GENETIC AND BEHAVIORAL ANALYSIS OF METHAMPHETAMINE-INDUCED ANOREXIA IN MICE

by

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ABBREVIATIONS

AVGBASE	average baseline consumption (Experiment 1)
B6	C57BL/6J inbred mouse strain
BXD	Recombinant inbred strain C57BL/6J by DBA/2J
cM	centimorgan
DA	Dopamine neurotransmitter
D2	DBA/2J inbred mouse strain
Drd1a or D1	Dopamine receptor subtype 1
F_2	genetically segregated intercross
lod	logarithm of the odds
MA	Methamphetamine hydrochloride
MICROPCTRED	percent reduction in food consumption (Experiment 3)
MICROPCTWRED	percent weight reduction (Experiment 3)
MICROAVGBASE	average baseline consumption (Experiment 3)
MDS	mesolimibic dopamine system
NE	norepinephrine
PCTRED	percent reduction in food consumption (Experiment 1)
PCTWRED	percent weight reduction (Experiment 1)
PCR	polymerase chain reaction
PFN	perifornical hypothalamic nuclei
PVN	paraventricular hypothalamic nuclei
QTLs	quantitative trait loci
r2	coefficient of genetic determination
RI	recombinant inbred strain
RFLP	restriction fragment length polymorphisms
SDP	strain distribution pattern of strain means
SSLP	simple sequence length polymorphisms
TOT	total food consumed on test day
270TOT	total food consumed during afternoon feeding period.
5-HT	serotonin

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ABSTRACT

Amphetamines decrease food consumption in both humans and animals. This thesis employed a two-phase genetic mapping strategy to detect and identify the genes mediating this effect. In the first phase, BXD recombinant inbred mice were tested on a five-day scheduled feeding paradigm which resulted in stable food intake. Mice from each strain were then divided into two equal groups. On Day 6, Group 1 was given the saline vehicle, s.c., whereas Group 2 was given 1.0 mg/kg methamphetamine (MA). The next day (Day 7) both groups were given MA. Consumption was measured for the first 30 minutes after injection on both Day 6 & 7. Significant drug and strain effects for the 24 strains tested were seen. Food consumption was decreased on average by 54%. In the extreme strains, food consumption was decreased by as much as 86% (BXD-1) or as little as 15% (BXD-11). Following behavioral testing, quantitative trait loci (QTL) analysis ($p< 0.01$) identified five putative QTLs near the markers *Hcf-3* on chromosome 4, *Fv2* on chromosome 9, *D12Nyu1* on chromosome 12, *Tpmt* on chromosome 13, and *D19Byu1* on chromosome 19. However, multiple regression analysis indicated support for at the most two QTLs. In the second phase, verification of putative QTLs using PCR in an independent F_2 population was attempted for the five loci using *Mit* microsatellite markers. Data from this phase were analyzed by the linkage analysis programs MAPMAKER/EXP and MAPMAKER/QTL. Results of the

confirmation process indicated the locus indicated by the marker *Tpmt* on chromosome 13 to be a suggestive linkage per Lander and Kruglyak standards (1995). This QTL is only 1 cM from the dopamine D1 receptor subtype (*Drd1a*) locus. This region has extensive linkage homology with portions of human chromosome 5 where the human dopamine D1 receptor locus is located. This finding supports previously reported pharmacology studies suggesting that the dopamine D1 receptor has a key role in the control of feeding behavior.

Finally, a microstructure study analyzing both consummatory and non-consummatory behaviors was performed with select BXD recombinant inbred strains which showed differential sensitivity to MA-induced anorexia. The study showed that MA at this dose not only reduced food intake, but also altered some non-consummatory behaviors. This raises the possibility that MA-induced anorexia is the consequence of D1 dopamine receptor mediation of both consummatory and non-consummatory behaviors.

INTRODUCTION

In recent years there has been a steady increase in the abuse of psychostimulant drugs. A class of commonly abused stimulants is the amphetamines. The most commonly abused amphetamine is methamphetamine hydrochloride (MA) which is sold "on the streets" as ice, crank, crystal, crystal meth, or speed. All of these forms of methamphetamine are easily synthesized from ephedrine, and are widely available (Cho, 1990).

The stimulant properties of ephedrine, the precursor of methamphetamine and amphetamine (AMP), were first identified by the Chinese over 5100 years ago. Amphetamine was initially introduced in 1932 as an ephedrine analog for bronchodilation in the treatment of nasal and bronchial congestion associated with colds. Methamphetamine, an n-methyl homologue of amphetamine, was introduced about the same time as amphetamine and was used for its similar bronchodilating effects (Cho, 1990). Soon after the introduction of these synthetic compounds their stimulant effects were discovered by abusers. Abuse of amphetamine and methamphetamine occurred as early as the 1940s and 1950s. Epidemics of abuse occurred in Japan in the 1950s, Sweden in the 1950s and the United States in the 1960s and 1970s. The development of ice, a new pure preparation of methamphetamine

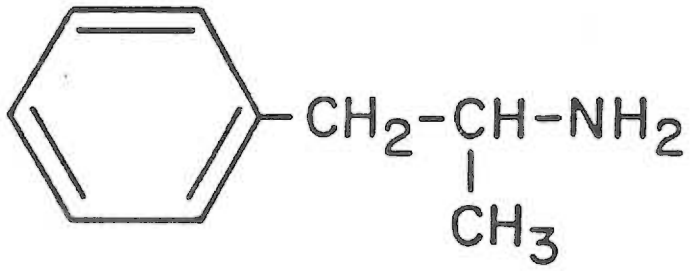
which is inhaled for quick absorption, has increased methamphetamine's abuse in recent years (Cho, 1990).

Methamphetamine has both central and peripheral effects similar to amphetamine. Centrally, methamphetamine causes the release of dopamine (DA) from newly synthesized pools, blocks dopamine reuptake, and inhibits dopamine catabolism by inhibition of monoamine oxidase. Methamphetamine also causes serotonin (5-HT), and norepinephrine (NE) to be released from monoamine containing neurons. Peripherally, methamphetamine stimulates dopaminergic, alpha- and beta-adrenergic systems (Cooper, Bloom and Roth, 1992; Surgue, 1987).

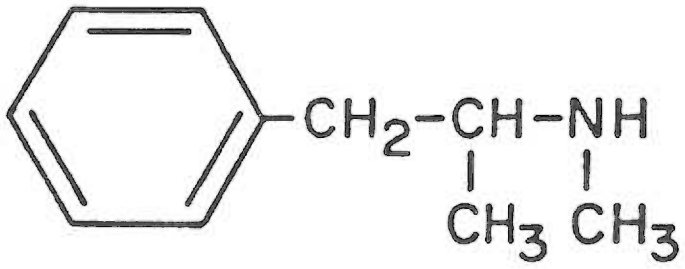
Past research has concentrated mostly on the biological mechanism(s) involved in amphetamine abuse rather than methamphetamine abuse. Recently, however, due to the fact that methamphetamine is far more often abused than amphetamine, research has targeted methamphetamine behavioral and biological effects. Since the two drugs are so similar in structure and action, it is reasonable to assume that conclusions drawn from studies of one drug can usually be generalized to the other drug (Figure 1).

Methamphetamine enhancement of central nervous system (CNS) catecholamine release has been suggested to account for some of the behavioral effects of the drug. At low doses amphetamines produce increased alertness, increased attention span, sympathetic arousal, psychomotor stimulation, and anorexia, while at high doses they produce stereotypy,

Figure 1. Schematic showing similarity in chemical structure of amphetamine and methamphetamine.



AMPHETAMINE (Methylphenethylamine)



METHAMPHETAMINE (Dimethylphenethylamine)

amphetamine psychosis, and convulsions (Seale, 1991).

Clinically, methamphetamine and related amphetamines have been used in the treatment of narcolepsy, attention deficit disorder and obesity (Tolstoi, 1989; Surgue, 1987). In addition, amphetamines are self-administered in non-human primates and rodents (Sannerud et al., 1989; Hoebel et al., 1989) and the release of dopamine by amphetamines has been proposed to play a key role in the reinforcing properties of this drug (Koob and Bloom, 1988). Peripherally, methamphetamine stimulates the cardiovascular system via catecholamine release (Lynch & House, 1992).

FEEDING BEHAVIOR CENTERS AND NEUROTRANSMITTERS

The hypothalamus is the area of the brain historically thought to control food intake. In the 1940's, lesioning of the ventromedial hypothalamus led to hyperphagia and weight gain, while damage to the lateral hypothalamus resulted in hypophagia and weight loss. From these earlier studies, the ventromedial and lateral hypothalamic nuclei were labeled as satiety and feeding centers. It is now known that this was an oversimplified categorization of a complex behavior system (Surgue, 1987). These earlier lesion studies not only destroyed nuclei, but also the neurotransmitter pathways that passed through them. Additional research has identified the important role of monoamines in feeding behavior.

Norepinephrine in the paraventricular nuclei of the hypothalamus (PVN) facilitates feeding, while serotonin in the PVN inhibits feeding. In the perifornical lateral hypothalamus (PFN) monamines including the amphetamines inhibit feeding (Hoebel et al., 1989).

DRUG-INDUCED ANOREXIA

In both humans and animal models, catecholaminergic drugs decrease food intake. This drug-induced anorexia has been extensively studied by pharmacological, microdialysis, and lesion studies. Investigators are interested in the ability of the drug to alter food intake, eating, feeding or ingestive behaviors. All of these terms are used interchangeably when discussing the effect of drug on feeding behavior. In this thesis, anorexia is defined as a reduction in food intake after drug administration, in other words drug-induced anorexia. This anorectic state is not the equivalent of the human condition of anorexia nervosa but is generally considered to be a reduction in food intake caused by a change in consumption or some aspect of feeding behavior.

THE EFFECT OF METHAMPHETAMINE ON FEEDING BEHAVIOR

Systemic injection of amphetamine decreases food intake in rats (Towell et al., 1988; Goodall, Trenchard & Silverstone, 1987; Surgue, 1987). Interestingly, at low doses of amphetamine (< 1.0 mg/kg) both inhibition and facilitation of feeding have been shown (Gilbert & Cooper, 1985; Sills & Vaccarino, 1991; Sills et al, 1993). Centrally, microinjections of amphetamine into the lateral hypothalamus (Leibowitz, 1975) or the nucleus accumbens produce anorexia in rats (Carr & White, 1986). In fact, amphetamines have been used as appetite suppressants in the treatment of obesity. However, amphetamine use is limited by its abuse liability, central stimulation and loss of efficacy due to the development of tolerance after repeated administration (Tolstoi, 1989). For these reasons, amphetamines are no longer used for this purpose in clinical practice.

Interestingly, the development of both tolerance and sensitization after repeated methamphetamine administration has been seen. Typically, tolerance to methamphetamine's anorectic effect develops after repeated low dose administration (Caul et al., 1988, Levitsky, Strupp & Lupoli, 1981). Sensitization to the locomotor activating effects and stereotypy is seen after low doses of methamphetamine, but more so after intermittent chronic treatment and repeated high doses (Eichler, Antelman & Black, 1980; Hooks et al., 1991).

It had been previously thought that the mechanism of the anorectic effect of amphetamine involved activation of the monoamines, dopamine, norepinephrine and serotonin (Leibowitz, 1975a; Leibowitz, 1975b; Hoebel et al., 1989; Gilbert & Cooper, 1985). Chemical lesion studies using 6-hydroxydopamine aimed at central catecholamine-containing neurons counteracted the anorectic effects of amphetamine (Samanin et al., 1975; Leibowitz, 1978). Systemic administration of DA agonists decreases food intake (Blundell & Latham, 1980). Additionally, central administration of amphetamine into the lateral hypothalamus produces anorexia in rats (Leibowitz, 1975). From numerous lesion and pharmacological studies, it has recently been proposed that amphetamine's anorectic effect is the result of an increase in DA, which in turn is responsible for the change in food consumption (Surgue, 1987 and Hoebel & Leibowitz, 1981). In addition, the involvement of the specific DA receptor subtypes, D1 and D2, has been investigated (Duterte-Boucher et al., 1990; Cooper et al., 1990; Gilbert & Cooper, 1985). D1 agonists decrease food consumption similar to amphetamine, while D2 antagonists have been shown to attenuate the effect of amphetamine on eating. Interestingly, depending on the paradigm used, D2 agonists may augment the anorectic effect of methamphetamine (Ujike et al., 1990; Martin-Iverson et al., 1988 and Rusk & Cooper, 1989). The exact role of these

dopamine receptor subtypes in feeding behavior is still unclear.

MICROSTRUCTURE FEEDING ANALYSIS

One reason why the underlying mechanisms involved in drug-induced anorexia have not been clearly established is the exclusive use of food consumption (g) as the only dependent measurement. Relying only on gross food consumption as the primary index of a drug's anorectic effect ignores the complexity of consummatory behavior and fails to detect the exact role a drug has on consumption. Studies of feeding behavior have shown consummatory behavior not to be continuous but to occur in discrete episodes (bouts) characterized by bouts of non-eating activities interspersed with bouts of eating. Non-eating activities include locomotor activity, rearing, grooming, oral behaviors other than eating, sniffing, and immobility (Blundell & Latham, 1980; Rusk & Cooper, 1989). Suppression of food consumption involves the alteration of this behavioral sequence. For example, drugs that reduce food consumption may do so by increasing the latency to eat, by prematurely ending a food consumption period (meal), or by producing competitive behaviors such as stereotypy (McGuirk et al., 1991; Blundell and Latham, 1980; Towell, Muscat & Wilner, 1988). Therefore, gross measurement of consumption fails to take into account a drug's effect on the behavior sequence of

consumption.

