Physiology & Behavior 103 (2011) 523–529

Contents lists available at ScienceDirect

Physiology & Behavior

journal homepage: www.elsevier.com/locate/phb

Body fat distribution and organ weights of 14 common strains and a 22-strain consomic panel of rats

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ARTICLE INFO

Article history:
Received 8 March 2011
Received in revised form 6 April 2011
Accepted 7 April 2011

Keywords:
Body fat distribution
Dissection
Adipose tissue
Organ size
Organ weight
Genetics
Consomics
QTL

ABSTRACT

The goal of this study was to determine the adiposity of a range of rat strains, including a panel of consomics, to estimate heritability. To that end, we assessed the body fat distribution and organ weights of groups of adult male rats from 3 outbred strains, 11 inbred strains and 22 consomic strains. We measured the weights of the gonadal, retroperitoneal, mesenteric, femoral, subscapular and pericardial white fat depots, the subscapular brown fat depot, the kidneys, liver, heart, spleen, and brain. Strains were compared for the measured weight of each of these adipose depots and organs, and also for these weights adjusted statistically for body size. All individual adipose depot and organ weights were highly heritable, in most cases $h^2 > 0.50$. The fourteen inbred and outbred rat strains were not very different in body length but there was a three-fold difference in body weight, and up to a twenty-fold difference in the weight of some adipose depots. Comparison of the FHH-Chr n^{BN} consomic strains with the FHH host strain revealed 98 quantitative trait loci (QTLs) for body composition and organ weight, with the introgressed chromosome reducing weight or adiposity in most cases. These results can be used to guide the choice of appropriate rat strains for future studies of the genetic architecture of obesity and body size.

Obesity is determined by the amount of lipid stored in adipocytes, which aggregate with other cell types to form adipose depots, sometimes called fat pads. There are five large depots in the rat: the gonadal, retroperitoneal, subscapular, inguinal and mesenteric, as well as several smaller ones (e.g., pericardial) [1]. The gonadal, retroperitoneal and mesenteric depots are associated with the viscera of the abdominal cavity whereas the inguinal and subscapular depots are subcutaneous. In humans, there are well-described metabolic consequences, such as increased risk of high blood pressure and diabetes, related to storing fat in the visceral versus subcutaneous adipose depots [2]. Thus, investigators are interested in the mechanisms whereby increased abdominal (visceral) obesity leads to the metabolic syndrome. The pattern of gene expression differs by adipose depot location but how and why particular depots ultimately differ in weight, or how they might change whole-body metabolism, is unclear [3,4]. However, one contributor to differences in fat pad weight is genotype [5]. Thus a genetic approach to understand the controls of adipose depot weight will be useful, especially in the rat, which is a well-characterized model for the study of nutrition and metabolism [6].

The purpose of this study is twofold. In *Experiment 1*, we wanted to obtain comprehensive information about the distribution of body fat in rats of different strains. To this end, we used rats from 3 outbred and 11 inbred strains. We measured the weights of six depots of white fat (gonadal, retroperitoneal, mesenteric, femoral, subscapular and pericardial) and subscapular brown fat. We also weighed the kidneys, liver, spleen, and brain. In *Experiment 2*, we wanted to begin to understand the underlying genetic effects on body composition. To this end, we measured the same traits in a panel of consomic (chromosome substitution) rat strains. Comparison of strains with a substituted chromosome with control rats allowed us to determine whether genes and their alleles on a particular chromosome change fatness.

1. Methods

1.1. Choice of strains

Over 1000 rat strains have been bred for research, of which about half are inbred [7,8]. Several considerations went into the choice of the strains used here. In *Experiment 1*, we measured three outbred strains that have been used extensively in rat obesity research [9–11], although rarely compared [12] (SD, LE, and WI; see Table 1 for full strain names and abbreviations). The long-term goal of this work is to conduct genetic analyses, and so when we began this study in

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doi:10.1016/j.physbeh.2011.04.006

Table 1
List of rat strain names and abbreviations used in this study.

Short abbreviation	Full strain abbreviation	Strain name
SD^{o}	Crl:CD(SD)	Sprague Dawley CD (IGS)
LE^{o}	Crl:LE	Long Evans
WI^{o}	Crl:WI	Wistar
BN	BN/SsNHsdMcwiCrl	Brown Norway
BUF	BUF/CrCrl	Buffalo
COP	COP/CrCrl	Copenhagen
DA	DA/OlaHsd	Dark Agouti
Dahl	Dahl SS/JrHsdMcwi	Dahl-S (salt-sensitive)
F344	F344/NTac	Fischer 344
FHH	FHH/EurMcwi(Crl)	Fawn Hooded Hypertensive
LEW	LEW/SsNHsd	Lewis
Nob	Noble/CrCrl	Noble
PVG	PVG/OlaHsd	Piebald Virol Glaxo
SHR	SHR/NCrl	Spontaneously Hypertensive
FHH-Chr n^{BN}	FHH/EurMcwi-Chr n$^{BN/SsNHsdMcwi}$	FHH-BN consomic (on chromosome n)

^{o} = outbred strain. Vendor: Crl = Charles River, www.criver.com; Tac = Taconic, www.taconic.com; Hsd = Harlan, www.harlan.com. For Crl:CD(SD), CD = Cesarean-derived, IGS = International Genetic Standard system of breeding. The FHH-Chr n^{BN}/Mcwi strain set used here involves chromosomes from the BN/SsNHsdMcwi (BN) strain introgressed onto the FHH/EurMcwi (FHH) background. "Chr n" refers to any of 22 chromosomes (1–20, X or Y).

December 2005, we chose to test seven commercially-available inbred strains that formed the basis of a panel of single nucleotide polymorphisms (SNPs) [13] (DA, Dahl, F344, FHH, LEW, PVG, and SHR). We also wanted to include maximum genetic diversity in order to capture the widest range of phenotypes. To do this, we consulted a rat phylogenetic tree based on microsatellite markers [14], which led us to include four additional inbred strains (BN, BUF, COP, and Nob), with also the consideration that these were commercially available. Later, we obtained 22 strains of FHH-Chr n^{BN}/Mcwi consomic rats (including the BN and FHH progenitor strains), which we measured in a separate experiment (*Experiment 2*). Consomic strains are useful because differences in phenotype between a consomic and host strain can be attributed to genes in the introgressed chromosome (for reviews, see [15,16]). In our case, each of the 20 autosomes and the X and Y chromosomes from the BN/SsNHsdMcwi (BN) strain have been introgressed onto the FHH/EurMcwi (FHH) background. These strains were developed by Jacob and colleagues at the Medical College of Wisconsin [17], who have collected basic biochemical, cardiac, vascular, histological and renal data from members of the FHH-Chr n^{BN} consomic set [18], but there have been no previous reports to our knowledge of body fat distribution.

