase 1	9q34.13
<i>AKT1</i>	v-akt Murine thymoma viral oncogene homolog 1	14q32.3
<i>AKT2</i>	v-akt Murine thymoma viral oncogene homolog 2	19q13.1-13.2
<i>ALMS1</i>	Alstrom syndrome 1	2p13
<i>ALPI</i>	Alkaline phosphatase, intestinal	2q37.1
<i>AMACR</i>	α -Methylacyl-coenzyme A racemase	5p13.2-q11.1
<i>ANGPTL4</i>	Angiopoietin-like 4	19p13.3
<i>ANGPTL6</i>	Angiopoietin-like 6	19p13.2
<i>ANMA</i>	Anisomastia (with obesity)	16q13-q21
<i>APOA1</i>	Apolipoprotein A-I	11q23.3
<i>APOA2</i>	Apolipoprotein A-II	1q23.1
<i>APOA4</i>	Apolipoprotein A-IV	11q23.3
<i>APOA5</i>	Apolipoprotein A-V	11q23
<i>APOB</i>	Apolipoprotein B [including Ag(x) antigen]	2p24.2
<i>APOC1</i>	Apolipoprotein C-I	19q13.2
<i>APOC3</i>	Apolipoprotein C-III	11q23.1-q23.2
<i>APOD</i>	Apolipoprotein D	3q26.2-qter
<i>APOE</i>	Apolipoprotein E	19q13.32
<i>AQP7</i>	Aquaporin 7	9p13
<i>AR</i>	Androgen receptor (dihydrotestosterone receptor, testicular feminization, spinal and bulbar muscular atrophy, Kennedy disease)	Xq11.2-q12
<i>ARID5B</i>	AT-rich interactive domain 5B (MRF1-like)	10q21.2
<i>ARL6</i>	ADP-ribosylation factor-like 6	3q11.2
<i>ASIP</i>	Agouti signaling protein, non-agouti homolog (mouse)	20q11.2-q12
<i>ASPA</i>	Aspartoacylase (Canavan disease)	17pter-p13
<i>ATP12A</i>	ATPase, H^+/K^+ transporting, non-gastric, α polypeptide	13q12.12
<i>ATP1A2</i>	ATPase, Na^+/K^+ transporting, $\alpha 2$ (+) polypeptide	1q23.1
<i>ATP1B1</i>	ATPase, Na^+/K^+ transporting, $\beta 1$ polypeptide	1q23.3
<i>ATP8B1</i>	ATPase, class I, type 8B, member 1	18q21-q22
<i>BATF</i>	Basic leucine zipper transcription factor, ATF-like	14q24.3
<i>BBS1</i>	Bardet-Biedl syndrome 1	11q13.1
<i>BBS2</i>	Bardet-Biedl syndrome 2	16q21

Appendix. (continued)

Gene or locus	Name	Location (NCBI)
<i>BBS3(ARL6)</i>	Bardet-Biedl syndrome 3	3p13-p12
<i>BBS4</i>	Bardet-Biedl syndrome 4 (myosin IXA)	15q22.3-23
<i>BBS5</i>	Bardet-Biedl syndrome 5	2q31
<i>BBS7</i>	Bardet-Biedl syndrome 7	4q27
<i>BBS8(TTC8)</i>	Tetratricopeptide repeat domain 8	14q32.1
<i>BDNF</i>	BDNF	11p13
<i>BF</i>	B-factor, properdin	6p21.31
<i>BR53</i>	Bombesin-like receptor 3	Xq26-q28
<i>BSCL2</i>	Bernardinelli-Seip congenital lipodystrophy 2 (seipin)	11q13
<i>BUB1B</i>	BUB1 budding uninhibited by benzimidazoles 1 homolog β (yeast)	15q15
<i>C19orf20</i>	Chromosome 19 open reading frame 20	19p13.3
<i>C3</i>	Complement component 3	19p13.3
<i>CAPN10</i>	Calpain 10	2q37.3
<i>CART</i>	Cocaine- and amphetamine-regulated transcript	5q13.2
<i>CAV1</i>	Caveolin 1	7q31.1
<i>CAV3</i>	Caveolin 3	3p25
<i>CBFA2T1</i>	Core-binding factor, runt domain, α subunit 2; translocated to, 1; cyclin D-related	8q21.3