The investigation of the role of catecholaminergic and serotonergic drugs on food consumption prompted the need for a more comprehensive experimental design to study consummatory behavior (Blundell & Latham, 1980). This design, namely microstructure analysis, allows the investigation of not only gross food consumption (g), but a detailed analysis of the behavioral sequence of consumption. Microstructure analysis provides the opportunity not only to study eating behavior but those non-eating behaviors that, when altered, could explain the behavioral mechanism of anorectic drug action (Blundell & Barridge, 1979; Blundell & Latham, 1980). In a microstructure study, an animal's feeding sequence is recorded using video camera equipment. This allows the investigator to quantify all behaviors occurring during a test period, and to identify alterations in the internal structure of the feeding sequence. If desired, each microstructure behavior can be quantified by the duration and frequency (Rusk and Cooper, 1989). The microstructure design allows for the exhaustive investigation of the effect of a drug on consummatory behavior.

As previously mentioned, microstructure analysis has been used to differentiate the anorectic action of different drugs. Catecholaminergic drugs have been found to decrease both the duration of meals and feeding bouts within a meal, while increasing the rate of eating within bouts (Blundell and Latham, 1980; Towell et al., 1988). In contrast, serotonergic

drugs have been found to decrease consumption by reducing the rate of eating while not altering the behavioral structure of the meal (Blundell & Latham, 1980; Samanin & Garattini, 1993; McGuirk et al., 1991).

To further elucidate the catecholaminergic anorectic effect, the microstructure design has been used to investigate what role, if any, dopamine receptor subtypes D1 and D2 have in drug-induced anorexia. Microstructure analysis has proven invaluable in the investigation of the effect of amphetamine on feeding behavior due to the stimulant properties of these drugs. It has been suggested that the anorectic effect seen after amphetamine administration may not be related to its action on the feeding process but, instead, is a consequence of amphetamine's ability to increase locomotor activity and produce stereotypy (Blundell and Barridge, 1979; Gilbert and and Cooper, 1985). This is a serious consideration since these behaviors compete with the eating process, therefore, producing a decrease in consumption. The microstructure analysis provides the means to examine this hypothesis. One group of investigators has found a difference between the D1 agonist, SKF 38393, and the D2 agonist, N-0437, on microstructure behaviors. The D1 agonist reduced the number of feeding bouts and reduced eating rate while the D2 agonist had no effect on either frequency or duration of bouts, but did reduce eating rate. In addition, SKF 38393 did not produce changes in any of the non-eating behaviors measured,

while N-0437 was shown to increase and decrease a number of the non-eating behavioral measurements (Rusk and Cooper, 1989; Cooper et al., 1990).

GENETICS OF THE EFFECT OF AMPHETAMINE

As with other drugs of abuse, the behavioral and physiological effects of amphetamine vary among individuals. In both humans and animals, this is true for both the sympathomimetic and locomotor activating effects of the drug (deWit et al., 1986; Kitahama & Valatx, 1979; Seale et al., 1984 and Logan et al., 1989). Twin studies have shown that there is a strong genetic component determining sensitivity to amphetamine and amphetamine-like drugs (Nurnberger, et al., 1982 and Crabbe et al., 1983). No studies have looked at the importance of heredity in the anorectic effect of amphetamine. However, a mouse mutation, *anx*, that produces anorexia nervosa in infant mice has been identified and mapped to chromosome 2 (Maltais, Lane, & Beamer, 1984). While neither the *anx* gene or its protein product have been identified, it is interesting that the alpha-2 adrenergic receptor (*Adra-2b*) has recently been mapped to this same region of chromosome 2 (near *Il-1a*) (Link et al., 1993). It is possible that sensitivity to the anorectic effect of amphetamine may also have some genetic determination. In addition, a preliminary investigation of mouse ingestive behavior suggests a genetic influence on

consummatory behavior (Gannon et al, 1992; Gannon, 1995).

GENE MAPPING AND QTL ANALYSES

Recently, with the expansion of the mouse genetic map, a genetic tool useful for studying the actions of drugs of abuse has emerged: quantitative trait locus (QTL) mapping. With this new approach, genes underlying complex drug-induced responses can be mapped. Essentially, genetic mapping provides the assignment of chromosome locations for genes. It is hoped that identification of putative genes controlling a trait will give a better understanding of the mechanism(s) involved in drug-induced responses, either by confirming an existing hypothesis or providing new avenues of investigation.

Construction of a genetic map depends on two phenomena; linkage and crossing-over (Green, 1981; Belknap and Crabbe, 1992). Linkage is the tendency of two genes on the same chromosome to be inherited together. Conversely, genes on different chromosomes are unlinked and are inherited independently. Crossing-over or recombination is the mutual exchange of genetic material by homologous chromosomes during meiosis. Recombination is more likely to occur between two loci that are widely separated compared to loci located in close proximity.

In both humans and animals there is strong evidence for

a role of heredity in the variability of behavioral responses to drugs (Crabbe and Harris, 1991). Evidence from the animal literature, family studies, comparisons among inbred strains, and numerous selected lines provides substantial proof that there is a genetic influence on drug use and abuse (Plomin, DeFries and McClearn, 1990; Plomin, McClearn & Gora-Maslak, 1991). In the human literature, family, adoption, and twin studies provide strong evidence for a genetic influence on drug use and abuse, especially alcohol (Cadoret et al., 1986; Devor & Cloninger, 1989; Blum et al., 1990; Uhl et al., 1992).

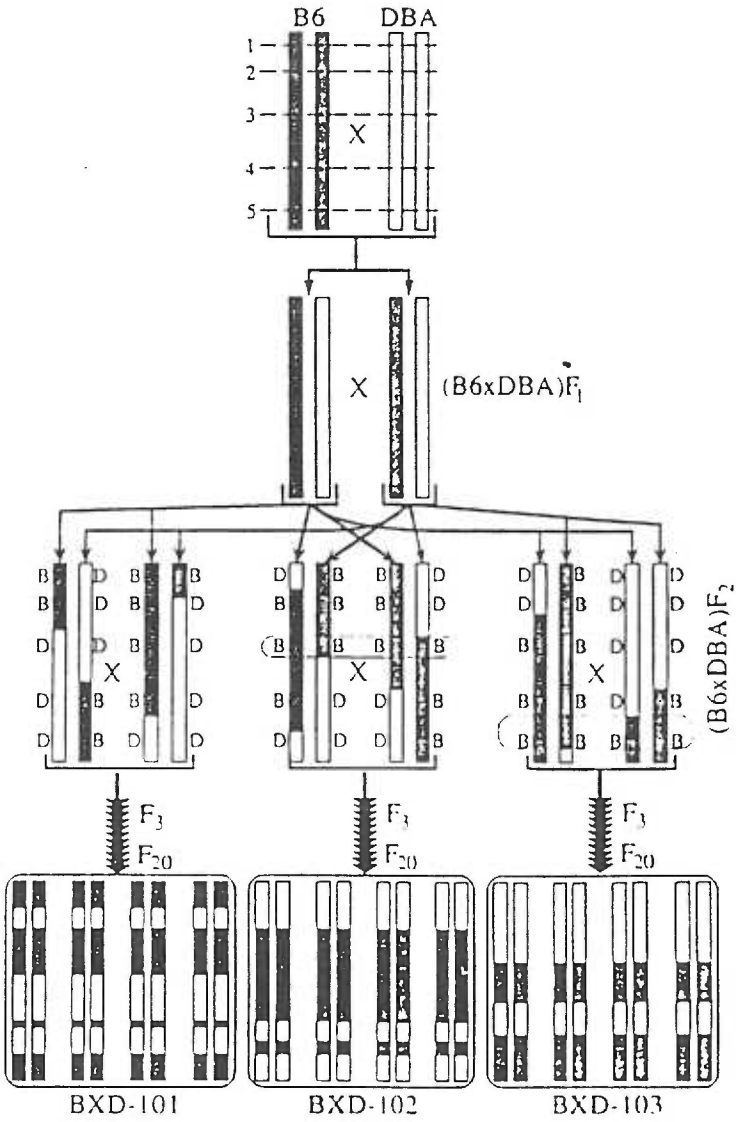
RECOMBINANT INBRED STRAINS

In animal research, a powerful animal model for studying the underlying genetics of complex, polygenic traits like drug abuse is recombinant inbred (RI) mouse strains. An RI series is derived from inbreeding (by separate brother by sister matings) of a genetically segregating F_2 generation derived from two progenitor (parental) strains. The most common set of recombinant inbred mouse strains used in behavioral research is the BXD RI series which was derived by Taylor (1978). In the case of the BXD RI series, the inbred strains C57BL/6J (B6) and DBA/2J (D2) were bred to produce a genetically uniform F_1 generation where animals were heterozygous at any locus at which the C57BL/6J and DBA/2J strains differed. The mice from the F_1 generation were mated

(intercrossed) to produce a genetically segregating F_2 generation. Originally, 32 recombinant inbred strains were derived from the F_2 generation by intermating brothers and sisters over 20 generations. Maximal inbreeding in the presently existing 26 BXD RI strains has been maintained for at least 70 generations, insuring that the genetic variance seen in the F_2 exists mainly between strains and not within strains. In other words, all individuals within a strain are genetically identical and they are homozygous at each locus. Since an average of 3-4 cross-overs per chromosome occurs during meiosis over 20 generations of full inbreeding, the RI strains represent chance recombination of the progenitor chromosomes in an inbred state (Bailey, 1981). See Figure 2 for a schematic of the BXD RI derivation.

The BXD RI series is most often used for genetic analysis because of the large number of strains available (26) and the extensive investigation of the progenitor strains, B6 and D2. A benefit of using the BXD RI series is the cumulative nature of the data. Any behavior characterized in the past in the BXD series can be correlated with other behavioral or physiological indices already collected from the BXD series. Furthermore, previously collected data on BXD strains can be analyzed for associations in the future with newly identified markers. The BXD series has been genotyped for over 1500 polymorphic genetic markers throughout the mouse genome on which the two progenitor strains differ

Figure 2. Schematic showing derivation of BXD RI strains from C57BL/6J (B6) and DBA/2J (D2) progenitors. The five B6 chromosome loci are shaded while D2 loci are not. In the F_2 generation, recombination is illustrated by the genotypes of the five loci. "B" signifies the B6 allele while "D" signifies the D2 allele. Animals are inbred through generation F_{20} to produce different homozygous strains. Taken from Silver (1995).


B6
DBA
1
2
3
4
5
X
X
(B6xDBA)F1
(B6xDBA)F2
F3
F20
BXD-101
BXD-102
BXD-103

genotypically, more than any other RI series. Typing of other markers is being aggressively pursued (Belknap et al., in press).

The BXD series was initially developed to determine major gene influences (Bailey, 1981; Taylor, 1978). If a single gene is responsible for a trait, and the parental strains differ, half of the RI strain means should resemble one parent and half resemble the other. This bimodal distribution suggests that the trait is controlled by a single gene locus with a major effect. In the traditional RI method, a comparison is made between the strain distribution pattern (SDP) for the trait (those RI strain means which resemble the B6 mean and those RI strain means which resemble the D2 mean) and the SDPs for previously mapped markers. If the two SDPs are closely matched, this suggests that the major gene locus controlling the trait is closely linked to the marker.

Most drug responses are continuous or quantitative and involve multiple-gene influences from several loci. These loci are termed quantitative trait loci or QTLs. Each QTL usually accounts for small amounts of the variance among individuals (Gora-Maslak, 1991; Belknap, 1992). Strong evidence for multiple-gene influences comes from genetic selection studies for various drug responses. If only one or two major genes are responsible for the etiology of a drug response, a distinct separation of the lines would be seen in the first few generations, with little further progress

thereafter. But, in many of the selection protocols, the lines diverge steadily over many generations. For example, the Long/Short Sleep (LS/SS) selected lines, which were selected for sensitivity to ethanol sleep time, diverged steadily over many generations before maximum separation of the lines occurred around the 10th generation (McClearn and Kakihana, 1973). Another example is the WSP/WSR mouse lines selected for differential expression of withdrawal severity after chronic ethanol exposure (Crabbe et al., 1983). The lines diverged after five selected generations and continued to steadily diverge until the 11th selected generation. The selected lines differ in seizure severity by approximately ten-fold. The estimated heritability in the WSP/WSR lines at generation X was approximately 26%. Genetic variance rarely accounts for more than half of the variance of the selected traits. This emphasizes the importance of nongenetic influences. Nongenetic or environmental influences such as nutrition, maternal fostering, housing conditions and experimental error account for the rest of the behavioral variability seen (Plomin et al., 1991).

The QTL method detects significant associations between a continuous trait and one or more previously mapped marker gene loci, suggesting linkage between a QTL affecting the continuous trait and a known marker locus. In the major gene approach, RI means must be bimodally distributed, while in the QTL approach, a continuous or unimodal distribution of RI

means can be handled. Another advantage of the RI QTL approach is that the progenitor strains do not have to differ on the trait of interest as is required in the single-gene approach. The QTL method calculates the correlation coefficient (r) between the BXD RI strain means for the trait of interest and a series of marker loci as an initial screen for candidate QTL (Gora-Maslak et al., 1991). This is accomplished by comparing the strain distribution of strain means for the trait of interest to the SDPs of the marker loci. A genotypic SDP is a list of which BXD RI strains possess the B6 allele and which possess the D2 allele. Each strain is scored as a 0 if the B6 allele is present, and a 1 if the D2 allele is present to facilitate statistical analysis. A significant correlation between a trait of interest and a marker locus suggests that a QTL affecting the trait of interest is located in the same chromosome region as the marker, i.e. the QTL and the marker locus are linked. The square of the correlation coefficient (r^2) gives the proportion of the genetic variance accounted for by a gene identified by a marker. The QTL approach is a powerful method to provisionally detect and map QTL sites.