Measurements were made on male rats because focusing on one sex has the advantage of simplifying genetic analyses. We are aware that body composition differs between male and female rats, and thus conclusions drawn about genetic architecture in one sex may not pertain to the other [19].

1.2. Maintenance

The experiment protocols were approved by the Animal Care and Use Committee of the Monell Chemical Senses Center. Rats were received at Monell when they were 47–59 days old and measured for some behavioral traits shortly thereafter [20,21]. All the rats were housed in the same vivarium, with an ambient temperature of 23 °C and fluorescent illumination between 0600 and 1800 h. Each rat was housed alone in a 25×18×19 cm hanging cage, with stainless steel back and side walls and a mesh front wall and floor. Powdered AIN-76A diet was available from a 4-oz glass jar that was attached with a stainless-steel spring to the front wall. AIN-76A is a semisynthetic diet containing by weight: 20% protein (casein), 65% carbohydrate (sucrose and cornstarch), 5% fat (corn oil), and 10% fiber (cellulose), minerals and vitamins. It has an energy content of ~15.9 kJ/g. The diet was purchased from Dyets Inc (Bethlehem, PA; catalog no. 100000; [22]). Deionized water was available from a 300-mL glass bottle equipped with a neoprene stopper and a stainless steel sipper tube. Food and water were always available except for brief periods when cups or bottles were replenished. Cardboard sheets under the rats' cages collected excrement and spillage, and were changed frequently.

1.3. Necropsy procedure

Rats in *Experiment 1* were 330–370 days old and those in *Experiment 2* were 123–162 days old when they were killed by injection of pentobarbital sodium and phenytoin sodium, and weighed (±0.1 g; these body weight data have been reported in publications focused on the behavioral test results [20,21]). Rats from Experiments 1 and 2 were of different ages when necropsied because those from Experiment 1 underwent more days of behavioral testing [20,21]. We used a ruler to measure body length, which we considered to be the distance between the bottom of the lower incisors to the anus. All organs were removed and weighed (±0.1 g). Landmarks for the gonadal, perirenal, retroperitoneal, mesenteric, femoral (inguinal), pericardial and subscapular (intrascapular) depots were based on dissection guidelines in the mouse [1]. Brown fat from the subscapular region was separated from white fat and weighed. Visceral fat weight was defined as the summed weight of intra-abdominal fat depots, i.e., gonadal, retroperitoneal and mesenteric. Total fat weight was the sum of the visceral fat weight plus the inguinal, subscapular and pericardial fat depot weights. For bilateral organs like the gonadal adipose depot and kidneys, both left and right organs were removed and weighed. The brain was transected at the brain stem distal to the cerebellum. Olfactory bulbs were removed and weighed with the whole brain.

1.4. Dependent variables and data analysis

Obesity depends on the proportion of fat mass relative to overall body size. We used two methods to assess this: (1) percent fat, and (2) standardized fat mass. Percent fat is the ratio of fat weight/body weight, and is frequently used as a measure of obesity in rodents, although it has limitations [23]. Standardized fat mass is fat mass computed after the variance associated with body size is removed [24] and, although more difficult to calculate, is more preferred from a statistical standpoint. We computed these two variables separately for visceral as well as total fat. Like fat mass, organ weight is usually considered relative to body size. Therefore, for individual organ weights including adipose depots, standardized values were obtained by adjusting for body size as described above.

Statistical analysis differed for the two experiments. For *Experiment 1*, differences among strains were evaluated using an ANOVA (body weight, body length, and percent fat), or a general linear model with body weight and length as covariates (fat mass, visceral fat mass, and individual organ weights), followed by Fisher's LSD post hoc tests to determine the pattern of strain differences. For *Experiment 2*, the parental strains and all consomic strains were compared by ANOVA or general linear models, as described above, but each consomic strain was then compared to the FHH host strain with post hoc tests. Significant differences between a consomic strain and the FHH strain were interpreted to mean that one or more QTL was present on that chromosome [25].

To determine the degree of genetic influence on the traits studied, the ratio of the between-strain sum of squares to the total sum of squares obtained from these analyses was used to estimate heritability (h^2) in the narrow sense [26]. These calculations were conducted separately using unadjusted and standardized values. The rationale for this procedure was that the unadjusted measures are most commonly used and thus allow comparison with other estimates, and the standardized measures provide information about the heritability

of adipose tissue and organ sizes independent of body size. All values presented in the text and tables are means ± standard deviations.

2. Results

2.1. Body weight and length (Tables 2 and 3)

The strains measured in *Experiment 1* varied in body weight, with the heaviest being almost three times the weight of the lightest (LE = 923 ± 98 g; DA = 330 ± 17 g). There was a narrower, albeit substantial, range of average body lengths, with the longest and shortest rats differing by almost 7 cm (SD = 278 ± 8 mm; PVG = 209 ± 10 mm).

In *Experiment 2*, the FHH background strain was heavier and longer than was the BN donor strain (Table 3). The consomic strains did not capture the full range of variation observed between these parental strains, although they still differed in body weight and length (i.e., from 378 ± 23 g to 483 ± 33 g, and 218 ± 11 mm to 239 ± 9 mm). The effect of substituting a BN chromosome (Chr) into the FHH host in most cases made the resulting strain more BN-like, i.e., it reduced both body weight (Chr 1, 2, 3, 4, 6, 8, 11, 14, 18, 20, X and Y) and body length (Chr 1, 3, 6, 8, 14, 16, 17, 18, 20, and X). However, substitution of chromosome 5 had the opposite effect: the FHH-Chr 5^{BN} strain was significantly heavier and longer than was the FHH host strain.

2.2. Total and visceral fat (Tables 4 and 5)

In *Experiment 1*, total body fat ranged six-fold, from 32 ± 4 g (DA) to 208 ± 65 g (LE), and fat as a percentage of body weight ranged almost three-fold, from 8 ± 1% (Dahl) to 22 ± 5% (LE). In general, the largest rats had the most fat and also had the highest percentage of fat, although there were a few exceptions (Table 4). For instance, the Nob strain was heavy but not very fat whereas the F344 strain was lighter, but had a higher percentage of fat than did strains of equivalent body size. For most strains, the majority of their fat was stored in visceral depots, the exception being the BUF strain (which had 49 ± 3% of its fat in visceral depots). The highest proportion of visceral fat was in the PVG rat strain (79 ± 3%; Table 4) and the lowest was in the BUF strain (49 ± 3%; Table 4).