<i>CBL</i>	Cas-Br-M (murine) ecotropic retroviral transforming sequence	11q23.3
<i>CCKAR</i>	Cholecystokinin A receptor	4p15.1-15.2
<i>CCKBR</i>	Cholecystokinin B receptor	11p15.4
<i>CCND3</i>	Cyclin D3	6p21
<i>CD36</i>	CD36 antigen (collagen type I receptor, thrombospondin receptor)	7q11.2
<i>CDH2</i>	Cadherin 2 (<i>N</i> -cadherin) (<i>N</i> -cadherin 1)	18q11.2
<i>CDKN1A</i>	Cyclin-dependent kinase inhibitor 1A	6p21.2
<i>CDKN1B</i>	p27Kip1	12p13.1-p12
<i>CEBPA</i>	CCAAT/enhancer-binding protein (C/EBP), α	19q13.1
<i>CEBPB</i>	C/EBP, β	20q13.13
<i>CEBPD</i>	C/EBP, Δ	8p11.2-11.1
<i>CHM</i>	Choroideremia (Rab escort protein 1)	Xq21.2
<i>CHRM3</i>	Cholinergic receptor, muscarinic 3	1q41-q44
<i>CIDEA</i>	Cell death-inducing DFFA-like effector a	18p11.21
<i>CLOCK</i>	Clock homolog (mouse)	4q12
<i>CNR1</i>	Cannabinoid receptor (brain)	6q14-q15
<i>CNTR</i>	Ciliary neurotrophic factor receptor	9p13.2
<i>COH1</i>	Cohen syndrome 1	8q22.2
<i>COL8A2</i>	Collagen, type VIII, α 2	1p34.3
<i>COMT</i>	Catechol <i>O</i> -methyltransferase	22q11.21
<i>CORIN</i>	Corin, serine peptidase	4p13-12
<i>CPE</i>	Carboxypeptidase E	4q32.3
<i>CPT1A</i>	Carnitine palmitoyltransferase 1A (liver)	11q13.1-13.2
<i>CRH</i>	Corticotropin-releasing hormone	8q13
<i>CRHR1</i>	Corticotropin-releasing hormone receptor 1	17q12-q22
<i>CRHR2</i>	Corticotropin-releasing hormone receptor 2	7p14.3
<i>CYB5R4</i>	Cytochrome b5 reductase 4	6pter-q22.33
<i>CYP11B2</i>	Cytochrome P450, family 11, subfamily B, polypeptide 2	8q21-q22
<i>CYP19A1</i>	Cytochrome P450, family 19, subfamily A, polypeptide 1	15q21.1
<i>CYP2D6</i>	Cytochrome P450, family 2, subfamily D, polypeptide 6	22q13.1
<i>CYP7A1</i>	Cytochrome P450, family 7, subfamily A, polypeptide 1	8q12.1
<i>DBH</i>	Dopamine β -hydroxylase (dopamine β -monooxygenase)	9q34
<i>DF</i>	D component of complement (adipsin)	19p13.3
<i>DGAT1</i>	Diacylglycerol <i>O</i> -acyltransferase homolog 1 (mouse)	8q24.3
<i>DHCR24</i>	24-Dehydrocholesterol reductase	1p33-31.1
<i>DIO1</i>	Deiodinase, iodothyronine, type I	1p33-p32
<i>DLK1</i>	Δ -Like 1 homolog (<i>Drosophila</i>)	14q32.3
<i>DNAJC3</i>	DnaJ (Hsp40) homolog, subfamily C, member 3	13q32
<i>DPT</i>	Dermatopontin	1q12-23
<i>DRD2</i>	Dopamine receptor D2	11q23.2
<i>DRD3</i>	Dopamine receptor D3	3q13.3
<i>DRD4</i>	Dopamine receptor D4	11p15.5
<i>EIF4EBP1</i>	Eukaryotic translation initiation factor 4E-binding protein 1	8p12

Appendix. (continued)

Gene or locus	Name	Location (NCBI)
<i>ENPP1</i>	Ectonucleotide pyrophosphatase/phosphodiesterase 1	6q23.1
<i>EREG</i>	Epiregulin	4q13.3
<i>ESD</i>	Esterase D/formylglutathione hydrolase	13q14.11
<i>ESR1</i>	Estrogen receptor 1	6q25.1