MOLECULAR GENETICS

Gene mapping of the mouse genome was a slow and arduous process in the beginning of the 20th century (Silver, 1992;

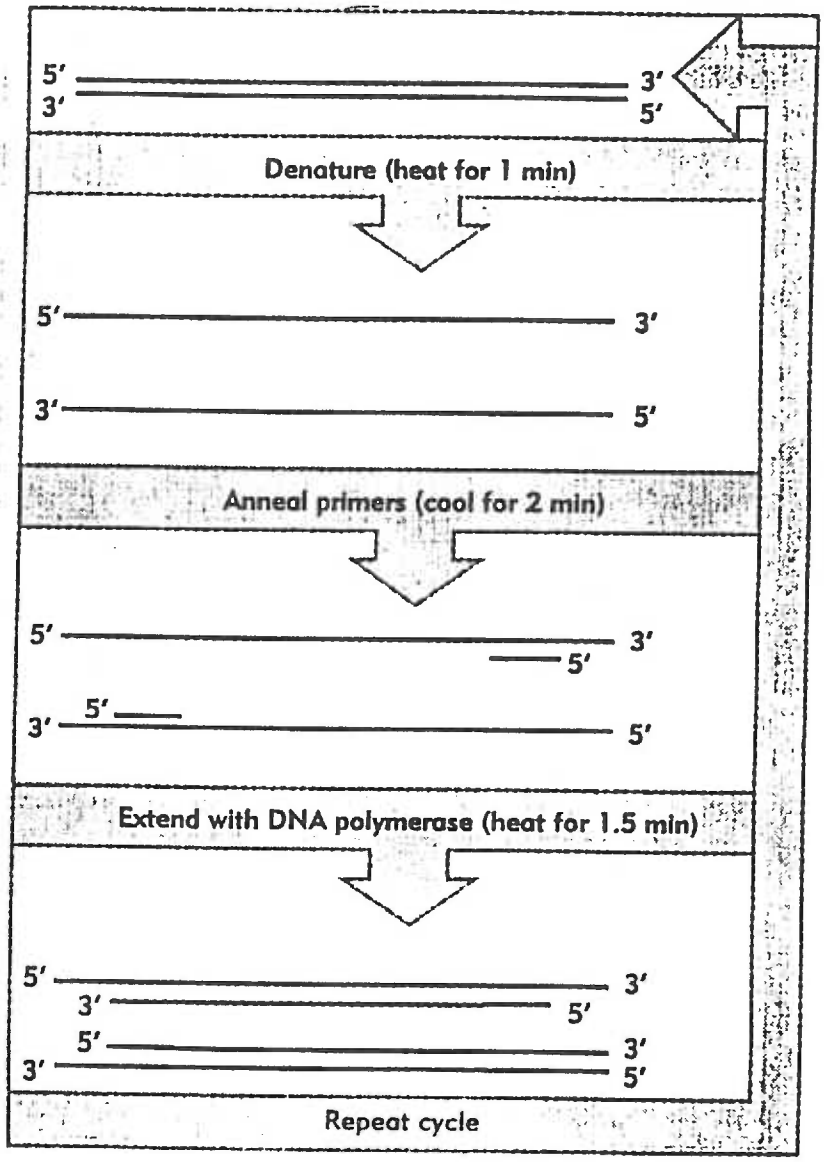
Dietrich et al., 1992). Mapping of simple Mendelian traits occurred one locus at a time, and mapping of quantitative traits was beyond the scope of available technology. The utilization of restriction fragment length polymorphisms (RFLPs) in the 1980's rapidly increased genetic mapping endeavors (Botstein et al., 1980). RFLPs are differences in DNA sequence made recognizable by restriction endonuclease digestion. They can arise from deletion or insertion of DNA or simple single nucleotide substitutions (Thompson et al., 1991). These DNA-based differences have been used to identify the position of hundreds of genes (Copeland et al., 1993). Even though application of RFLPs has allowed the genetic mapping of simple Mendelian and quantitative traits, there are several major limitations. First, typing RFLPs is time-consuming and labor-intensive. Second, identifying RFLPs within a cloned region is not a simple matter, and requires a large number of DNA probes. Finally, polymorphisms are often limited among inbred laboratory strains (Dietrich et al., 1992; Silver, 1992). An alternative source of DNA polymorphisms, microsatellites, was identified by Weber and May (1989). A microsatellite is a genomic DNA sequence that consists of nucleotide repeats in multiple tandem copies. Microsatellites or simple sequence repeats (SSRs) are highly polymorphic, present at high density throughout the mammalian genome and are relatively easy to identify and type. Typing of microsatellites by polymerase chain reaction (PCR) (Saiki

et al., 1985) allows results to be obtained rapidly with minimal investment. For example, Dietrich et al. (1992) typed 317 simple sequence polymorphisms (SSLPs) by using PCR with primers flanking the microsatellites. This makes microsatellites powerful tools for genetic mapping and they have even been called 'universal genetic mapping reagents' (Silver, 1992).

The utility of forward genetics, working from DNA to behavior, is dependent on the ability to obtain enough gene product to be analyzed. As previously mentioned, PCR analysis has allowed the typing of simple sequence polymorphisms. This powerful *in vitro* technique enables the amplification of microsatellites that vary in the number of repeated sequences. The PCR technique uses two oligonucleotide primers that complement each end of the targeted microsatellite marker locus. The primers are oriented such that DNA synthesis of the microsatellite sequence occurs. Amplification of the DNA segment occurs through repeated cycles of heat denaturation of the DNA, annealing of primers to complementary microsatellite sequences, and extension of annealed primers. A 10^5-fold increase in DNA can be seen after numerous cycles (4-25) (Lewin, 1994) (see Figure 3 for schematic of PCR).

The identification of microsatellites and the use of PCR analyses to type SSLPs has become a powerful tool of molecular geneticists to map genes that are involved in complex behaviors. This powerful technique has been used to identify

Figure 3. Polymerase chain reaction (PCR) schematic from Lewin, 1994. The schematic shows the three main steps in PCR.


5′
3′
3′
5′
Denature (heat for 1 min)
5′
3′
3′
5′
Anneal primers (cool for 2 min)
5′
3′
5′
5′
3′
5′
Extend with DNA polymerase (heat for 1.5 min)
5′
3′
3′
5′
5′
3′
3′
5′
Repeat cycle

genes involved in rat hypertension (Hilbert et al., 1991; Jacob et al., 1991), autoimmune type I diabetes (Todd et al., 1991), mouse epilepsy (Rise et al., 1991), emotionality (Flint el al., 1995), voluntary morphine drinking (Berrettini et al., 1994) and morphine-induced analgesia (Belknap et al., 1995). Recently, a gene involved in alcohol withdrawal seizures seen in mice has been identified (Buck et al., submitted).

MAPMAKER

Once genotyping has occurred the information gained from this procedure can be further analyzed using computer statistical analysis. MAPMAKER is a computer program that has been developed to construct genetic linkage maps and map genes underlying a complex trait under study (Lander etal., 1987). MAPMAKER is separated into two packages, MAPMAKER/EXP 3.0 and MAPMAKER/QTL 1.1. MAPMAPKER/EXP 3.0 is a linkage analysis package for constructing primary linkage maps of genotyping from backcross, F_2 and F_3 intercross and RI lines (Lincoln et al., 1988; Lincoln et al., 1992). In general, MAPMAKER/EXP 3.0 makes a linkage map and orders the markers used in genotyping. MAPMAKER/QTL 1.1 is a computer program used to compute logarithm of the odds (lod score) for putative QTLs in an F_2 and backcross population (Lincoln et al., 1992). MAPMAKER/QTL is used with selective genotyping. Selective genotyping is a procedure where only the extreme phenotypes of

a tested population are genotyped. It decreases the number of animals genotyped, therefore decreasing experimental cost and labor (Lander & Botstein, 1989). The MAPMAKER/QTL program is a maximum likelihood method that can be used with missing data, in other words a linear regression model with missing data. Therefore, as long as phenotypic data are recorded for all progeny, the genotypic value for the non-extreme genotyped progeny can be entered as missing data. MAPMAKER/QTL assumes a normal distribution of the phenotypic data.

By using both versions, MAPMAKER makes it possible to determine whether the genotypic data supports a QTL of significant value and where the QTL is in relation to the chromosomal markers used.

USE OF F_2 POPULATION

The RI QTL method is only a provisional mapping technique that is useful as an initial screen in mapping candidate QTLs underlying a trait (Belknap et al., 1996). This is true due to the enhanced number of false positives expected due to the multiple correlations. As the result of the many comparisons that are calculated in a BXD QTL analyses (N>1500), it is probable that with $p<0.05$ at least 5 out of every 100 correlations will be due to chance. With this in mind, the RI QTL approach is the first phase in the detection of the genetic determination of a trait. Confirmation of the BXD

data can be made by using other genetic models such as other RI series, standard inbred strains, congenic lines, and F_2 or backcross populations to independently support the candidate QTLs (Belknap, 1992; Plomin et al., 1991). One powerful confirmation tool is the molecular genetic classification of individual F_2 mice derived from crosses between the parental inbred strains. The animals are tested on the behavioral trait of interest and then genomic DNA is taken from the low and high responding mice. Using PCR, oligonucleotide primers that amplify the marker locus identified in the first stage are then tested in the F_2 population to confirm which putative QTLs are involved in the determination of the trait of interest. This dual phase strategy is a powerful method for detecting and mapping gene loci controlling a quantitative trait. For example, Belknap et al. (1995) were able to detect and map a QTL on Chromosome 10 with a strong influence on morphine-induced analgesia in BXD RIs and in a B6D2F2 cross. This study exemplifies the two-phase approach for gene mapping. Initially, the BXD strains were used to screen the genome to identify six candidate QTL sites. In the next phase, animals from the B6D2F2 cross were genotyped for the most promising QTL on chromosome 10. Additionally, the MAPMAKER program was used to construct a linkage map of the chromosome 10 markers and confirmed the presence of a QTL in the B6D2F2. This finding complemented current work concerning opioid research and a relatively small area on chromosome 10

as an important region in opioid action (Berretini et al, 1994).

As stated earlier, the advantage of using RI series in behavioral studies is the cumulative nature of the data. For instance, Seale et al (1985) investigated the hyperthermic effect of a 20 mg/kg i.p. dose of *d*-amphetamine in 10 BXD RI strains and their progenitors. Seven of the BXD RI strains and the D2 parent became markedly hyperthermic, while the other three BXD RI strains and the B6 parent showed a smaller hyperthermic response. This bimodal pattern of the RI strain means is highly suggestive of a single major gene effect. Six years later, Gora-Maslak et al (1991) subjected the data to QTL analysis using 173 marker loci, and significant correlations (associations) were found with the *Lamb-2* (r^2=0.92) locus, and the nearby *Ltw-4* locus on chromosome 1. Correlations at $p< 0.05$ were also found on chromosome 3, chromosome 6 and chromosome 17. In 1992, Belknap and Crabbe using a marker set with more than double the gene loci (360) redid the QTL analyses of amphetamine's hyperthermic effect. These investigators further supported the importance of the *Lamb-2* locus on chromosome 1. This locus accounted for 92% of the genetic variance which strongly suggests a major gene influence on amphetamine hyperthermia. This QTL analysis of an amphetamine-induced behavior is the only one published as yet. In addition, this major gene effect is quite unique since usually many minor gene loci underlie the genetic

multiple gene model of genetic determination and emphasizes the need for confirmation testing of putative QTLs identified from the BXD results.

SPECIFIC AIMS

The aim of this thesis is to identify the mechanisms mediating MA-induced anorexia using a behavioral genetic approach.

It is hypothesized that using a two-phase gene mapping strategy the neuroreceptor systems mediating MA-induced anorexia can be identified. Specifically, it is hypothesized that this study will provide further evidence that the dopamine receptor system plays a critical role in MA-induced anorexia.

PURSUIT OF SPECIFIC AIMS

The present work employed genetic and behavioral techniques to identify the mechanism(s) underlying the influence of methamphetamine on food consumption. The Experimental section of this thesis is divided into three experiments categorized into two parts.

In Part I, Experiment 1 and Experiment 2 identified the quantitative trait loci (QTLs) that influence the continuously distributed or quantitative trait methamphetamine-induced anorexia using a two-phase gene mapping strategy. The anorectic effect of methamphetamine on food consumption was tested using a panel of BXD RI mice. These data were subjected to a QTL analysis and the putative QTLs confirmed in an F_2 population. Finally, the linkage analysis programs,

MAPMAKER/EXP 3.0 and MAPMAKER/QTL 1.1, were used to construct a genetic linkage map and locate the gene(s) underlying MA's effect.

In Part II, Experiment 3 studied the effect of MA on consummatory behavior by using a microstructure design to provide a clearer picture of MA's effect on all behaviors involved in food consumption.

The thesis concludes with a general discussion of each experiment and discussion of the significance and implications of the findings herein.