In *Experiment 2*, there were marked differences between the FHH and BN progenitor strains in total and visceral fat, although the proportion of visceral fat was the same for these strains (Table 5). For the consomic strains, the absolute amount of fat was generally less than that of the FHH strain (with the exception of the FHH-Chr 5^{BN} strain) and the range of fat was narrower, from a low of 29 ± 5 g (FHH-Chr 1^{BN}) to a high of 51 ± 10 g (FHH-Chr 5^{BN}). Likewise, the percent of fat relative to body weight varied little, from 7 ± 1% (FHH-Chr 1^{BN}) to 11 ± 2% (FHH-Chr 5^{BN}) and the ratio of visceral-to-total fat was narrow [66 ± 3% (FHH-Chr 3^{BN}) to 71 ± 4% (FHH-Chr 5^{BN})]. The presence of QTLs for total fat mass depended on how fat was adjusted for body size. Using regression methods to adjust for body size, 14 consomic strains differed from the host in total fat (Chr 1, 3, 4, 5, 6, 9, 11, 13, 14, 15, 18, 19, 20 and X) and 13 for visceral fat (Chr 1, 3, 4, 5, 6, 9, 11, 13, 14, 15, 18, 19 and X). When fat was expressed as a ratio (percent fat) the number detected was only 7 for total fat (Chr 1, 9, 14, 15, 18, 19 and X) and 6 for visceral fat (Chr 1, 2, 5, 12, 13 and 18; Table 4).

2.3. Individual adipose depots (Tables 6 and 7)

The white adipose depots differed markedly in weight among the strains, with the largest difference observed for subscapular fat. For this depot, the PVG strain had the least (1.1 ± 0.5 g) whereas the LE

Table 2
Mean body weights and lengths of rats measured in *Experiment 1*.

Strain	n	Body weight, g	Body length, mm
LE	5	923 ± 98^{a}	272 ± 15^{a}
SD	8	856 ± 134^{b}	278 ± 8^{a}
BUF	8	$774 \pm 38^{c,d}$	$246 \pm 8^{b,d-h}$
WI	8	$760 \pm 105^{c,d}$	259 ± 16^{c}
LEW	8	583 ± 50^{e}	$236 \pm 10^{b,d-k}$
COP	8	$514 \pm 38^{f-i}$	$238 \pm 14^{b,d-l,k}$
Nob	8	$494 \pm 55^{f-k}$	$241 \pm 11^{b,d-i}$
Dahl	4	$484 \pm 10^{f-l}$	$230 \pm 7^{d-k}$
FHH	8	$464 \pm 43^{f-l}$	$236 \pm 8^{b,d-k}$
SHR	5	$450 \pm 14^{g-l}$	$232 \pm 4^{d-k}$
F344	8	$439 \pm 36^{g-m}$	$225 \pm 9^{d,g-k}$
BN	7	$423 \pm 24^{h-m}$	$230 \pm 9^{d,e,g-k}$
PVG	8	$383 \pm 26^{k-n}$	$209 \pm 10^{l,m}$
DA	7	$330 \pm 17^{m,n}$	$215 \pm 5^{j,l,m}$

For strain abbreviations, see Table 1. Values are means ± standard deviations. Strains are ordered from heaviest to lightest. Means that do not share a common superscript ($^{a-n}$) differ significantly by post–hoc testing. For instance, the LE and SD strain differ in body weight.

Table 3
Mean body weights and lengths of consomic rats measured in *Experiment 2*.

Strain	n	Body weight, g	Body length, mm
FHH	10	434 ± 32	232 ± 5
FHH-Chr 1^{BN}	10	392 ± 20*	224 ± 11*
FHH-Chr 2^{BN}	10	405 ± 34*	226 ± 10
FHH-Chr 3^{BN}	10	395 ± 20*	225 ± 8*
FHH-Chr 4^{BN}	10	409 ± 13*	229 ± 9
FHH-Chr 5^{BN}	10	483 ± 33*	239 ± 9*
FHH-Chr 6^{BN}	10	378 ± 23*	217 ± 11*
FHH-Chr 7^{BN}	10	439 ± 33	233 ± 13
FHH-Chr 8^{BN}	10	398 ± 63*	222 ± 10*
FHH-Chr 9^{BN}	10	413 ± 27	230 ± 7
FHH-Chr 10^{BN}	10	437 ± 38	228 ± 8
FHH-Chr 11^{BN}	10	394 ± 23*	226 ± 7
FHH-Chr 12^{BN}	10	430 ± 17	228 ± 3
FHH-Chr 13^{BN}	10	422 ± 37	229 ± 7
FHH-Chr 14^{BN}	10	380 ± 24*	218 ± 11*
FHH-Chr 15^{BN}	10	427 ± 33	228 ± 8
FHH-Chr 16^{BN}	5	444 ± 7	223 ± 7*
FHH-Chr 17^{BN}	5	434 ± 34	224 ± 8*
FHH-Chr 18^{BN}	10	393 ± 43*	221 ± 10*
FHH-Chr 19^{BN}	6	421 ± 19	226 ± 8
FHH-Chr 20^{BN}	10	407 ± 25*	225 ± 6*
FHH-Chr X^{BN}	5	402 ± 21*	223 ± 11*
FHH-Chr Y^{BN}	10	408 ± 29*	228 ± 7
BN	15	334 ± 18*	211 ± 6*

Values are means ± standard deviations.
* $p < 0.05$ relative to FHH strain.

Table 4
Indices of obesity obtained from rats measured in *Experiment 1*.

Strain	Total fat, g†	Visceral fat, g†	Total fat, % of body weight	Visceral fat, % of total fat
LE	208 ± 65^{a}	111 ± 24^{a}	22 ± 5^{a}	56 ± 7^{b}
SD	166 ± 70^{b}	$102 \pm 34^{c,d}$	$19 \pm 5^{b,c}$	$62 \pm 5^{c-e}$
BUF	168 ± 13^{b}	$82 \pm 2^{a,b}$	$22 \pm 2^{a,b}$	49 ± 3^{a}
WI	154 ± 58^{b}	$91 \pm 24^{b,c}$	$20 \pm 5^{a-c}$	61 ± 10^{c}
LEW	127 ± 22^{c}	$78 \pm 10^{a,b}$	$22 \pm 3^{a,b}$	$62 \pm 5^{c-e}$
COP	76 ± 14^{d}	51 ± 11^{e}	$15 \pm 2^{d,e}$	$67 \pm 4^{e,f}$
Nob	$62 \pm 18^{d,e}$	39 ± 9^{f}	$12 \pm 2^{e,f}$	$63 \pm 4^{c-f}$
Dahl	$38 \pm 6^{f,g}$	26 ± 4^{i}	8 ± 1^{g}	68 ± 4^{f}
FHH	$51 \pm 10^{e,f}$	$36 \pm 8^{f-h}$	$11 \pm 1^{f,g}$	71 ± 5^{f}
SHR	$45 \pm 7^{f,g}$	$35 \pm 6^{g-i}$	$10 \pm 1^{f,g}$	77 ± 3^{g}
F344	75 ± 9^{d}	53 ± 7^{d}	$17 \pm 1^{c,d}$	71 ± 2^{f}
BN	$38 \pm 7^{f,g}$	$25 \pm 4^{h,i}$	9 ± 2^{g}	$66 \pm 3^{d-f}$
PVG	$43 \pm 8^{f,g}$	$33 \pm 5^{f,g}$	$11 \pm 2^{f,g}$	79 ± 3^{g}
DA	32 ± 4^{g}	$21 \pm 4^{g-i}$	$10 \pm 1^{f,g}$	$66 \pm 3^{d-f}$

Values are means ± standard deviations. Group sizes are given in Table 2. Strains are ordered from heaviest to lightest body weight, following the list in Table 2. Total fat = sum of weights of all dissected adipose tissue. Visceral fat = sum of weights of gonadal, retroperitoneal and mesenteric pads. †Differences among strains in total and visceral fat were assessed with general linear models using body weight and length as covariates. See Table 2 for a description of superscripts.