<i>ESR2</i>	Estrogen receptor 2 (ER β)	14q23.2
<i>ESRRA</i>	Estrogen-related receptor α	11q13
<i>FABP1</i>	Fatty acid-binding protein 1, liver	2p11
<i>FABP2</i>	Fatty acid-binding protein 2, intestinal	4q27
<i>FABP4</i>	Fatty acid-binding protein 4, adipocyte	8q21
<i>FABP5</i>	Fatty acid-binding protein 5 (psoriasis-associated)	8q21.13
<i>FASN</i>	Fatty acid synthase	17q25
<i>FGF21</i>	Fibroblast growth factor 21	19q31.1-qter
<i>FGFR3</i>	Fibroblast growth factor receptor 3 (achondroplasia, thanatophoric dwarfism)	4p16.3
<i>FKHL18</i>	Forkhead-like 18 (<i>Drosophila</i>)	20q11.1-11.2
<i>FMR1</i>	Fragile X mental retardation 1	Xq28
<i>FOSB</i>	FBJ murine osteosarcoma viral oncogene homolog B	19q13.32
<i>FOXA2</i>	Forkhead box A2	20p11
<i>FOXC2</i>	Forkhead box C2 (MFH-1, mesenchyme forkhead 1)	16q22-q24
<i>FSHR</i>	Follicle-stimulating hormone receptor	2p21
<i>FXD4</i>	FXD domain containing ion transport regulator 4	10q11.21
<i>GABRG3</i>	γ -Aminobutyric acid A receptor, $\gamma 3$	15q11-q12
<i>GAD2</i>	Glutamate decarboxylase 2 (pancreatic islets and brain, 65 kDa)	10p11.23
<i>GAL</i>	Galanin	11q13.2
<i>GAMT</i>	Guanidinoacetate <i>N</i> -methyltransferase	19p13.3
<i>GAS6</i>	Growth arrestic-specific 6	13q34
<i>GAST</i>	Gastrin	17q21
<i>GCGR</i>	Glucagon receptor	17q25
<i>GCK</i>	Glucokinase (hexokinase 4, maturity onset diabetes of the young 2)	7p15.3-p15.1
<i>GDF3</i>	Growth differentiation factor 3	12p13.1
<i>GDF8</i>	Growth differentiation factor 8	2q232.2
<i>GFPT1</i>	Glutamine-fructose-6-phosphate transaminase 1	2p13
<i>GFRA2</i>	GDNF family receptor $\alpha 2$	8p21
<i>GH1</i>	Growth hormone (GH) 1	17q22-q24
<i>GHR</i>	GH receptor	5p13-12
<i>GHRH</i>	GH-releasing hormone	20q11.2
<i>GHRHR</i>	GH-releasing hormone receptor	7p14
<i>GHRL</i>	Ghrelin, GH secretagogue receptor ligand	3p26-p25
<i>GHSR</i>	GH secretagogue receptor	3q26.31
<i>GIPR</i>	Gastric inhibitory polypeptide receptor	19q13.3
<i>GLO1</i>	Glyoxalase I	6p21.3-p21.1
<i>GNAS</i>	GNAS complex locus	20q13.2-q13.3
<i>GNB3</i>	Guanine nucleotide-binding protein (G-protein), β polypeptide 3	12p13
<i>GNG3</i>	Guanine nucleotide-binding protein (G-protein), $\gamma 3$	11p11
<i>GPAM</i>	Glycerol-3-phosphate acyltransferase, mitochondrial	10q25.2
<i>GPC1</i>	Glypican 1	2q35-q37
<i>GPC3</i>	Glypican 3	Xq26.2
<i>GPC4</i>	Glypican 4	Xq26.1
<i>GP2D</i>	Glycerol-3-phosphate dehydrogenase 2 (mitochondrial)	2q24.1
<i>GPHB5</i>	Glycoprotein hormone $\beta 5$	14q23.2
<i>GPR10</i>	G-protein-coupled receptor 10	10q26.13
<i>GPR109A</i>	G-protein-coupled receptor 109A	12q24.31
<i>GPR24</i>	G-protein-coupled receptor 24	22q13.3