PART 1. TWO-PHASE QUANTITATIVE TRAIT LOCI MAPPING STUDY

EXPERIMENT 1: IDENTIFICATION OF QTL MARKERS

RECOMBINANT INBRED FEEDING STUDY

METHODS

Male mice from 24 of the possible 26 BXD recombinant inbred mouse strains and their progenitor strains, B6 and D2, obtained from breeding colonies at the Veterans Affairs Medical Center, Portland, Oregon, (V.A.M.C.) were used in this study. Two BXD strains were unavailable at the time of the experiment. Animals were transported to another building for behavioral testing at least one week prior to the start of each experiment. All testing occurred in the animal's colony room. Mice, 55-115 days old at the beginning of each experiment, were weighed (baseline weight), individually housed in standard shoebox cages and maintained on a 12 h light-dark cycle (lights on at 0730 h). Tap water was available ad lib. At the time of individual housing and throughout the experiment, animals were maintained on a limited food access schedule, with powdered food available near light onset, approximately 0740 to 0810 h and from 1100 to 1530 h. Powdered food (rodent laboratory chow #5001, Ralston Purina Co.) contained in food cups was placed in cages and measured by weighing at the beginning and end of each

feeding period. Glass specimen jars were used as food cups. A 3/4" diameter circle was cut in the lid of each cup which allowed mice entry of only their head and upper paws into the cup, and restricted climbing into or knocking food out of the cup. Body weights were determined each morning at light onset (0730 h). If an animal lost more than 20% of baseline weight during the experiment, two to three hours of additional food access were provided from approximately 1530 to 1830 h until body weight loss was less than 20% of baseline weight or until termination of the experiment.

Over a 12-month period, 14 to 29 mice per strain were tested across 20 different experimental passes (N=417). Each experimental pass involved 16 to 30 mice from 24 BXD strains. The progenitor, B6 (n=12) and D2 (n=8) strains were tested across experimental passes 21-24. They were tested at this due to availability of mice and were tested with animals used in Experiment 3. Each experimental pass lasted nine days. On Baseline Day, animals were weighed for baseline weight and individually housed with water and powdered food ad lib. The next day, the powdered food was removed in the morning and mice were fasted for 18 h. On subsequent Days 1-5, animals were weighed and given limited access to food as described previously. Food consumption was measured after the 30 and 270 minutes access periods.

Animals were equally divided into two experimental groups. Group 1 received saline s.c. on Day 6 and 1 mg/kg

methamphetamine s.c. on Day 7, while Group 2 received 1 mg/kg methamphetamine s.c. on both Day 6 and Day 7. All injections were administered 15 minutes prior to the 30 minute food access period. Injections were made at this time to obtain the optimal drug effect. Drug was made fresh on Day 6 of each experimental pass. Food consumption was measured as on previous days. In addition, on Days 6 and 7, behavior during the morning food access period was monitored. Each animal was observed for 30 seconds beginning approximately 30 minute postinjection for different behaviors. The 8 microstructure behavioral categories as defined by Rusk and Cooper (1989) with modifications were: 1) *sitting*, stationary and not engaged in any other behavior; 2) *grooming*, washing, licking and/or scratching involving head, vibrissae or any body surface; 3) *eating*, mouse on food cup and actively obtaining or consuming powdered food; 4) *drinking*, actively obtaining water from water bottle spout; 5) *sniffing*, up-and-down or side-to-side head movements accompanied by vibrissae movement; 6) *rearing*, front limbs raised with head and body raised; 7) *climbing*, actively hanging from or moving on top of cage lid; 8) *line crossing*, locomotor activity assessed by entire body of mouse crossing a line between front and back of cage. Rearing, and line crossings were counted, while grooming, eating, drinking, climbing and sniffing were measured in seconds of behavior. Behaviors were assessed in experimental passes three through 20 (N=355). One observer quantified

behaviors in these passes. It was not until the third pass that it was deemed necessary to observe the animal's behavior. Therefore, behaviors were assessed only in the experimental passes three through 20 (N=355). Observation of progenitor strains were not collected in this experiment since they were tested in experimental passes 21-24 which included animals from Experiment 2.

DATA ANALYSIS

All statistical analyses and graphical representations were performed with the IBM DOS version of SYSTAT and SYGRAPH software packages. Drug Day was always an animal's first exposure to the drug. For Group 1, Drug Day was Day 7, while for Group 2 initial Drug Day was Day 6. AVGBASE was calculated by averaging 30 minute food consumption across Day 3, Day 4 and Day 5. Percent reduction in food intake in drug-treated mice (PCTRED) was calculated by subtracting the 30 minute food consumption (g) on Drug Day from the average baseline consumption (AVGBASE), and then dividing by AVGBASE. This number was then multiplied by 100 to create a percentage score. Also calculated on Drug Day were percent body weight loss, the 270 minute time point food consumption, and total food consumed. Baseline body weight was taken at the beginning of each experiment when animals were individually housed. Percent reduction in body weight (PCTWRED) was measured as the ratio of the difference of the baseline weight

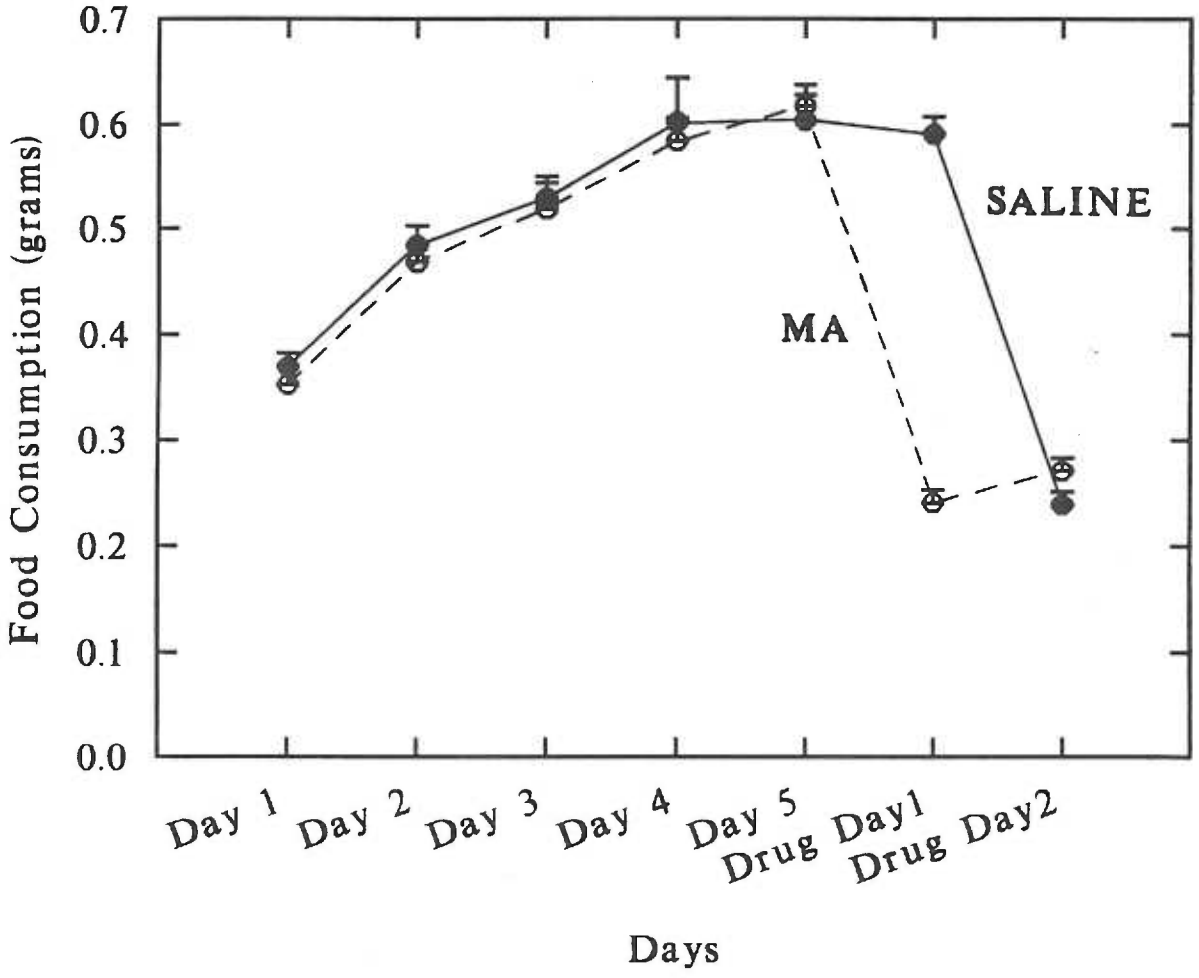
and Day 7 body weight to baseline body weight. This number was then multiplied by 100 to create a percentage score. Since body weight was measured at the beginning of each experimental day, Day 7 body weight would reflect any difference in body weight as a consequence of the previous days treatment. Total food consumed (TOT) was calculated as the total food in grams consumed per Drug Day. The afternoon food access period (270TOT) was defined as food consumed in grams on Drug Day. A two-way analysis of variance (ANOVA) (strain x treatment group) was run for each behavioral index. A one-way ANOVA (treatment group) for food consumption after the 30 minute period on Day 6 and Day 7 was run as well. In addition, a t-test was performed to determine if food consumption differed on Day 6 and Day 7 in Group 2. This provided a measure of tolerance or sensitization. Genetic correlations (Pearson product-moment) were calculated among all indices using strain means.

RESULTS

Figure 4 shows the daily food consumption for all animals during the 30 minute test period across Days 1-5. Food consumption on these baseline days steadily increased and appeared to be stabilized by Day 5, the last baseline day. In fact, Day 4 & 5 consumption was similar for all strains. No significant difference between strain and day was seen for these two baseline consumption days. AVGBASE, the average

Figure 4. Daily food consumption for Group 1 and Group 2 across experimental days. On Drug Day 1, animals received either saline s.c. (Group 1) or 1 mg/kg MA s.c. (Group 2) 15 minutes prior to the 30 minute food access period. On Drug Day 2 both Group 1 and Group 2 received 1 mg/kg MA s.c. 15 minutes prior to food access period. As shown, food consumption steadily increased and was stabilized by Day 5. Administration of MA reduced food intake in Group 1 on Drug Day 1 and in both Group 1 and Group 2 on Drug Day 2. There was no significant difference between Group 1 and Group 2 on Drug Day 2. Group 1 on Drug Day 1 did differ from Group 1 on Drug Day 2 suggesting a tolerance might have developed.

Consumption Across Days



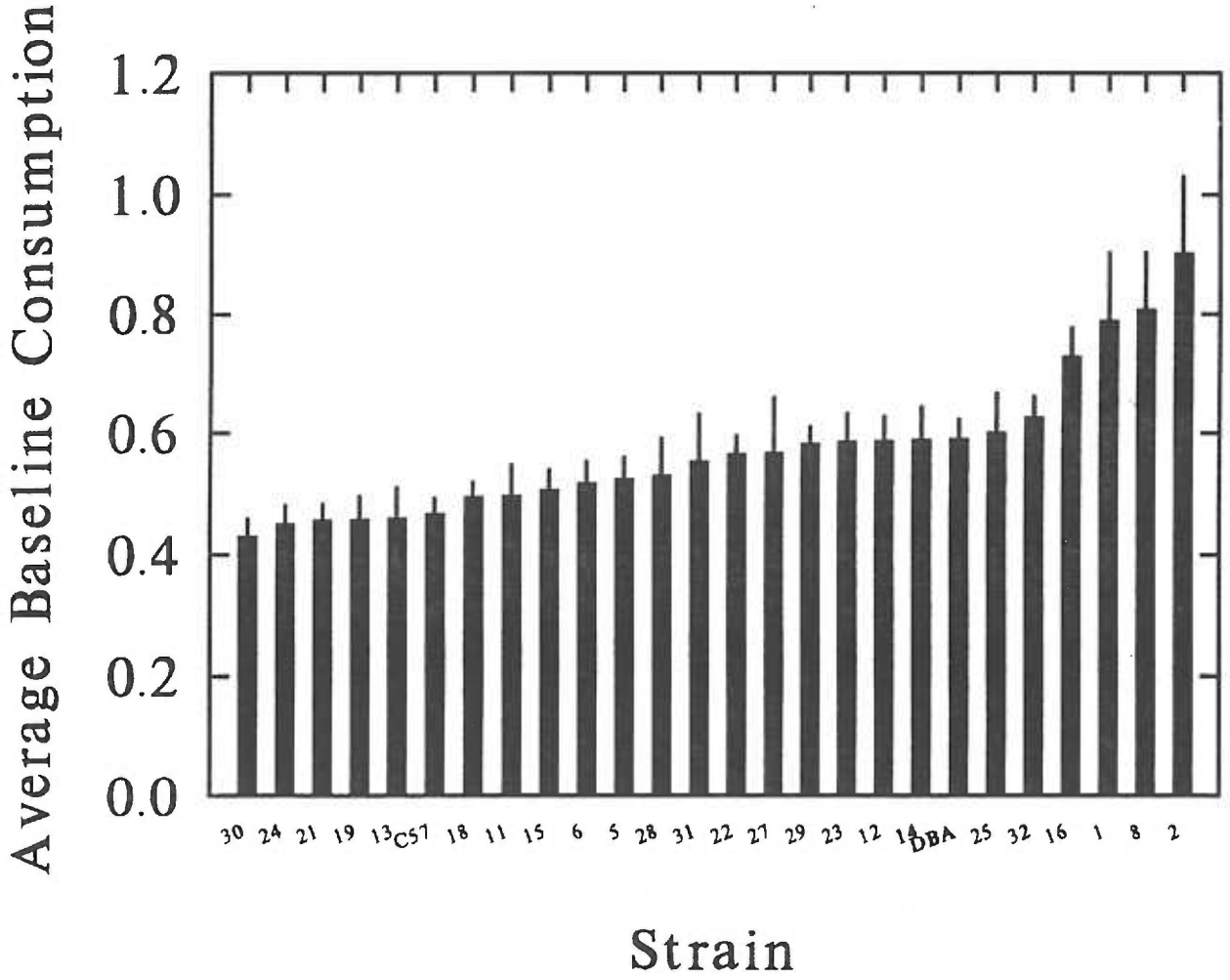
baseline consumption for Days 3,4 & 5 was used as the measure of pretreament food consumption. No significant difference between treatment groups or interaction on this measure was seen. However, there was a significant main effect of strain [(25,380)=3.907, p< 0.0001] for AVGBASE. The strain distribution for AVGBASE is shown in Figure 5. The coefficient of genetic determination (r^2) or heritability (h^2), which indexes the proportion of the total variability due to genetic sources for this dependent variable, was 0.229.