Table 5
Indices of obesity obtained from FHH-Chr n^{BN} consomic rats measured in *Experiment 2*.

Strain	Total fat, g†	Visceral fat, g†	Total fat, % of body weight	Visceral fat, % of total fat
FHH	44±11	30±7	10±2	67±4
FHH-Chr 1^{BN}	29±5*	20±5*	7±1*	71±4*
FHH-Chr 2^{BN}	40±8	28±5	10±1	70±3*
FHH-Chr 3^{BN}	36±7*	24±4*	9±2	66±3
FHH-Chr 4^{BN}	39±8*	26±6*	9±2	67±3
FHH-Chr 5^{BN}	51±10*	36±8*	11±2	71±4*
FHH-Chr 6^{BN}	38±6*	26±4*	10±1	69±2
FHH-Chr 7^{BN}	41±5	28±4	9±1	69±4
FHH-Chr 8^{BN}	41±13	27±9	10±3	68±5
FHH-Chr 9^{BN}	32±6*	22±4*	8±1*	70±5
FHH-Chr 10^{BN}	42±9	29±6	10±2	69±3
FHH-Chr 11^{BN}	38±9*	26±6*	9±2	68±4
FHH-Chr 12^{BN}	46±9	33±6	11±2	71±4*
FHH-Chr 13^{BN}	37±9*	26±6*	9±2	71±3*
FHH-Chr 14^{BN}	31±8*	21±6*	8±2*	68±4
FHH-Chr 15^{BN}	35±6*	25±4*	8±2*	69±4
FHH-Chr 16^{BN}	44±9	29±6	10±2	67±2
FHH-Chr 17^{BN}	46±11	30±5	11±2	65±4
FHH-Chr 18^{BN}	35±11*	22±8*	9±2*	62±4*
FHH-Chr 19^{BN}	37±9*	24±6*	9±2	66±5
FHH-Chr 20^{BN}	39±6*	27±4	10±1	69±5
FHH-Chr X^{BN}	32±3*	22±2*	8±0*	68±2
FHH-Chr Y^{BN}	37±9	26±6	9±2	70±4
BN	15±4*	11±3*	5±1*	69±3

Values are means±standard deviations. Group sizes are given in Table 3. Total fat = sum of weights of all dissected adipose tissue. Visceral fat = sum of weights of gonadal, retroperitoneal and mesenteric pads. †Differences among strains in total and visceral fat were assessed with general linear models using body weight and length as covariates.
* $p<0.05$ relative to FHH strain.

strain had almost 20-fold more (21.1±5.2 g). There was only about a 2-g (3-fold) difference between the strains with the least and most brown fat (Dahl = 0.77±0.23 g, LE = 2.41±0.72 g).

In both experiments, the BN strain had smaller adipose depots than did the FHH strain, although the differences were not significant in *Experiment 1* for the retroperitoneal, femoral, subscapular, and pericardial depots, or in *Experiment 2* for the brown fat depot. Several of the consomic strains had adipose depot weights significantly below those of the FHH stain [gonadal (N = 12), retroperitoneal (N = 9), mesenteric (N = 10), femoral (N = 16), and subscapular (N = 2)]. However, the FHH strain was exceeded by the FHH-Chr 5^{BN} strain in weight of the gonadal, retroperitoneal, and mesenteric depots, by the FHH-Chr 9^{BN} strain in weight of the pericardial depot, and by the FHH-Chr 2^{BN}, 4^{BN} and 12^{BN} strains in weight of the brown fat depot. Relative to all the other strains, the BN strain had considerably less of all the white fat depots (Table 7).

2.4. Organ weights (Tables 8 and 9)

Kidneys ranged in weight from 2.30±0.23 g (PVG) to 5.01±0.68 g (SD), liver from 13.4±1.4 g (DA) to 48.6±8.7 g (LE), heart from 1.05±0.15 g (PVG) to 2.57±0.50 g (LE), spleen from 0.70±0.06 g (BN) to 1.42±0.18 g (BUF), and brain from 1.80±0.09 g (PVG) to 2.27±0.08 g (LEW). In both experiments, kidneys, liver and heart were heavier in the FHH than BN rats; indeed, the BN strain had the lightest kidneys, liver, and heart of all the strains measured in *Experiment 2*. In contrast, the BN strain had a heavier brain than did the FHH strain in both experiments, and a heavier brain than did all the consomic strains. The spleen was heavier in FHH than BN rats in *Experiment 1* but not *Experiment 2*. There were QTLs involving kidney weight on Chr 1, 5, 10 and 14, liver weight on Chr 1, 4, 5, 8 and 14, heart weight on Chr 5, spleen weight on Chr 1, 5, 7, 8, 10 and 16, and brain weight on Chr 1, 10 and 16.

2.5. Heritability (Table 10)

The pattern of heritability was generally similar between the two experiments, although heritability was uniformly higher in *Experiment 1* than *Experiment 2*. Heritability was higher for unadjusted than adjusted measures, probably due to the heritability of overall body size. However, even after adjustment for body size, heritability for adipose depot and organ weight was still relatively high. For example, 74% of the variance in gonadal adipose depot weight could be explained by genotype after standardization for body weight and length.

3. Discussion

The three outbred and 11 inbred strains we measured in *Experiment 1* showed a more-or-less continuous distribution of body size, fatness and organ weights, with a three-fold difference in body weight and almost a twenty-fold difference in adipose depot weights between some strains. This range of phenotypic diversity was perhaps to be expected, given that laboratory rat strains are derived from many sources [27] and we deliberately chose some of the strains to provide maximum genetic diversity. Nevertheless, the range is impressive, and it certainly rivals that seen among mouse strains, which also differ over three-fold in body weight [19,28].

The body composition data obtained here provide a basis for choosing experimental models for complex trait analysis. As the

Table 6
Adipose depot weights of rats measured in *Experiment 1*.