<i>GPR35</i>	G-protein-coupled receptor 35	2q37.3
<i>GPR40</i>	G-protein-coupled receptor 40	19q13.1
<i>GPR7</i>	G-protein-coupled receptor 7	8p22-q21.13
<i>GPX1</i>	Glutathione peroxidase 1	3p21.3
<i>GRM5</i>	Glutamate receptor, metabotropic 5	11q14.3
<i>GRM8</i>	Glutamate receptor, metabotropic 8	7 (q31.3-q32.1)
<i>GSK3B</i>	Glycogen synthase kinase 3 β	3q13.3
<i>GYP A</i>	Glycophorin A (includes MN blood group)	4q31.1

Appendix. (continued)

Gene or locus	Name	Location (NCBI)
<i>GYS1</i>	Glycogen synthase 1 (muscle)	19q13.33
<i>H6PD</i>	Hexose-6-phosphate dehydrogenase (glucose 1-dehydrogenase)	1pter-p36.13
<i>HCRT</i>	Hypocretin (orexin) neuropeptide precursor	17q21
<i>HD</i>	Huntington (Huntington disease)	4p16.3
<i>HDC</i>	Histidine decarboxylase	15q21-q22
<i>HEXB</i>	Hexosaminidase B (β polypeptide)	5q13
<i>HMGA2</i>	High-mobility group AT-hook 2	12q15
<i>HRH1</i>	Histamine receptor H1	3p25
<i>HRH3</i>	Histamine receptor H3	20q13.3
<i>HSD11B1</i>	Hydroxysteroid (11- β) dehydrogenase 1	1q32-q41
<i>HSD11B2</i>	Hydroxysteroid (11- β) dehydrogenase 2	16q22
<i>HSD3B1</i>	Hydroxy- Δ -5-steroid dehydrogenase, 3 β - and steroid Δ -isomerase 1	1p11.2
<i>HSPA1B</i>	Heat shock 70-kDa protein 1B	6p21.31
<i>HTR1B</i>	5-Hydroxytryptamine (serotonin) receptor 1B	6q14.1
<i>HTR2A</i>	5-Hydroxytryptamine (serotonin) receptor 2A	13q14.11
<i>HTR2C</i>	5-Hydroxytryptamine (serotonin) receptor 2C	Xq24
<i>ICAM1</i>	Intercellular adhesion molecule 1 (CD54), human rhinovirus receptor	19p13.2
<i>IDE</i>	Insulin-degrading enzyme	10q23-q25
<i>IDH1</i>	Isocitrate dehydrogenase 1 (NADP ⁺), soluble	2q33.3
<i>IFRD1</i>	Interferon-related developmental regulator 1	7q22-q31
<i>IGF1</i>	Insulin-like growth factor 1 (somatomedin C)	12q23.3
<i>IGF2</i>	Insulin-like growth factor 2 (somatomedin A)	11p15.5
<i>IGFBP6</i>	Insulin-like growth factor-binding protein 6	12q13
<i>IGKC</i>	Immunoglobulin kappa constant	2p11.2
<i>IL1RN</i>	Interleukin 1 receptor antagonist	2q14.2
<i>IL6</i>	Interleukin 6 (interferon, β 2)	7p21
<i>IL6R</i>	Interleukin 6 receptor	1q22
<i>INPPL1</i>	Inositol polyphosphate phosphatase-like 1	11q23
<i>INS</i>	Insulin	11p15.5
<i>INSR</i>	Insulin receptor	19p13.3-p13.2
<i>IPW</i>	Imprinted in Prader-Willi syndrome	15q11.2
<i>IRS1</i>	Insulin receptor substrate 1	2q36.3
<i>IRS2</i>	Insulin receptor substrate 2	13q34
<i>ISL1</i>	ISL1 transcription factor, LIM/homeodomain, (islet-1)	5q11.2
<i>KCNA3</i>	Potassium voltage-gated channel, shaker-related subfamily, member 3	1p13.3
<i>KCNJ11</i>	Potassium inwardly rectifying channel, subfamily J, member 11	11p15.1
<i>KEL</i>	Kell blood group	7q35
<i>KLF5</i>	Kruppel-like factor 5 (intestinal)	13q22.1