The effect of methamphetamine on food consumption in both Group 1 and Group 2 on both drug days collapsed across strains is shown in Figure 4. There was a significant effect of Group due to a decrease in food consumption in the MA-injected group (Group 2) as compared to saline-injected group (Group 1) on Day 6 [(1,427)=286.288, p< 0.0001). On Day 7, when both groups received MA, a difference in food consumption was seen between Group 1 and Group 2 [(1,430)=4.571, p=0.033] due to an increase in consumption in Group 2. There was no significant strain difference on Day 7. A t-test showed that Day 6 food consumption was significantly less than Day 7 food consumption in Group 2 [t=2.609, df=214, p=0.01] suggesting a possible development of tolerance after one prior exposure to MA.

The dependent variable used to assess drug effect was PCTRED (percent reduction in consumption). DRUG DAY was food consumed during the 30 minutes test period on Day 7 for Group

Figure 5. BXD strain means (±SEM) for average baseline consumption (g). Average baseline consumption (AVGBASE) was calculated by averaging 30 minute food consumption across Day 3, Day 4 and Day 5. As shown there was a significant strain distribution for AVGBASE.

BXD FEEDING STUDY



1 and Day 6 for Group 2. These days were the first exposure to drug for each group. A one-way ANOVA showed no significant difference between treatment groups for DRUG DAY, therefore, data from both groups were pooled into one DRUG DAY variable.

In addition, there was no significant strain by group difference for DRUG DAY. A two-way ANOVA (group x strain) showed a significant main effect of strain for PCTRED [(25,379)=4.807, p< 0.0001] but no significant main effect of group or interaction. Figure 6 shows the strain distribution for PCTRED. In the extreme BXD strains, food consumption was decreased as much as 86% (BXD1) or as little as 15% (BXD11). The coefficient of genetic determination for PCTRED was 0.285.

To assess the effect of the experimental design on body weight, and to see if body weight had an effect on drug sensitivity across strains, percent reduction in body weight (PCTWRED) was calculated. PCTWRED was calculated as the (Baseline Body Weight - Day 7 Body Weight/ Baseline Body Weight) expressed as a percentage. There was a significant strain effect [(25,380)=10.369, p< 0.0001] and group effect [(1,380)=13.245, p< 0.0001], but no interaction effect for PCTWRED. Group 1 had a mean PCTWRED of 16.252% (±S.E.M. 0.411) while Group 2 had a mean PCTWRED of 17.676% (±S.E.M 0.395). Three strains had a mean loss of more than 20% of baseline body weight (BXD13, BXD24 & BXD27) but appeared healthy and active, so their data were used in the calculations. The strain distribution for PCTWRED is shown in

Figure 6. BXD strain means (±SEM) for MA-induced anorexia expressed as PCTRED. PCTRED was calculated by subtracting the 30 min, food consumption on initial drug day from AVGBASE and then dividing by AVGBASE. Animals received 1 mg/kg 15 minutes prior to food access period. As shown, there was a significant strain distribution for PCTRED. In the extreme strains, food consumption was decreased by as much as 86% (BXD-1) or as little as 15% (BXD-11).

BXD FEEDING STUDY

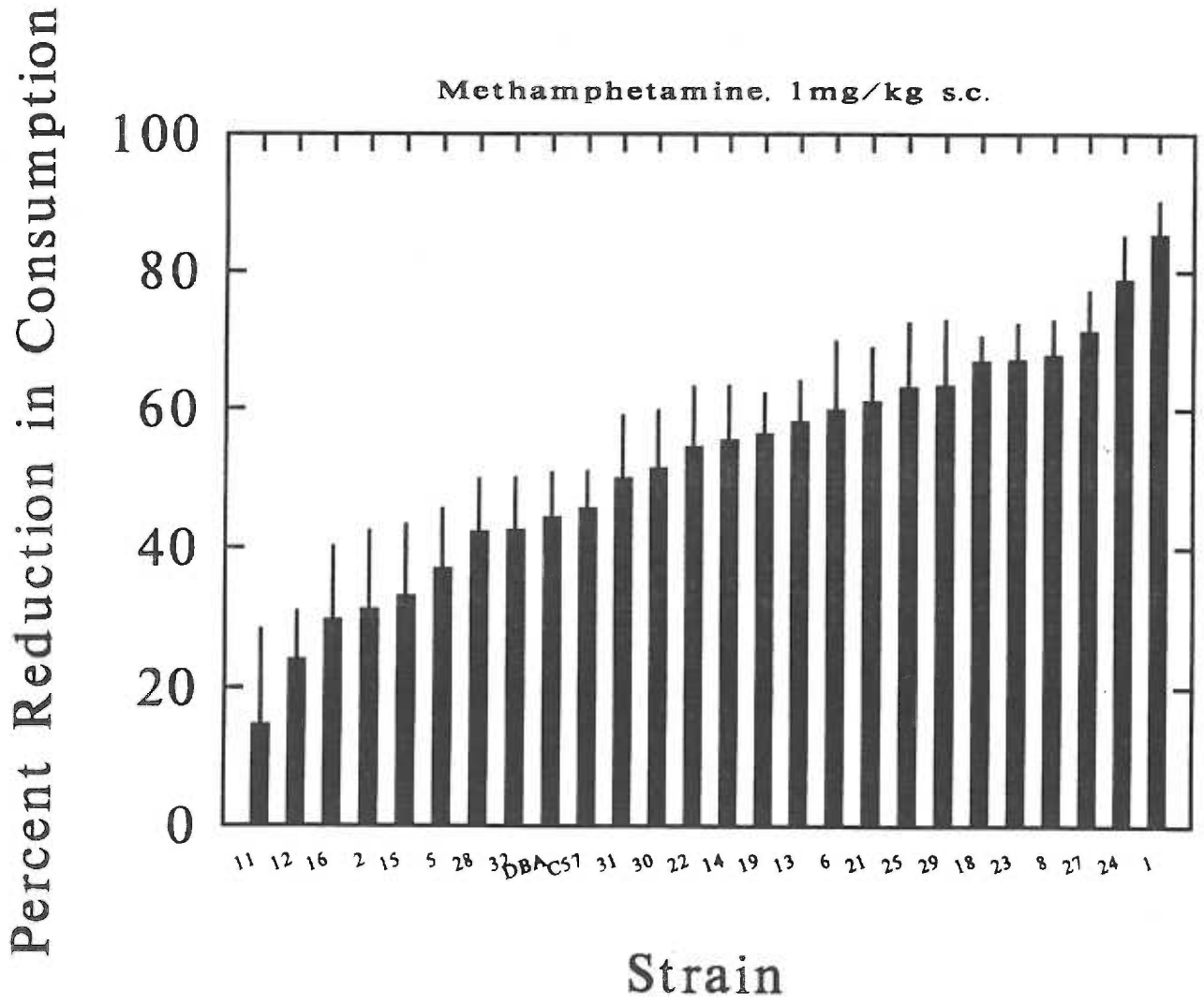
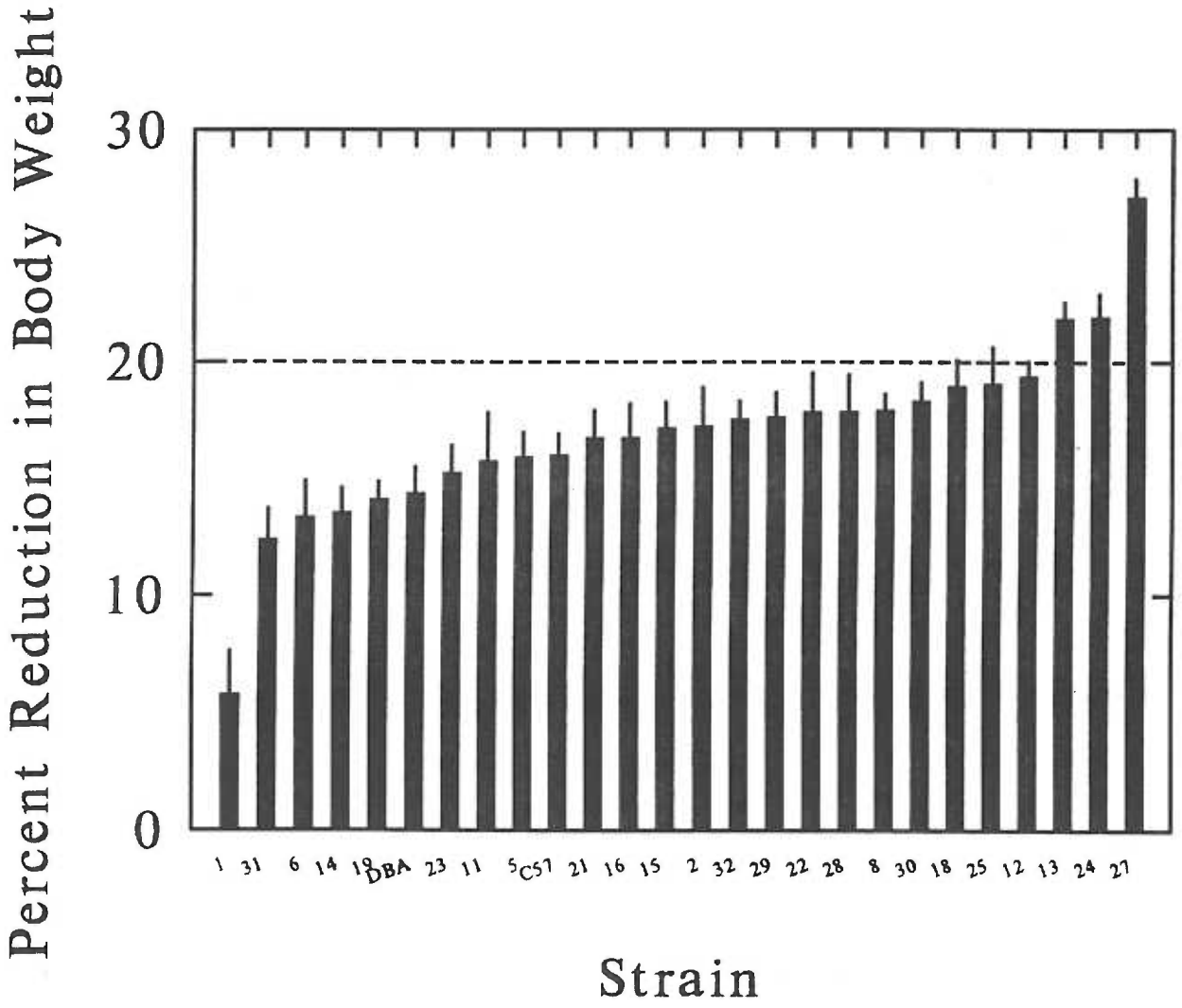


Figure 7. The coefficient of genetic determination for PCTWRED was 0.432.

To assess any rebound consumption effect, two other measures of food consumption were assessed, 270TOT and TOT. 270TOT was the index for food consumed (g) during the afternoon feeding period on the first drug day, while TOT was calculated as the total food consumed (g). A two-way ANOVA showed a significant main effect of strain [$(25,378) = 6.652$, $p < 0.0001$], but no group effect for 270TOT. Group 1 had a mean consumption of 2.602 g (±S.E.M. 0.060) while Group 2 had a mean consumption of 2.479 g (±S.E.M. 0.047) for the afternoon feeding period on their respective first drug days. There was also a significant strain effect [$(25,378)=6.497$, $p < 0.0001$], but no group effect for TOT. Group 1 had a mean consumption of 2.841 g (±S.E.M. 0.062) while Group 2 had a mean consumption of 2.724 g (±S.E.M. 0.049) for total food consumed on the first drug Day. Genetic correlations (Pearson's r correlations of strain means) for the different indices measured during the experiment are shown in Table 1. PCTWRED was negatively correlated with both TOT ($r=-0.786$, $p < 0.0001$) and 270TOT ($r=-0.818$, $p < 0.0001$). There was a significant correlation between TOT and 270TOT ($r=0.973$, $p < 0.0001$). Most importantly, however, there was no significant correlation of PCTRED with either PCTWRED or AVGBASE, indicating that this index of drug-induced anorexia

Figure 7. BXD strain means (±SEM) for percent reduction in body weight (PCTWRED). PCTWRED was calculated by subtracting Day 7 bodyweight from baseline bodyweight and dividing by baseline bodyweight. The dashed line indicates the threshold of 20% or more loss of initial body weight. As shown, there was a significant strain distribution for PCTWRED. Those strains losing more than 20% of initial body weight appeared healthy and active.

BXD FEEDING STUDY



	PCTRED	PCTWRED	TOT	270TOT	AVGBASE
PCTRED	1.00	-0.006	-0.270	-0.065	-0.021
PCTWRED		1.00	-0.786	-0.818	-0.253
TOT			1.00	0.973	0.283
270TOT				1.00	0.195
AVGBASE					1.00

Table 1. Genetic correlations (Pearson's r correlations of strain means) for different indices taken on initial drug day. PCTRED, percent reduction in feeding behavior after 1 mg/kg MA administration; PCTWRED, percent reduction in body weight at end of experiment; TOT, total food consumed on initial drug day; 270TOT, total food consumed during afternoon feeding period; AVGBASE, average consumption across Day 3, Day 4, and Day 5. Underlined correlations are significant at $p < 0.0001$.