Strain	Gonadal	Retroperitoneal	Mesenteric	Femoral	Subscapular	Pericardial	Brown fat
LE	28.7 ± 6.3^{a}	61.3 ± 15.3^{a}	$21.2\pm5.2^{b,c}$	74.7 ± 25.4^{a}	21.1 ± 5.2^{a}	$0.79\pm0.37^{b,c}$	2.41 ± 0.72^{a}
SD	27.6 ± 5.5^{a}	54.9 ± 26.2^{b}	13.9 ± 2.8^{c}	50.9 ± 23.1^{b}	12.6 ± 1.4^{b}	$0.93\pm0.50^{a,b}$	$1.03\pm0.35^{c-f}$
BUF	24.3 ± 1.9^{b}	32.8 ± 2.2^{d}	24.8 ± 2.3^{a}	70.6 ± 5.9^{a}	14.1 ± 7.1^{b}	1.26 ± 0.74^{a}	2.07 ± 0.42^{b}
WI	28.9 ± 5.4^{a}	42.0 ± 13.4^{c}	19.8 ± 6.5^{c}	46.7 ± 28.1^{b}	$16.3\pm12.4^{a,b}$	$0.73\pm0.38^{b,c}$	$1.24\pm0.22^{c,d}$
LEW	17.0 ± 1.6^{c}	$37.2\pm6.2^{c,d}$	$23.6\pm3.6^{a-b}$	34.5 ± 7.7^{c}	13.5 ± 8.9^{b}	$0.63\pm0.22^{b-d}$	$1.08\pm0.16^{c-f}$
COP	13.1 ± 1.9^{d}	$19.6\pm3.6^{e,f}$	18.7 ± 7.6^{c}	19.7 ± 2.9^{d}	4.3 ± 2^{c}	$0.60\pm0.29^{b-e}$	$1.22\pm0.36^{c-e}$
Nob	13.5 ± 2.7^{d}	$17.0\pm4.2^{e-g}$	$8.1\pm2.7^{e-g}$	18.8 ± 5.9^{d}	4.2 ± 3.6^{c}	0.28 ± 0.18^{f}	$0.94\pm0.32^{d-f}$
Dahl	$9.9\pm1.3^{e,f}$	9.3 ± 1.3^{h}	$6.8\pm1.7^{f,g}$	$6.7\pm2.8^{e,f}$	2.3 ± 0.9^{c}	$0.26\pm0.07^{e,f}$	$0.77\pm0.23^{f,g}$
FHH	10.6 ± 1.8^{e}	$14.3\pm3.8^{f-h}$	$11.5\pm2.4^{d,e}$	$11.7\pm4.6^{d-f}$	2.6 ± 1.2^{c}	$0.47\pm0.12^{c-f}$	$0.82\pm0.10^{f,g}$
SHR	$9.6\pm0.7^{e,f}$	$14.3\pm1.1^{f-h}$	$10.7\pm4.6^{d-f}$	8.3 ± 1.4^{f}	1.4 ± 1.2^{c}	$0.36\pm0.14^{d-f}$	1.33 ± 0.36^{c}
F344	17.5 ± 2.2^{c}	21.7 ± 3.1^{e}	13.9 ± 2.8^{d}	$17.8\pm1.9^{d,e}$	4.1 ± 1.9^{c}	0.29 ± 0.20^{f}	0.61 ± 0.27^{g}
BN	$8.1\pm0.9^{f,g}$	9.5 ± 1.5^{h}	7.1 ± 1.6^{g}	10.1 ± 2.5^{f}	2.6 ± 1.5^{c}	$0.34\pm0.18^{d-f}$	$1.20\pm0.17^{c-e}$
PVG	12.7 ± 1.6^{d}	$12.2\pm2.6^{g,h}$	$8.5\pm1.7^{e-g}$	7.9 ± 2.7^{f}	1.1 ± 0.5^{c}	0.25 ± 0.12^{f}	$1.23\pm0.19^{c,d}$
DA	7.4 ± 1.1^{g}	8.6 ± 1.1^{h}	5.4 ± 1.4^{g}	9.2 ± 1.1^{f}	1.4 ± 0.4^{c}	0.29 ± 0.05^{f}	$0.91\pm0.13^{e-g}$

Values are means±standard deviations (g). Group sizes are given in Table 2. Gonadal = epididymal adipose depot; retroperitoneal includes perirenal fat; mesenteric = fat associated with the large and smell intestines; femoral = fat under skin of hindlimbs; subscapular = fat underneath the skin between the shoulder blades; pericardial = fat clinging to heart; brown fat = brown tissue near the white subscapular depot. Strains are ordered from heaviest to lightest in body weight. Differences among strains were assessed with general linear models using body weight and length as covariates. See Table 2 for a description of superscripts.

Table 7
Adipose depot weights of FHH-Chr n^{BN} consomic rats measured in *Experiment 2*.