<i>LDLR</i>	Low-density lipoprotein receptor (familial hypercholesterolemia)	19p13.2
<i>LEP</i>	Leptin (obesity homolog, mouse)	7q31.3
<i>LEPR</i>	Leptin receptor	1p31
<i>LHB</i>	Luteinizing hormone β polypeptide	19q13.32
<i>LIPA</i>	Lipase A, lysosomal acid, cholesterol esterase (Wolfman disease)	10q23.2-q23.3
<i>LIPC</i>	Lipase, hepatic	15q21-23
<i>LIPE</i>	Lipase, hormone-sensitive	19q13.2
<i>LMNA</i>	Lamin A/C	1q23.1
<i>LPIN1</i>	Lipin 1	2p25.1
<i>LPL</i>	Lipoprotein lipase	8p21.3
<i>LRPAP1</i>	Low-density lipoprotein receptor-related protein-associated protein 1	4p16.3
<i>LTA</i>	Lymphotoxin α (TNF superfamily, member 1)	6p21.3
<i>MACS2</i>	SAH family member, acyl-coenzyme A synthetase for fatty acids	16p12.3
<i>MAGEL2</i>	MAGE-like 2	15q11.2
<i>MAOA</i>	Monoamine oxidase A	Xp11.4-p11.3
<i>MAPK3</i>	Mitogen-activated protein kinase 3	6p12-p11.2
<i>MAPK8IP1</i>	Mitogen-activated protein kinase 8-interacting protein 1	11p11.2
<i>MC3R</i>	Melanocortin 3 receptor	20q13.2-q13.3
<i>MC4R</i>	Melanocortin 4 receptor	18q22
<i>MC5R</i>	Melanocortin 5 receptor	18p11.21
<i>MECP2</i>	Methyl CpG-binding protein 2 (Rett syndrome)	Xq28

Appendix. (continued)

Gene or locus	Name	Location (NCBI)
<i>MED12</i>	Trinucleotide repeat contain mediator of RNA polymerase II transcription, subunit 12 homolog (yeast)	Xq13.1
<i>MEHMO</i>	Mental retardation, epileptic seizures, hypogonadism and -genitalism, microcephaly, and obesity syndrome	Xp22.13-p21.1
<i>MEN1</i>	Multiple endocrine neoplasia I	11q13
<i>MEST</i>	Mesoderm-specific transcript homolog (mouse)	7q32
<i>MKKS</i>	McKusick-Kaufman syndrome	20p12
<i>MKRN3</i>	Makorin, ring finger protein, 3	15q11.2
<i>MLXIPL</i>	MLX-interacting protein-like	7q11.23
<i>MMP11</i>	Matrix metalloproteinase 11 (stromelysin 3)	22q11.23
<i>MMP19</i>	Matrix metalloproteinase 19	12q14
<i>MRXS11</i>	Mental retardation, X-linked, syndromic 11	Xq26-q27
<i>MRXS7</i>	Mental retardation, X-linked, syndromic 7	Xp11.3-q22.1
<i>MT1A</i>	Metallothionein 1A (functional)	16q13
<i>MTTP</i>	Microsomal triglyceride transfer protein	4q24
<i>MYC</i>	Avian myelocytomatosis viral (v-myc) oncogene homolog	8q24.12-q24.13
<i>NCB5OR</i>	NADPH cytochrome B5 oxidoreductase	6pter-q22.33
<i>NCOA3</i>	Nuclear receptor coactivator 3	20q13.13
<i>NDN</i>	Necdin homolog (mouse)	15q11.2
<i>NHLH2</i>	Nescient helix loop helix 2	1p12-p11
<i>NMB</i>	Neuromedin B	15q22-qter
<i>NMU</i>	Neuromedin U	4q12
<i>NOS2A</i>	Nitric oxide synthase 2A (inducible, hepatocytes)	17q11.2-12
<i>NPB</i>	Neuropeptide B	17q25.3
<i>NPR3</i>	Natriuretic peptide receptor C/guanylate cyclase C (anti-natriuretic peptide receptor C)	5p14-p13