RECOMBINANT INBRED OBSERVATIONAL STUDY

RESULTS

Two-way ANOVAs for strain and treatment group were obtained for each of the eight behavioral measurements. A significant increase in sniffing [(1,307)=10.220, p= 0.002], line crossing [(1,307)=33.615, p< 0.001], sitting [(1,307)=5.221, p= 0.023], and rearing [(1,307)=4.104, p= 0.046] was seen in the drug group (Group 2) as compared to the saline group (Group 1) on Day 6. There was a decrease in eating [(1,307)=23.729, p< 0.001] in the drug group as compared to the saline group. In addition, there was a significant strain effect for drinking [(23,307)=1.655, p= 0.032] and line crossing [(23,307)=1.906, p= 0.008] on this day. A comparison of both groups receiving MA on Day 7 showed no significant group effect, but did show a significant strain effect for eating [(23,305)=1.613, p< 0.039] and sitting [(23,305)=1.679, p<0.028]. No significant interaction was seen for any index on either day. Interrater reliability was not assessed for these data. Since only one observer assessed a 30 second observational period and data were not collected for all strains including progenitor strains, a more detailed microstructure study was done in Experiment 3.

QUANTITATIVE TRAIT LOCI ANALYSIS

METHODS

To map the QTLs involved in methamphetamine's effects on feeding behavior, the BXD RI strain means for PCTRED were correlated with the allelic distributions of 679 marker gene loci previously mapped to specific chromosome regions. At the time of the QTL analysis the data set was limited to a little over 600 markers, this number has now increased to over 1500 available markers. Markers were obtained from the marker data bank maintained by Dr. John K. Belknap and updated by Dr. Steve Mitchell. All markers have been shown to be polymorphic in the progenitor B6 and D2 strains. A strain distribution (SDP) of BXD RI strains was created for each marker locus. A SDP is a list of which BXD RI strains possess the B6 allele and which possess the D2 allele. Each strain was arbitrarily scored as a 0 if the B6 allele was present, and a 1 if the D2 allele was present for each marker. An example of the SDP for some of the markers on chromosome 13 is given in Figure 8. Correlation coefficients (r) were determined between each of the phenotypes, PCTRED, AVGBASE, PCTWRED and each marker locus. A significant positive correlation between a phenotype and a marker indicates an association between the D2-like allele at that locus and a high phenotypic value. Alternately, a significant negative correlation indicates an association of the B6-like allele with a high phenotypic

Figure 8. Strain distribution for Chromosome 13. Allelic distribution for the 26 BXD RI strains of some of the marker loci on chromosome 13. The number 0 represents the C57BL/6J allele while the number 1 represents the DBA/2J allele. Spaces represent unknown allele type. Change in number sequence represents a cross-over. Each numerical column represents a BXD RI strain.

CHROMOSOME	NO.	LOCUS	ALIAS	1	2	5	6	8	9	11	12	13	14	15	16	18	19	20	21	22	23	24	25	27	28	29	30	31	32
Thirteen	957	Hist1		1	0	0	0	0	0	0	1	1	1	0	0	1		0	0	0	1	0	1	1	0	1	0	1	1
Thirteen	958	D13Kyo1	Hh1tts	1	0	0	0	0	0	0	1	1	1	0	0	1	0	0		0	1	0	0	1	1	1	0	0	1
Thirteen	959	D13Byu1		0	1	0	1	0	1	0	1	1	1	0	1	1	0	1	1	0	0	1	1	0	1	0	1	1	0
Thirteen	960	Il9	D24, D13Mit13,I		1	0	1	0	1	0	1	1	1	0	1	0	0	1	1	0	0	1	1	1	1	0			
Thirteen	961	Ms6-1		0	1	0	1	0	1	0	1	1	1	0	1	0	0	1	1	0		1	1	1	1	0	1	1	1
Thirteen	962	Iapls1-13		0	1	0	1	0	1	0	1	1	1	0	1	0	0	1	1	0	0	1	1	1	1	0	1	1	1
Thirteen	963	D13Ncvs44	B417	0	1		1	0	1		1	1	0	0	1	0	0	1	1	0	0	0	1	1	1	0	1	0	
Thirteen	964	Tpmt		0	1	1	0	0	1	1	1	1	0	0	1	0	0		1	0	0	0	1	1	1	0	1	1	1
Thirteen	965	D13Ncvs45	B515	0	1	0	1	0	1	1	1	1	1	0	1	0	0	1	1	0	1	0	1	1	1	0	1	1	1
Thirteen	966	Pmv41	Pmv-41	0	1	0	1	0	1	1	1	1	1	0	1	0	0	1	1	0	1	1	1	1	1	0	1	1	1
Thirteen	967	Hmg1-rs3		0	1	0	1	0	1	1	1	1	1	0	1	0		1	1	0	1	1	1	1	1	0	1	1	
Thirteen	968	Hmg1-rs5		0	1	0	1	0	1	1	1	1	1	0	1	0	0	1		0	1		1	1	1	0			
Thirteen	969	D13Ncvs33	B8	0	1	0	1	0	1	1	1	1	1	0	1	0	0	1	1	0	1	1	1	1		0	1	1	1
Thirteen	970	D13Ncvs34	D23	0	1	0	1	0	1	1	1	1	1	0	1	0	0	1	1	0	1	1	1	1		0	1	1	1
Thirteen	971	Iapls1-1		1	1	0	1	0	1	1	1	1	1	0	1	0	0	1	1	0	1	1	1	1	1	0	1	1	1
Thirteen	972	D13Ncvs35	B211	0	1	0	1	0	1	1	1		1	1	1	0	0	1	1	0	1	1	1	1		0	1	0	1
Thirteen	973	Srd5a1	Srd5a-1	0	1	0	1	0	1	1	1	1	1	1	1	0	0	1	1	0	1	1	1	1	1	0	1	0	1
Thirteen	974	D13Byu2		0	1	0	1		1	1	1	1	1	1	1	0	0	1	1	0	1		1	1		0	1	0	1
Thirteen	975	D13Byu3		0	1	0	1	1	1	1	1	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	0	1	1
Thirteen	976	Xmv13	Xmv-13	1	1	0	1	0	1	1	1	1	1	1	1	0	0	1	1	0	1	1	1	1	1	0	1	0	1
Thirteen	977	D13Mit11	A91		1	0	1	0	1	1	1	1	1	1	1	0	0	1	1	0	1	1	0	1	0	0			
Thirteen	978	D13Ncvs46	B530	1	1	0	0	0	1	1	1			1	1	0	0	1	1	0	1	1	0	1	0	0	1	1	1
Thirteen	979	D13Mit9	M147		1	0	0	0	1	1	1	1	1	1	1	0	0	1	1	0	1	1	0	1	1	0			
Thirteen	980	Rasa		1	1	0	0	0	1	1	1	1	1	1	1	0	0	1		0	1	1	0	1	1	0	1	1	1
Thirteen	981	P198-13		1	1	0	0	0	1	1	1	1	1	1	1	0	0	1	1	0	1	1	0	1	1	0	1	1	1
Thirteen	982	As1	As-1	1	1	0	0	0	1	1	1	1	1	1	1	0	0	0	1	0	1	1	0	1	1	0			1
Thirteen	983	Lth1	Lth-1	1	1	0	0	0	1	1	1	1	1	1	1	0	0	1	1	0	1	1	0	1	1	0			1
Thirteen	984	D13Byu4		1	1	0	0	0	1	1	1	1	1	1	1	0	0	1	1	0	0	1	0	1	1	0	1	1	1
Thirteen	985	D13Mc1	D13McC1	1	1	0	0	0	1	1	1	1	1	1	1	0	0	1	1	0	1	1	0	1	1	0	1	1	1
Thirteen	986	D13McC2	KB3, D13McC2	1	1	0	0	0	1	1	1	1	1	1	1	0	0	1	1	0	1	1	0	1	1	0	1	1	1
Thirteen	987	D13Bir1		1	1	0	0	0	1	1	1	1	1	1	1	0	1	1	1	0	1	1	0	1	1	0	1	1	0
Thirteen	988	D13Ncvs9	B348	1	1	0	0	0	1	1	1	1	1	1	1	0	1	1	1	0	1	1	0	1		0	1	1	1
Thirteen	989	D13Ncvs36	D350	1	1	0	0	0	1	1	1	1	1	1	1	0	1	1	1	0	1	1	0	1		0	1	1	1
Thirteen	990	D13Ncvs37	D188	1	1	0	0	0	1	1	1	1	1	1	1	0	1	1	1	0	1	1	0	1		0	1	1	1
Thirteen	991	Iapls2-6		1	1	0	1	0	1	1	1	1	1	0	1	0	1	1	1	0	1	1	0	1	1	0	0	1	1
Thirteen	992	Pmv9	Pmv-9	1	1	0	1	0	1	1	1	1	1	0	1	0		1	1	1	1	1	0	1	1	0	0	1	1
Thirteen	993	Iapls3-14		1	1	0	1	0	1	1	1	1	1	0	1	0	1	1	1	1	1	1	0	1	1	0	0	1	1
Thirteen	994	D13Ncvs39	B213	1	0	0	1	0	1	1	1	0	1	0	1	0	1	1	1	1	1	1	0	1		0	0	1	1

value.

Since large numbers of multiple correlations can give rise to fortuitous findings, a more stringent criterion ($p < 0.01$) was adopted to identify QTL markers for pursuit in the $B6D2F_2$ study of the phenotype (Experiment 2).

In addition, multiple regression analysis of the candidate QTLs for the PCTRED phenotype (Experiment 1) was performed. Multiple regression looks at the impact of all the markers jointly and corrects for intercorrelations among the markers that could arise with a $p<0.01$, therefore, limiting the number of QTLs the data can support. However, multiple regression does not identify which QTLs are the "real ones" and which are false positives. Multiple regression also provides an estimate of the total amount of genetic variance accounted for by all the significant QTLs.

RESULTS

The three main behavioral indices PCTWRED, AVGBASE and PCTRED were subjected to QTL analyses to provisionally map polygenes influencing the specific phenotype. A summary of the QTL analysis results based on the BXD data for these traits is given in Table 2. A total of five candidate QTL sites emerged at the $p< 0.01$ level on five different chromosomes for the PCTWRED phenotype. Only one candidate QTL site emerged at the $p< 0.01$ level for the AVGBASE phenotype.

CANDIDATE QTLs FOR PCTRED ($p < 0.01$)

MARKER	CHROMOSOME	LOCATION(cM)	CORRELATION
Hcf-3	4	N	0.582
Fv2	9	61	0.558
D12Nyu1	12	25	0.597
Tpmt	13	27	-0.524
D19Byu1	19	4	0.593

CANDIDATE QTLs FOR PCTWRED ($p<0.01$)

MARKER	CHROMOSOME	LOCATION (cM)	CORRELATION
Pmv-24	1	74	0.521
Iapll-11	7	N	-0.564
D12Mit5	12	30	0.66
D14Mit7	14	28	-0.57
Pmv-42	15	62	0.626

CANDIDATE QTL FOR AVGBASE ($p<0.01$)

MARKER	CHROMOSOME	LOCATION (cM)	CORRELATION
D10Mc1	10	N	0.611

Table 2. The marker name and location as well as the correlation value is given for the three phenotypes measured, PCTRED, PCTWRED, and AVGBASE. Chromosomal locations are given in centimorgans (cM). When no chromosomal location is known the letter N is used. All correlations were at the $p \leq 0.01$ level.

The analysis of the PCTRED phenotype detected a total of five candidate QTL sites at the $p< 0.01$ level on five different chromosomes (Table 3). Positive correlations indicate that the possession of the D2 allele is associated with higher scores, while negative correlations indicate that the B6 allele is associated with higher scores (greater drug effect).