Strain	Gonadal	Retroperitoneal	Mesenteric	Femoral	Subscapular	Pericardial	Brown fat
FHH	8.4 ± 1.8	11.0 ± 2.9	10.1 ± 2.4	11.6 ± 3.2	2.9 ± 1.7	0.34 ± 0.16	0.61 ± 0.19
FHH-Chr 1^{BN}	5.7 ± 1.2*	7.8 ± 1.7*	6.9 ± 1.8*	6.0 ± 0.9*	1.8 ± 0.7	0.25 ± 0.17	0.49 ± 0.22
FHH-Chr 2^{BN}	8.3 ± 1.7	11.4 ± 2.1	8.2 ± 2.1*	9.2 ± 1.9*	2.3 ± 1.4	0.42 ± 0.17	0.79 ± 0.39*
FHH-Chr 3^{BN}	6.9 ± 1.4*	8.4 ± 1.1*	8.2 ± 2.0*	9.4 ± 1.9*	2.4 ± 1.1	0.33 ± 0.14	0.65 ± 0.28
FHH-Chr 4^{BN}	7.0 ± 1.2*	10.2 ± 2.4	8.9 ± 2.7	9.7 ± 1.5*	2.4 ± 1.1	0.36 ± 0.13	0.79 ± 0.20*
FHH-Chr 5^{BN}	11.5 ± 2.1*	12.6 ± 3.0*	12.1 ± 2.1*	12.2 ± 2.8	2.5 ± 1.6	0.49 ± 0.24	0.78 ± 0.30
FHH-Chr 6^{BN}	7.6 ± 0.9	9.4 ± 1.9*	8.9 ± 1.8	8.6 ± 1.3*	2.7 ± 1.0	0.33 ± 0.16	0.62 ± 0.13
FHH-Chr 7^{BN}	8.5 ± 1.3	10.6 ± 1.8	9.2 ± 1.6	9.7 ± 1.5*	2.4 ± 1.2	0.28 ± 0.08	0.64 ± 0.21
FHH-Chr 8^{BN}	7.7 ± 2.2	11.0 ± 3.7	8.7 ± 3.1	10.5 ± 3.9	2.7 ± 1.5	0.37 ± 0.16	0.58 ± 0.24
FHH-Chr 9^{BN}	7.0 ± 1.4*	7.9 ± 1.6*	6.9 ± 1.5*	7.1 ± 1.8*	1.8 ± 1.0*	0.91 ± 0.19*	0.61 ± 0.24
FHH-Chr 10^{BN}	8.1 ± 1.9	11.5 ± 2.3	9.5 ± 2.5	8.6 ± 4.1*	3.0 ± 1.4	0.38 ± 0.11	0.60 ± 0.15
FHH-Chr 11^{BN}	6.9 ± 1.8*	9.2 ± 1.9*	9.6 ± 3.2	9.6 ± 2.3*	2.0 ± 0.7	0.38 ± 0.14	0.64 ± 0.14
FHH-Chr 12^{BN}	8.9 ± 1.2	12.1 ± 2.3	11.6 ± 2.7	9.9 ± 2.6*	3.0 ± 1.9	0.50 ± 0.17	0.80 ± 0.26*
FHH-Chr 13^{BN}	7.4 ± 1.4*	9.8 ± 2.2	9.0 ± 2.4	8.5 ± 2.9*	2.1 ± 1.2	0.48 ± 0.21	0.70 ± 0.28
FHH-Chr 14^{BN}	5.9 ± 1.1*	8.0 ± 2.0*	7.0 ± 2.7*	7.4 ± 1.3*	1.9 ± 1.0	0.29 ± 0.16	0.73 ± 0.25
FHH-Chr 15^{BN}	6.8 ± 1.4*	9.3 ± 1.7*	8.5 ± 1.9	8.3 ± 1.7*	2.2 ± 1.3	0.33 ± 0.08	0.61 ± 0.11
FHH-Chr 16^{BN}	8.2 ± 1.7	11.1 ± 1.5	10.0 ± 2.8	11.6 ± 2.7	2.5 ± 1.3	0.28 ± 0.02	0.70 ± 0.24
FHH-Chr 17^{BN}	8.7 ± 0.7	11.1 ± 2.1	9.8 ± 2.3	11.0 ± 3.5	5.1 ± 3.3*	0.41 ± 0.05	0.74 ± 0.06
FHH-Chr 18^{BN}	6.7 ± 1.9*	8.2 ± 3.1*	6.9 ± 2.8*	9.6 ± 2.8*	3.1 ± 1.4	0.27 ± 0.13	0.60 ± 0.22
FHH-Chr 19^{BN}	6.3 ± 2.1*	9.9 ± 2.3	8.2 ± 2.9*	9.4 ± 2.1*	2.6 ± 1.3	0.33 ± 0.12	0.74 ± 0.27
FHH-Chr 20^{BN}	8.3 ± 1.2	10.5 ± 1.7	8.2 ± 1.3*	9.1 ± 1.7*	2.7 ± 1.7	0.39 ± 0.18	0.69 ± 0.22
FHH-Chr X^{BN}	6.5 ± 0.8*	7.5 ± 1.1*	7.9 ± 0.6*	7.6 ± 1.0*	2.4 ± 1.1	0.28 ± 0.04	0.51 ± 0.10
FHH-Chr Y^{BN}	7.4 ± 2.2*	9.6 ± 1.7	9.2 ± 2.6	8.6 ± 2.0*	2.2 ± 1.3	0.34 ± 0.18	0.64 ± 0.18
BN	4.1 ± 1.2*	3.5 ± 2.9*	2.8 ± 0.7*	3.7 ± 0.9*	1.0 ± 0.5*	0.18 ± 0.04*	0.58 ± 0.14

Values are means ± standard deviations (g). Group sizes are given in Table 3. See Table 6 for description of adipose depots. Differences among strains were assessed with general linear models using body weight and length as covariates.
* $p<0.05$ relative to FHH strain.

genetic organization of inbred rat strains is revealed [29,30], trait information becomes increasingly useful to identify genes that contribute to the range of body size and fatness. Thus the survey of outbred and inbred rats is helpful in identifying target strains for further study. For instance, our results indicate that the PVG strain would be a model of visceral obesity, especially when contrasted with the BUF strain, which had the smallest proportion of abdominal fat.

The FHH-Chr n^{BN} consomic survey was undertaken as a first step toward mapping and identifying genetic variants for obesity and other body composition traits in rats. The use of these consomic strains provides greater experimental control than can be achieved with natural populations or crosses of inbred rodents, because with this method, only a single chromosome is manipulated in each strain. Moreover, because each consomic rat is inbred, experiments can be repeated (or the effect of an irreversible treatment can be compared) using genetically identical individuals. We found that the BN strain is smaller, lighter, and leaner than the FHH strain, and substituting a chromosome (with the exception of Chr 5) from the BN strain into the FHH host strain reduces body size, fatness and the weight of many organs. More than half of the chromosomes conferred a detectable reduction in body weight, length and fatness. Moreover, although the consomic strains had phenotypes that were often closer to the BN strain than the FHH strain, they rarely captured the entire difference between the parental strains. For example, whereas the FHH strain had 10.1 g mesenteric fat and the 22 consomic strains ranged between 6.9 and 11.6 g of mesenteric fat, the BN strain had only 2.8 g of mesenteric fat. On the other hand, the effect of swapping a single chromosome was sometimes quite large, and if the effects of multiple chromosomes were summed together, they would far exceed the difference between the host and donor strains. These results confirm the principle that body weight and obesity are determined by multiple genes, and there is epistasis among loci. This result is consistent with the general observation that interactions among loci are common for body composition and other complex traits [31,32].

Estimating the number of genes involved in body composition is not possible from the results of *Experiment 2* because multiple genes could contribute to the differences in body weight or fatness between a consomic and host strain. Previous studies have shown that a QTL identified from a consomic strain often decomposes into multiple linkages when the chromosome is broken into smaller pieces, i.e., congenics [32], and thus rats may be similar to humans which apparently have dozens of loci of small effect [33]. It is a conservative assumption that BN and FHH strains have at least 10–20 QTLs for body weight and fatness, and probably many additional ones, those with smaller effects which are usually not detected in a conventional breeding study. We have estimated, based on a survey of the effects of gene knockouts, that at least a third of all genes contribute to body weight [34]. This is also likely to be the case for body fatness and fat distribution.

This conclusion is sobering for those attempting to identify genes for body weight or obesity, but our results show more encouragement for the genetic dissection of some endophenotypes. For example, investigators interested in the genetic control of pericardial fat (a depot tied to specific cardiac effects [35]) might focus on chromosome 9 because the FHH-Chr 9^{BN} strain was a clear outlier on this trait. Similarly, subscapular fat weight was linked to only two chromosomes, Chr 9 and

Table 8
Organ weights of rats measured in *Experiment 1*.