<i>NPY</i>	Neuropeptide Y	7p15.1
<i>NPY1R</i>	Neuropeptide Y receptor Y1	4q31.3-q32
<i>NPY2R</i>	Neuropeptide Y receptor Y2	4q31
<i>NPY5R</i>	Neuropeptide Y receptor Y5	4q31-q32
<i>NR0B2</i>	Nuclear receptor subfamily 0, group B, member 2	1p35.3
<i>NR1H2</i>	nuclear receptor subfamily 1, group H, member 2	19q13.3-13.2
<i>NR1I3</i>	Nuclear receptor subfamily 1, group I, member 3	1q23.3
<i>NR3C1</i>	Nuclear receptor subfamily 3, group C, member 1 (glucocorticoid receptor)	5q31
<i>NTRK2</i>	Neurotrophic tyrosine kinase receptor type 2	9q22.1
<i>NTSR1</i>	Neurotensin receptor 1 (high affinity)	20q13-20q13
<i>OPRM1</i>	Opioid receptor, μ 1	6q24-q25
<i>ORM1</i>	Orosomucoid 1	9q33.1
<i>PAH</i>	Phenylalanine hydroxylase	12q22-q24.2
<i>PARP1</i>	Poly (ADP-ribose) polymerase family, member 1	1q41-q42
<i>PAX6</i>	Paired box gene 6 (aniridia, keratitis)	11p13
<i>PCSK1</i>	Proprotein convertase subtilisin/kexin type 1	5q15-q21
<i>PCSK1N</i>	Proprotein convertase subtilisin/kexin type 1 inhibitor	Xp11.23
<i>PEG3</i>	Paternally expressed 3	19q31.4
<i>PEMT</i>	Phosphatidylethanolamine N-methyltransferase	17p11.2
<i>PGD</i>	Phosphogluconate dehydrogenase	1p36.22
<i>PGR</i>	Progesterone receptor	11q22.2
<i>PHF6</i>	PHD finger protein 6	Xq26.3
<i>PIK3R1</i>	Phosphoinositide-3-kinase, regulatory subunit 1 (p85 α)	5q13.1
<i>PIP5K2B</i>	Phosphatidylinositol-4-phosphate 5-kinase, type II, β	17q12
<i>PLA2G1B</i>	Phospholipase A2, group IB (pancreas)	12q23-q24.1
<i>PLIN</i>	Perilipin	15q26
<i>PLSCR1</i>	Phospholipid scramblase 1	3q23
<i>PLSCR3</i>	Phospholipid scramblase 3	17p13.1
<i>PMCH</i>	Promelanin-concentrating hormone	12q23-q24.1
<i>PMM2</i>	Phosphomannomutase 2	16p13.2
<i>PNMT</i>	Phenylethanolamine N-methyltransferase	17q21.2
<i>POMC</i>	proopiomelanocortin (adrenocorticotropin/ β -lipotropin/ α -melanocyte stimulating hormone/ β -melanocyte stimulating hormone/ β -endorphin)	2p23.3
<i>PON1</i>	Paraoxonase 1	7q21.3
<i>PON2</i>	Paraoxonase 2	7q21.3

Appendix. (continued)

Gene or locus	Name	Location (NCBI)
<i>POR</i>	P450 (cytochrome) oxidoreductase	7q11.2
<i>PPARA</i>	Peroxisome proliferative-activated receptor, α	22q13.31
<i>PPARD</i>	Peroxisome proliferative-activated receptor, Δ	6p21.2-p21.1
<i>PPARG</i>	Peroxisome proliferative-activated receptor, γ	3p25
<i>PPARGC1A</i>	Peroxisome proliferative-activated receptor, γ , coactivator 1 α	4p15.1-15.2
<i>PPARGC1B</i>	Peroxisome proliferative-activated receptor, γ , coactivator 1, β	5q32
<i>PPP1R3A</i>	Protein phosphatase 1, regulatory (inhibitor) subunit 3A (glycogen and sarcoplasmic reticulum-binding subunit, skeletal muscle)	7q31.1