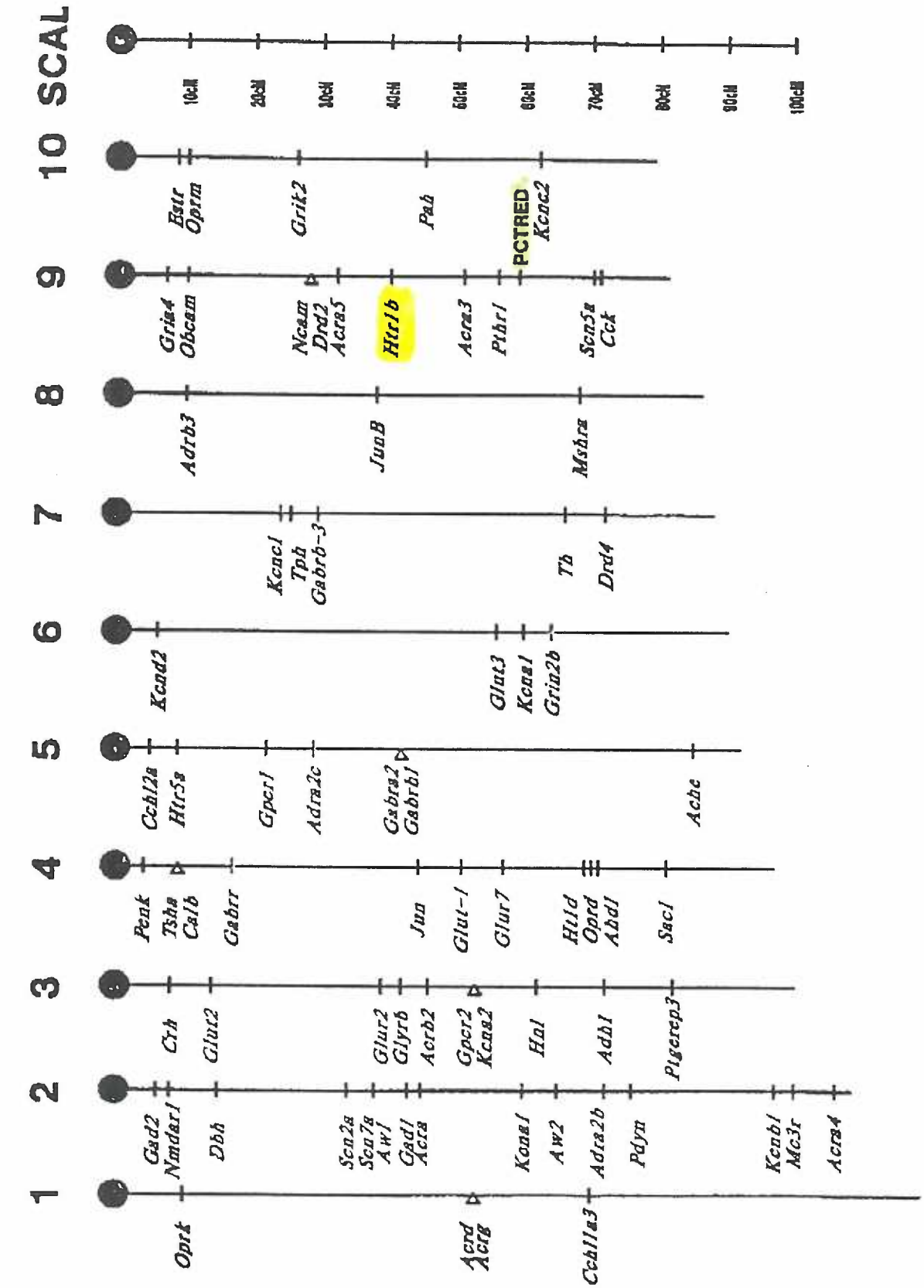
Neurochemically relevant candidate genes within 10 cM of the QTLs include the serotonin 1b receptor (*Htr1b*) on chromosome 9, the dopamine receptor subtype 1a (*Drd1a*) on chromosome 13, and the beta adrenergic receptor kinase-1 (*Bark-1*) on chromosome 19. Candidate genes are genes whose locations have been identified and could possibly influence the phenotype under investigation. Figure 9 shows the genomic locations of candidate genes for various neuroreceptors, encoding proteins, and provisional PCTRED QTLs. The raw data of the complete QTL analysis for all three phenotypes using the marker data bank are given in Appendix 1 (Dr. Steve Mitchell provided the formal QTL printout).

Multiple regression analysis of the five candidate QTLs indicated that the data only support the possibility of no more than two QTLs, not five, and that the amount of genetic variation accounted for by the candidate QTLs is 53.5%.

MARKER	CHROMO-SOME	cM	r (p<)	MIT MARKER	MARKER LOCATION (cM)	B6/D2 MARKER BP DIFF.
Hcf-3	4	N[a,b,c]	0.582 (0.004)	NONE	NONE	NONE
Fv2	9	61[a] 54[c] 55[b]	0.558 (0.007)	*D9Mit12* *D9Mit20*	55[a],49[b],52[c] 61[a],57[b,c]	5bp 8bp
D12Nyu1	12	20[b,c] 25[a]	0.597 (0.003)	*D12Mit3* *D12Mit2*	32[a],26[b,c] 21[a],18[b,c]	4bp 17bp
Tpmt	13	40[b,c] 27[a]	-0.524 (0.009)	*D13Mit11* *D13Mit3* *D13Mit13* *D13Mit23* *D13Mit21*	36.5[a],48[b,c] 11[a],19[b,c] 33[a],44[b,c] 44[b,c] 44[b,c]	11bp 37bp 6bp 8bp 4bp
D19Byu1	19	4[a] 3[c] N[b]	0.593 (0.002)	NONE	NONE	NONE

Table 3. The marker name, and location for the five putative QTLs for MA-induced anorexia are presented in this table. The *r* and level of significance for each QTL is given. In addition, the table provides the name of the *Mit* markers used in the confirmation process. QTL and marker locations on a chromosome are given in centimorgans (cM). When no chromosomal location is known the letter N is used. Base pair difference for the progenitor at a given marker are also given. At the time of this experiment, no *Mit* markers for *Hcf-3* and *D19Byu1* met the necessary criteria so as to be useful. Chromosomal locations were taken for a) Mammalian Genome, 1995, b) Mouse Genome, gbase 1994 and c) Mammalian Genome, 1994.

Figure 9 a and b. Schematic representation of genomic locations of candidate genes and provisional quantitative trait loci (QTLs) influencing the anorectic effect of methamphetamine. All 19 mouse chromosomes and the X chromosome are shown. The length of a chromosome is measured in centimorgans (cM). One cM represents 1% recombination distance. Candidate gene locations for neuroreceptors and encoding proteins are given by the latest Mammalian Genome and are in italics. Those neurochemical relevant candidate genes within 10 cM of the provisional QTLs are highlighted in yellow. **PCTRED** highlighted in green marks the known location of the QTLs for this study. (Figure provided by Dr. Steve Mitchell)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60cM
70cM
80cM
90cM
100cM
Oprk
Gad2
Nmdar1
Dbh
Scn2a
Scn7a
Awl
Gadl
Acra
Kcns1
Aw2
Adra2b
Pdyn
Kcnb1
Mc3r
Acra4
Acrd
Acrs
Ccbl1a3
Cth
Glut2
Glur2
Glyrb
Acrb2
Gper2
Kcns2
Hal
Adb1
Pigerep3
Penk
Tshs
Calb
Gabrr
Jun
Glut-1
Glur7
Htld
Oprd
Abd1
Sacl
Ccbl2a
Htr5a
Gpcr1
Adra2c
Gabra2
Gabrb1
Acbe
Kcnd2
Glut3
Kcns1
Grin2b
Kcnc1
Tph
Gabrb-3
Tb
Drd4
Adrb3
JunB
Msbra
Gria4
Obcam
Ncam
Drd2
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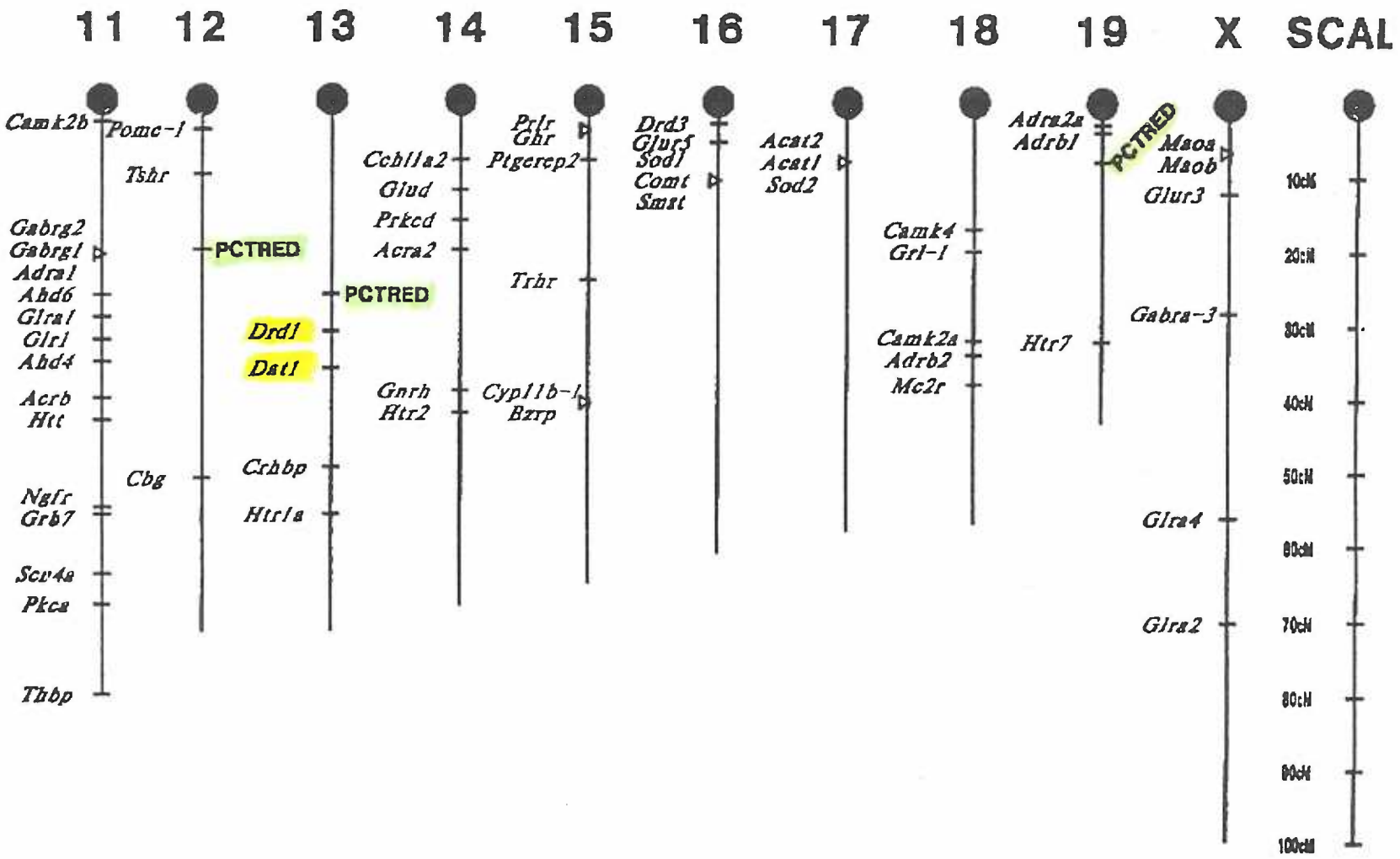
PCTRED
Kcnc2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Camk2b
Pomc-1
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Gabrg2
Gabrg1
Adra1
Ahd6
Glra1
Glr1
Ahd4
Acrb
Htt
Cbg
Ngfr
Grb7
Scv4a
Pkca
Thbp
PCTRED
Drd1
Dat1
Crhbp
Htr1a
PCTRED
Cchl1a2
Glud
Prkcd
Acra2
Gnrh
Htr2
Prlr
Ghr
Ptgerep2
Trhr
Cyp11b-1
Bzrp
Drd3
Glur5
Sod1
Comt
Smst
Acat2
Acat1
Sod2
Camk4
Grl-1
Camk2a
Adrb2
Mc2r
Adra2a
Adrb1
Htr7
PCTRED
Maoa
Maob
Glur3
Gabra-3
Glra4
Glra2
10cM
20cM
30cM
40cM
50cM
60cM
70cM
80cM
90cM
100cM

SUMMARY

This study showed methamphetamine dramatically reduced food consumption in recombinant inbred mice. No difference between the BXD progenitors, B6 and D2, was seen at this dose, excluding the possibility of identifying a single major gene effect. However, the strain distribution for reduction in food consumption after MA administration showed genetic variance in the BXD recombinant inbreds. This variance and the moderately low heritability of the phenotype, roughly 30%, suggest that a combination of genetic and environmental factors explains the phenotypic variance. QTL analysis identified five putative QTLs for MA anorectic effect, *Hcf-3*, *Fv2*, *D12Nyu1*, *Tpmt*, *D19Byu1*. Interestingly, three of the five QTLs were within 10 cM of candidate genes of dopamine (*Tpmt*), serotonin (*Fv2*) and adrenergic receptor systems (*D19Byu1*). However, multiple regression supports the possibility of only two significant QTLs.

Heritability for baseline feeding (AVGBASE) was low and only one significant QTL (*D10Mc1*) was detected. This finding suggests that this index has little genetic variation. Conversely, heritability of the phenotypic measurement of body weight (PCTWRED) was 43%, suggesting a strong genetic component to this index. QTL analysis identified 5 different QTLs on 5 different chromosomes, *Pmv-24*, *Iapl1-11*, *D12Mit5*, *D14Mit7*, *Pmv-42*. Since this was not the primary phenotype

of interest, further statistical analysis and confirmation of QTLs were not purusued. Reduction in food consumption after MA administration was independent of either weight loss or average baseline consumption. This was shown by the lack of significant correlation between PCTRED, PCTWRED and AVGBASE.

EXPERIMENT 2: CONFIRMATION OF QTL MARKERS

As mentioned earlier, gene mapping of a trait requires at least two phases: identification and confirmation of QTL markers. Once behavioral testing was completed and the location of QTLs that influence MA-induced anorexia (PCTRED) identified, confirmation of the QTL locations was attempted in a B6D2F_2 population. Confirmation of QTL markers identified for the other two phenotypes (PCTWRED and AVGBASE) was left for a later date due to a primary interest in the PCTRED phenotype.

Confirmation was done by using the polymerase chain reaction (PCR). Genotyping individual mice for simple sequence length polymorphisms (SSLPs) by PCR in a segregating F_2 population allows independent confirmation of the gene loci associated with MA-induced anorexia. QTL markers that met the $p<.01$ criterion in the BXD study (Experiment 1), and had adjacent MIT markers commercially available, dictated which marker loci to pursue in the F_2 studies (Experiment 2). In addition, coat color was used to confirm QTLs near known coat color loci.