Strain	Kidneys	Liver	Heart	Spleen	Brain
LE	4.95 ± 0.58^{a}	48.6 ± 8.7^{a}	2.57 ± 0.50^{a}	1.17 ± 0.22^{d}	2.24 ± 0.18^{a}
SD	5.01 ± 0.68^{a}	36.6 ± 7.6^{b}	2.19 ± 0.32^{b}	$1.19 \pm 0.27^{b,c}$	$2.23 \pm 0.08^{a,b}$
BUF	$4.30 \pm 0.29^{b,c}$	29.4 ± 2.3^{c}	$2.00 \pm 0.60^{b,c}$	1.42 ± 0.18^{a}	$1.82 \pm 0.11^{d,e}$
WI	$4.63 \pm 0.65^{a,b}$	30.0 ± 3.2^{c}	$1.98 \pm 0.30^{b,c}$	1.29 ± 0.24^{b}	$2.18 \pm 0.06^{a,b}$
LEW	$3.23 \pm 0.26^{d,e}$	$22.9 \pm 2.8^{e-g}$	$1.45 \pm 0.13^{d,e}$	$0.73 \pm 0.08^{e,f}$	2.27 ± 0.08^{a}
COP	$2.99 \pm 0.25^{e,f}$	$21.9 \pm 2.9^{f,g}$	$1.53 \pm 0.30^{d,e}$	0.61 ± 0.09^{g}	$1.87 \pm 0.08^{d,e}$
Nob	3.55 ± 0.87^{d}	$25.2 \pm 4.7^{d,e}$	$2.06 \pm 0.39^{b,c}$	$1.23 \pm 0.13^{b,c}$	$1.90 \pm 0.05^{d,e}$
Dahl	4.18 ± 0.48^{c}	$27.3 \pm 0.6^{c,d}$	$2.04 \pm 0.18^{b,c}$	$1.30 \pm 0.16^{a-c}$	$2.11 \pm 0.05^{b,c}$
FHH	4.04 ± 0.45^{c}	$24.9 \pm 2.5^{d-f}$	$1.71 \pm 0.10^{c,d}$	0.84 ± 0.08^{d}	1.93 ± 0.04^{d}
SHR	3.56 ± 0.12^{d}	$21.5 \pm 3.0^{f,g}$	2.15 ± 0.23^{b}	0.84 ± 0.05^{e}	$2.20 \pm 0.07^{a,b}$
F344	$2.99 \pm 0.36^{e,f}$	$22.5 \pm 2.3^{e-g}$	$1.23 \pm 0.12^{e,f}$	0.97 ± 0.12^{d}	2.08 ± 0.08^{c}
BN	2.82 ± 0.22^{f}	21.5 ± 2.8^{g}	1.43 ± 0.22^{e}	$0.70 \pm 0.06^{f,g}$	2.05 ± 0.16^{c}
PVG	2.30 ± 0.23^{h}	17.0 ± 1.9^{h}	1.05 ± 0.15^{f}	$0.74 \pm 0.09^{e,f}$	1.80 ± 0.09^{e}
DA	2.44 ± 0.12^{g}	13.4 ± 1.4^{i}	$1.32 \pm 0.18^{e,f}$	$0.65 \pm 0.04^{f,g}$	$1.84 \pm 0.12^{d,e}$

Values are means ± standard deviations (g). Group sizes are given in Table 2. Differences among strains were assessed with general linear models using body weight and length as covariates. See Table 2 for a description of superscripts.

Table 9
Organ weights of FHH-Chr n^{BN} consomic rats measured in *Experiment 2*.

Strain	Kidneys	Liver	Heart	Spleen	Brain
FHH	3.07 ± 0.21	19.6 ± 1.9	1.57 ± 0.25	0.74 ± 0.15	1.73 ± 0.09
FHH-Chr 1^{BN}	2.79 ± 0.22*	17.1 ± 2.0*	1.49 ± 0.16	0.86 ± 0.10*	1.88 ± 0.17*
FHH-Chr 2^{BN}	2.91 ± 0.19	19.6 ± 2.3	1.60 ± 0.19	0.67 ± 0.08	1.75 ± 0.08
FHH-Chr 3^{BN}	3.06 ± 0.36	18.9 ± 1.6	1.52 ± 0.17	0.68 ± 0.09	1.72 ± 0.08
FHH-Chr 4^{BN}	2.91 ± 0.19	17.3 ± 1.3*	1.59 ± 0.26	0.77 ± 0.11	1.83 ± 0.08
FHH-Chr 5^{BN}	3.75 ± 0.24*	22.4 ± 2.4*	1.90 ± 0.19*	0.84 ± 0.07*	1.78 ± 0.21
FHH-Chr 6^{BN}	3.08 ± 0.31	18.1 ± 2.1	1.65 ± 0.15	0.77 ± 0.15	1.76 ± 0.09
FHH-Chr 7^{BN}	3.01 ± 0.38	20.3 ± 2.8	1.60 ± 0.22	0.64 ± 0.04*	1.81 ± 0.09
FHH-Chr 8^{BN}	2.95 ± 0.15	16.8 ± 3.4*	1.67 ± 0.28	0.64 ± 0.13*	1.82 ± 0.06
FHH-Chr 9^{BN}	3.13 ± 0.33	18.7 ± 2.6	1.57 ± 0.34	0.69 ± 0.08	1.83 ± 0.15
FHH-Chr 10^{BN}	3.58 ± 0.79*	20.4 ± 2.7	1.69 ± 0.30	0.91 ± 0.19*	1.92 ± 0.14*
FHH-Chr 11^{BN}	2.89 ± 0.12	18.6 ± 0.8	1.64 ± 0.22	0.70 ± 0.10	1.74 ± 0.12
FHH-Chr 12^{BN}	3.21 ± 0.38	20.6 ± 2.1	1.51 ± 0.34	0.81 ± 0.17	1.74 ± 0.24
FHH-Chr 13^{BN}	3.24 ± 0.35	20.0 ± 1.6	1.67 ± 0.28	0.76 ± 0.22	1.77 ± 0.18
FHH-Chr 14^{BN}	2.70 ± 0.25*	16.5 ± 1.6*	1.55 ± 0.22	0.67 ± 0.09	1.77 ± 0.19
FHH-Chr 15^{BN}	3.02 ± 0.34	18.7 ± 2.8	1.60 ± 0.15	0.82 ± 0.12	1.78 ± 0.06
FHH-Chr 16^{BN}	3.15 ± 0.39	20.3 ± 1.0	1.66 ± 0.28	1.00 ± 0.15*	1.91 ± 0.15*
FHH-Chr 17^{BN}	3.40 ± 0.45	20.6 ± 0.7	1.84 ± 0.04	0.80 ± 0.05	1.90 ± 0.13*
FHH-Chr 18^{BN}	2.85 ± 0.31	18.3 ± 2.1	1.53 ± 0.18	0.72 ± 0.10	1.77 ± 0.06
FHH-Chr 19^{BN}	3.12 ± 0.14	19.9 ± 1.2	1.76 ± 0.18	0.76 ± 0.05	1.77 ± 0.09
FHH-Chr 20^{BN}	2.96 ± 0.22	18.9 ± 1.3	1.60 ± 0.25	0.78 ± 0.15	1.73 ± 0.06
FHH-Chr X^{BN}	3.02 ± 0.28	19.0 ± 0.6	1.57 ± 0.15	0.68 ± 0.07	1.70 ± 0.08
FHH-Chr Y^{BN}	3.02 ± 0.21	18.2 ± 1.9	1.58 ± 0.28	0.70 ± 0.08	1.79 ± 0.06
BN	2.18 ± 0.19*	14.5 ± 1.8*	1.30 ± 0.21*	0.73 ± 0.12	1.99 ± 0.12*