<i>PPP1R3C</i>	Protein phosphatase 1, regulatory (inhibitor) subunit 3C	10q23-q24
<i>PPY</i>	Pancreatic polypeptide	17q21
<i>PRKAA2</i>	Protein kinase, AMP-activated, $\alpha 2$ catalytic subunit	1p31
<i>PRKAG3</i>	AMP-activated protein kinase, AMP-activated, $\gamma 3$ non-catalytic subunit	2q35
<i>PRKAR1A</i>	protein kinase, cAMP-dependent, regulatory, type I, α (tissue-specific extinguisher 1)	17q24.3
<i>PRKAR2B</i>	Protein kinase, cAMP-dependent, regulatory, type II, β	7q22
<i>PRKCQ</i>	Protein kinase C, θ	10p15
<i>PRLHR</i>	Prolactin-releasing hormone receptor	10q26.13
<i>PROPI</i>	Prophet of Pit1, paired-like homeodomain transcription factor	5q35.3
<i>PROX1</i>	Prospero-related homeobox 1	1q32.2-q32.3
<i>PTPN1</i>	Protein tyrosine phosphatase, non-receptor type 1	20q13.1-q13.2
<i>PTPN11</i>	Protein tyrosine phosphatase, non-receptor type 11 (Noonan syndrome 1)	12q24
<i>PTPNS1</i>	Protein tyrosine phosphatase, non-receptor type substrate 1	20p13
<i>PTPRF</i>	Protein tyrosine phosphatase, receptor type, F	1p34
<i>PTTG1</i>	Pituitary tumor-transforming 1	5q35.1
<i>PWCR1</i>	Prader-Willi syndrome critical region 1	15q11.2
<i>PWLSX</i>	Prader-Willi-Like Syndrome, X-linked	Xq23-q25
<i>PYY</i>	Peptide YY	17q21.1
<i>RAI1</i>	Retinoic acid induced 1	17p11.2
<i>RASGRF1</i>	Ras protein-specific guanine nucleotide-releasing factor 1	15q24
<i>REN</i>	Renin	1q32
<i>RETN</i>	Resistin (FIZZ3)	19p13.2
<i>RETNLB</i>	Resistin-like β	3q13.1
<i>RPS6KA3</i>	Ribosomal protein S6 kinase, 90 kDa, polypeptide 3	X p22.2-p22.1
<i>RPS6KB1</i>	Ribosomal protein S6 kinase, 70 kDa, polypeptide 1	17q23.2
<i>RSC1A1</i>	Regulatory solute carrier protein, family 1, member 1	1p36.1
<i>RXRG</i>	Retinoid X receptor γ	1q22-q23
<i>SAH</i>	SA hypertension-associated homolog (rat)	16p13.11
<i>SCARB1</i>	Scavenger receptor class B, member 1	12q24.31
<i>SCD1</i>	Stearyl-coenzyme A desaturase 1	10
<i>SDC1</i>	Syndecan 1	2p24.1
<i>SDC3</i>	Syndecan 3 (N-syndecan)	1pter-p22.3
<i>SERPINE1</i>	Serpin peptidase inhibitor, clade E (nexin, plasminogen activator inhibitor type 1), member 1	7q21.3-q22
<i>SFRP1</i>	Secreted frizzled-related protein 1	8p12-p11.1
<i>SGBS2</i>	Simpson-Golabi-Behmel syndrome, type 2	Xp22
<i>SGK</i>	Serum/glucocorticoid regulated kinase	6q23
<i>SH2B</i>	SH2-B homolog	16p11.2
<i>SIM1</i>	Single-minded homolog 1 (<i>Drosophila</i>)	6q16.3-q21
<i>SLC2A2</i>	Solute carrier family 2 (facilitated glucose transporter), member 2	3q26.31
<i>SLC2A4</i>	Solute carrier family 2 (facilitated glucose transporter), member 4	17p13
<i>SLC6A1</i>	Solute carrier family 6 (neurotransmitter transporter, γ -aminobutyric acid), member 1	3p25-p24