Values are means ± standard deviations (g). Group sizes are given in Table 3. Differences among strains were assessed with general linear models using body weight and length as covariates.
* $p<0.05$ relative to FHH strain.

17, suggesting that the underlying genetic architecture may be relatively simple, at least in the FHH-BN pair of strains measured here. These results also demonstrate that organ size genes can be distinct from those contributing to body size.

In this study, we measured body weight, length and fatness, as well as organ weights. Not all traits were equally heritable. Body weight was generally the most heritable, both in *Experiment 1* and *Experiment 2*. Heritability was lower in *Experiment 2* than *1*, perhaps because the consomic rats had less genetic diversity or were younger, which would allow less time for a phenotype to develop, compared with the inbred and outbred rats in *Experiment 1*. One pattern we observed was that the traits with the highest heritability also had the largest number of QTLs. As an example, for adipose depots, the heritability was ranked from high to low, as follows: gonadal ($h^2 = 0.47$), retroperitoneal (0.47), mesenteric (0.42), femoral (0.41), subscapular (0.18) and pericardial (0.11). The numbers of QTLs involved were 13, 10, 11, 16, 2, and 1 respectively. Therefore there is a relationship between heritability and the number of consomic strains that differ from the host background, which confirms that the heritability is accounted for by multiple QTLs on different chromosomes. The direction of effect of the QTL generally matched the difference between the host and donor strains, e.g., the substituted chromosome from the smaller BN rats reduced body weight and length. This pattern was reversed for brain size, here the BN had the larger brain and the effect of its QTL-containing chromosomes was to increase it. Overall these observations suggest an orderly genetic architecture in which higher heritability is related to QTL abundance, with QTLs having the direction of allelic effects expected based on the host and donor strain differences.

Table 10
Heritability (h^2) estimates for adipose depot and organ weights.

Trait	*Experiment 1*		*Experiment 2*	
	Unadjusted	Adjusted	Unadjusted	Adjusted
Body weight	0.88	NA	0.51	NA
Body length	0.79	NA	0.33	NA
Gonadal fat	0.88	0.74	0.47	0.31
Retroperitoneal fat	0.81	0.56	0.47	0.33
Mesenteric fat	0.76	0.63	0.42	0.27
Femoral fat	0.80	0.44	0.41	0.33
Subscapular fat	0.39	0.18	0.18	0.14
Pericardial fat	0.50	0.24	0.11	0.10
Brown fat	0.71	0.57	0.13	0.13
Kidney	0.82	0.55	0.50	0.30
Liver	0.84	0.62	0.42	0.19
Heart	0.70	0.51	0.20	0.13
Spleen	0.81	0.77	0.32	0.32
Brain	0.76	0.71	0.20	0.22
Fat, total	0.80	0.42	0.46	0.30
Visceral, total	0.84	0.63	0.48	0.31
% Fat	0.79	NA	0.42	NA
% Visceral fat	0.76	NA	0.26	NA

Heritability was estimated from the ratio of $SS_{between\ strains}/SS_{total}$ from the ANOVA results. Unadjusted = the actual weight of the tissue or organ. Adjusted = based on standardized residuals from regression analysis with body weight and body length as covariates, as described in the text.

There are surprisingly few rat strain comparisons of body weight or composition available in the literature (e.g., [12,36–40]). There is a repository of body weight and organ information available for the FHH-Chr n^{BN} consomic strain set on-line [41], but comparison between our results and data in this repository is difficult because the on-line data originates from animals of different ages fed different diets. Compared with rats, more strain surveys have been undertaken with mice [19,28], and this includes several studies using consomic mouse strains to test their resistance or susceptibility to obesity when fed a diet high in fat and calories [42–44]. In this study, our goal was not to study diet and obesity, even though relative to typical laboratory chows, the diet we chose has a slightly higher energy density (15.9 vs. ~13 kJ/g) and more carbohydrate (68% vs. ~59% of total energy) from sucrose compared with laboratory chow. Based on comparisons made in mice [45], the AIN-76A diet appears to be more "obesigenic" than chow but not nearly as much as the high fat diets typically used to produce obesity. Therefore our study does not specifically model dietary obesity, and the rats maintained a low-to-average proportion of body fat, e.g., less than 20% in most cases. Studies of dietary susceptibility would be of interest, especially because previous work in the Sprague Dawley strain suggests that it harbors genes and their alleles that can affect dietary obesity [9].

In summary, rats fed a standard, nutritionally complete diet show tremendous diversity in body size and adiposity. The distribution of body fat is highly heritable, making it possible to study genes with the propensity to store visceral versus subcutaneous fat, or other patterns of fat deposition. The data provided here can guide the choice of rat strains used to understand the genetic and physiological bases of this diversity. QTL studies could map obesity loci from the BN and FHH strains, and could focus on obesity or visceral obesity, or the weight of individual adipose depots.

Acknowledgments

We thank Dr. Mary Shimoyama and other members of the RGD rat forum for advice choosing the rat strains measured here. Stacy Hultine, Stephanie Craw, Brian Gantick and Laurie Pippett provided excellent technical assistance. Supported by NIH grant DK-46791 and DC-10149 to MGT and DK-58797 to DRR. The consomic rats used in this study were provided by a Seed Grant Program sponsored by Physiogenix Inc. [The consomic rats are no longer available from Physiogenix but can be obtained from Dr. Mindy Dwinell at the Medical College of Wisconsin. We thank Dr. Howard Jacob and colleagues for generating and distributing these rats.] Comments from Dr. Mark I. Friedman on drafts of this manuscript are gratefully acknowledged.

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