<i>SLC6A14</i>	Solute carrier family 6 (amino acid transporter), member 14	Xq23-q24
<i>SLC6A3</i>	Solute carrier family 6 (neurotransmitter transporter, dopamine), member 3	5p15.33
<i>SNRPN</i>	Small nuclear ribonucleoprotein polypeptide N	15q12
<i>SOAT2</i>	Sterol <i>O</i> -acyltransferase 2	12q13.13
<i>SOCS3</i>	Suppressor of cytokine signaling 3	17q25.3
<i>SORBS1</i>	Sorbin and SH3 domain containing 1	10q24.1
<i>SOX8</i>	SRY (sex determining region Y)-box 8	16p13.3
<i>SPARC</i>	Secreted protein, acidic, cysteine-rich (osteonectin)	5q31.3-q32
<i>SREBF1</i>	Sterol regulatory element-binding transcription factor 1	17p11.2
<i>STAT3</i>	Signal transducer and activator of transcription 3 (acute-phase response factor)	17q21.31
<i>STAT5B</i>	Signal transducer and activator of transcription 5B	17q11.2

Appendix. (continued)

Gene or locus	Name	Location (NCBI)
<i>STK25</i>	Serine/threonine kinase 25 (STE20 homolog, yeast)	2q37.3
<i>TBX3</i>	T-box 3 (ulnar mammary syndrome)	12q24.21
<i>TCF1</i>	Transcription factor 1, hepatic; LF-B1, hepatic nuclear factor (HNF1), albumin proximal factor	12q24.31
<i>TGFB1</i>	Transforming growth factor, β 1 (Camurati-Engelmann disease)	19q13.31
<i>TH</i>	Tyrosine hydroxylase	11p15.5
<i>THRA</i>	Thyroid hormone receptor, α [erythroblastic leukemia viral (v-erb-a) oncogene homolog, avian]	17q11.2
<i>THRB</i>	Thyroid hormone receptor, β [erythroblastic leukemia viral (v-erb-a) oncogene homolog 2, avian]	3p24.1
<i>TKT</i>	Transketolase (Wernicke-Korsakoff syndrome)	3p14.3
<i>TNF</i>	TNF (TNF superfamily, member 2)	6p21.3
<i>TNFRSF1B</i>	TNF receptor superfamily, member 1B	1p36.21
<i>TUB</i>	Tubby homolog (mouse)	11p15.5
<i>TXNIP</i>	Thioredoxin-interacting protein	1q21.1
<i>UBL5</i>	Ubiquitin-like 5	19p13.3
<i>UBR1</i>	Ubiquitin protein ligase E3 component n-recognin 1	15q13
<i>UCP1</i>	Uncoupling protein 1 (mitochondrial, proton carrier)	4q28-q31
<i>UCP2</i>	Uncoupling protein 2 (mitochondrial, proton carrier)	11q13.3
<i>UCP3</i>	Uncoupling protein 3 (mitochondrial, proton carrier)	11q13
<i>UNC5C</i>	unc-5 homolog C (<i>C. elegans</i>)	4q21-q23
<i>VDR</i>	Vitamin D (1,25- dihydroxyvitamin D3) receptor	12q13.11
<i>VGF</i>	VGF nerve growth factor inducible	7q22
<i>VLDLR</i>	Very-low-density lipoprotein receptor	9p24
<i>VSX1</i>	Visual system homeobox 1 homolog, CHX10-like (zebrafish)	20p11.21
<i>WBSR14</i>	Williams Beuren syndrome chromosome region 14	7q11.23
<i>WNT10B</i>	Wingless-type MMTV integration site family, member 10B	12q13
<i>WT1</i>	Wilms tumor 1	11p13
<i>WTS</i>	Wilson-Turner X-linked mental retardation syndrome	Xq21.2-q22
<i>ZFP36</i>	Zinc finger protein 36, C3H type, homolog (mouse)	19q13.1