F_2 FEEDING STUDY

METHODS

Male F_2 mice (n=109) and 20 progenitor strain mice were

tested on the feeding behavior protocol previously described. All mice were 60-100 days old at time of experiment. B6D2 F_1 breeding pairs were obtained from The Jackson Laboratory and bred in V.A.M.C breeding colonies to produce the F_2 intercross mice. All behavioral testing was done exactly as with the BXD RIs except that 65 F_2 mice and 9 progenitor strain mice were tested in one experimental pass. The other 44 F_2 mice and 11 progenitor mice were tested concurrently over five different experimental passes. In addition, coat color was recorded for each animal tested. At the end of a pass, all animals were sacrificed by cervical dislocation and spleens were removed for DNA isolation. PCTRED was calculated as previously described for each animal. The mice which displayed at least an 80% decrease in food consumption in the behavioral paradigm were considered high responders, whereas mice which displayed at most a 20% decrease in food consumption in the behavioral paradigm were considered low responders. The DNA from those animals displaying the highest (n=22) and lowest (n=29) sensitivity to MA-induced anorexia was then used for PCR analysis. In addition, DNA samples from the B6 and D2 mice tested at the same time as the F_2 mice were also analyzed. MIT microsatellite markers (Dietrich et al., 1992) were used to genotype individual mice. An MIT marker was chosen based on: 1) its proximity to a QTL marker significantly associated with the behavioral index in Experiment 1, 2) a large enough polymorphism between progenitor strains to be detectable using

agarose gels (ie., at least a 4 base-pair difference), and 3) commercial (Research Genetics, Inc.) availability of primers for each marker. The nearest two flanking markers were used for all the suspected QTLs pursued. The markers chosen for each QTL can be seen in Table 3. This genotyping method is similar to that used by Dietrich, et al. (1992) and Taylor and Reifsnyder (1993).

ISOLATION OF GENOMIC DNA FROM MOUSE SPLEEN

Each mouse was sacrificed by cervical dislocation before the spleen was removed. Susan Richards assisted in harvesting spleen DNA. Whole spleens were placed in centrifuge tubes containing 50 ml of a (10X) Hanks solution comprised of $NaHCO_3$ and double filtered deionized water (Milli-Q). To isolate DNA, spleens were strained through tea strainers and as much extract as possible was collected. The extract was placed in 50 ml tubes and centrifuged (1000 rpm) for 10 minutes. The supernatant was poured off and the remaining cell pellet was resuspended in 10 ml lysis buffer. Spleen DNA was kept on ice during all steps of processing. 40 ul of RNAse (DNAse free) was added to the pellet and the tube was incubated at 37°C in an oven for 1 hour while being gently rotated. 670 ul of 10% SDS and 785 ul of 8 mg/ml proteinase K solution were added to each tube, mixed gently and incubated in an oven at 37°C while being gently rotated overnight. The next morning, after being

allowed to equilibrate to room temperature, 3.33 ml of saturated NaCl was added to each sample and spun twice (3750 rpm) for 20 minutes. Between spins, the supernatant was transferred to a 50 ml tube and the pellet discarded. To precipitate the DNA, 2 volumes of 100% EtOH was added to each tube. The tubes were gently rocked by hand until a large, white DNA precipitate could be collected on a sterile glass pipette wand. Each DNA sample was placed in a 15 ml tube and washed twice with 70% EtOH, air dried and deposited in a 10 ml tube containing TE' buffer. TE' buffer was prepared by combining 0.88 g of Tris HCL, 0.53 g Tris Base and 37.3 mg of Na_2EDTA and dissolving in 1 liter of Milli-Q. DNA samples were refrigerated until future use in PCR reactions.

GENOTYPING USING MIT MICROSATELLITE MARKERS

Oligonucleotide primer pairs specific to each microsatellite marker locus were obtained from Research Genetics, Inc. PCR was used to amplify microsatellites. A standard mixture containing spleen DNA, primer pairs, thermostable thermus flavus (*Tfl*) DNA polymerase and deoxynucleotide triphosphates (dNTPs) was placed in a thermal cycler (P-E 9600 or MJ Research PTC-100) at 94°C for 3 minutes for initial DNA denaturation. This was followed by 40 cycles of 94°C for 1 minute, 58°C for 2 minutes and 72°C for 3 minutes, ending with 7 minutes at 72°C. PCR reaction products

were maintained at 4°C until bromphenol blue was added to each sample for visualization purposes. PCR samples were then frozen until further use. Frozen PCR samples were allowed to thaw before loading into an agarose gel.

A PCR reaction was carried out for nine MIT markers across all 29 high and 22 low responders, and progenitor strain DNA samples. Six markers were tested consecutively over a 10-day period. Samples of the 123 bp DNA ladder (Sigma), the PCR product mixture of each progenitor, and each F2 mouse were placed in wells of an ethidium bromide-stained agarose gel (4% FMC NuSieve). Electrophoresis was carried out for 5 minutes at 100 V followed by 2-4 hr at 70 V. DNA samples were separated by molecular weight. On completion of electrophoresis, gels were transferred to UV viewing boxes. The B6 and D2 band locations were identified and compared to the 123 BP DNA ladder. F2 bands were identified as either B6- or D2-like, or containing both B6 and D2 bands (heterozygous). Two separate investigators identified bands and compared results. Any discrepancy was reread by both investigators. Band identification was agreed upon or categorized as noninformative.

At a later date, a new MIT marker closer in proximity to the provisional QTL identified on Chromosome 13 became available for testing. PCR reactions were carried out exactly as before, except a new agarose gel recently available was used (3% Metaphor). Metaphor is a higher resolution gel than

NuSieve in that it allows detection of genomic bands which differ by as few as 6 base pairs (bp) out of a 200 bp total length. As before, F2 bands were identified as either B6-, or D2-like, or containing both B6 and D2 bands by two different investigators.

DATA ANALYSIS

All statistical analyses and graphical representations were performed with the IBM DOS version of SYSTAT and SYGRAPH software packages. In the F_2 data, since only the highest and lowest scoring mice were genotyped, Chi square was calculated for the high and low groups and the genotypic data generated from the PCR procedure (2x3 table).

As defined by Fisher (1958) and explicated by Sokal and Rohlf (1981), the *p*-values for a given marker or markers can be combined to determine the joint probability of two or more experiments that are testing the same hypothesis. Fisher's method utilizes the fact that *p*-values are distributed as chi square with two degrees of freedom per *p*-value. The equation for combining *p*-values from *t* experiments is: $-2\Sigma \ln p = \chi^2$, with $df=2t$. Therefore, significant *p*-values from the RI and F^2 data were combined to generate a joint probability.

The linkage analysis package, MAPMAKER, comprised of MAPMAKER/EXP 3.0 and MAPMAKER/QTL 1.1, was used to construct a genetic linkage map and map the gene(s) underlying PCTRED,

respectively. MAPMAKER/EXP 3.0 is a linkage analysis program that constructs linkage maps of markers in segregating experimental crosses (Lincoln, et al., 1988; Lincoln et al., 1992). This program was used to create a linkage map for the four chromosome 13 microsatellite markers (*D13Mit3*, *D13Mit13*, *D13Mit21* and *D13Mit23*). MAPMAKER/QTL 1.1 maps quantitative trait loci controlling a phenotype with respect to the linkage map. It extends the traditional gene dosage/phenotype correlation to calculate lod scores for putative QTLs, and assess both additive and dominance effects of a QTL. In addition, it has a built-in genotyping error check routine and uses interval mapping, which confers greater power to map QTLs that are between markers compared to simple linear statistics, e.g. r (Lincoln et al., 1992).

Both the dilute (d) and brown coat (b) color loci were used for the confirmation of candidate QTLs identified on chromosome 4 (*Hf3-3*) and chromosome 9 (*Fv2*), respectively. Recording of coat color allowed each animal to be identified as either dilute (dd) or nondilute (D-) and black (B-) or brown (bb). A one-way ANOVA grouped on coat color was run for the dependent variable PCTRED using the entire sample (n=109).

RESULTS

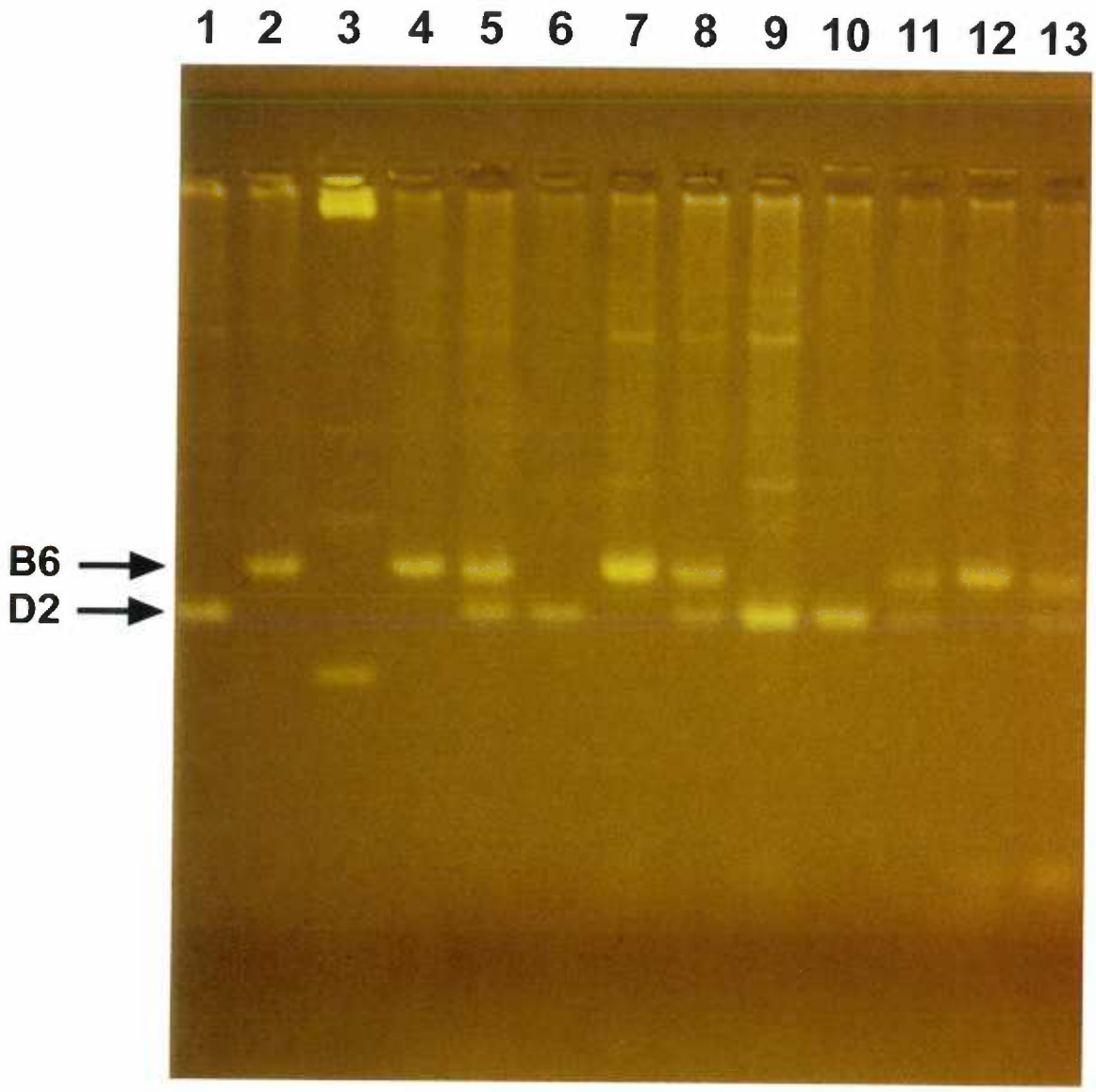
Of the 109 F_2 mice that were tested, 22 mice were classified as high responders and 29 were classified as low responders according to the criterion previously described in

the methods section. These 51 mice were genotyped for nine MIT microsatellite markers listed in Table 3. Figure 10 shows an example of genotyped D2, B6, and F_2 DNA for the *D13Mit3* microsatellite maker by PCR.

Chi squares were calculated for each of the MIT markers. A significant chi square for the *D12Mit2* marker (χ^2 = 4.188, p< 0.041) was found, but the genotyping data were in the opposite direction relative to the BXD RI data, and thus represents a disconfirmation. In other words, in the BXD RI study the correlation coefficient was positive (the DBA/2J allele conferred higher anorexia scores), while in the F_2 data the correlation coefficient was negative (the C57BL/6J allele conferred higher anorexia scores). Confirmation of the chromosome 13 QTL (*Tpmt*) was indicated by a significant chi square (χ^2= 11.004, p<0.004) and same direction (-r) (C57BL/6J allele conferred higher anorexia scores) for the *D13Mit13* marker. No other significant chi squares for the other MIT markers tested were found.

As was the case with the BXD data, the B6 allele was associated with the greater anorectic effect of methamphetamine. F2 percent reduction data (PCTRED) plotted as a function of genotype at marker locus *D13Mit13* is shown in Figure 11. The association between *D13Mit13* and PCTRED accounted for 16% of the phenotypic variance (r^2) in the F_2 population.

Figure 10. Ethidium bromide stained agarose gel of genotypic data for the *D13Mit3* microsatellite marker by PCR. Lane 1 shows the migration of DNA from DBA/2J mice, Lane 2 shows migration of DNA from C57BL/6J mice, Lane 3 shows the standard 123 bp Ladder migration and Lanes 4-13 show the migration of DNA from F_2 mice. Lanes 4, 7, & 12 were homozygous for the C57BL/6J allele, Lanes 6, 9, & 10 were homozygous for the DBA/2J allele while, Lanes 5, 8, 11 & 13 were heterozygous for both the DBA/2J and C57BL/6J allele.

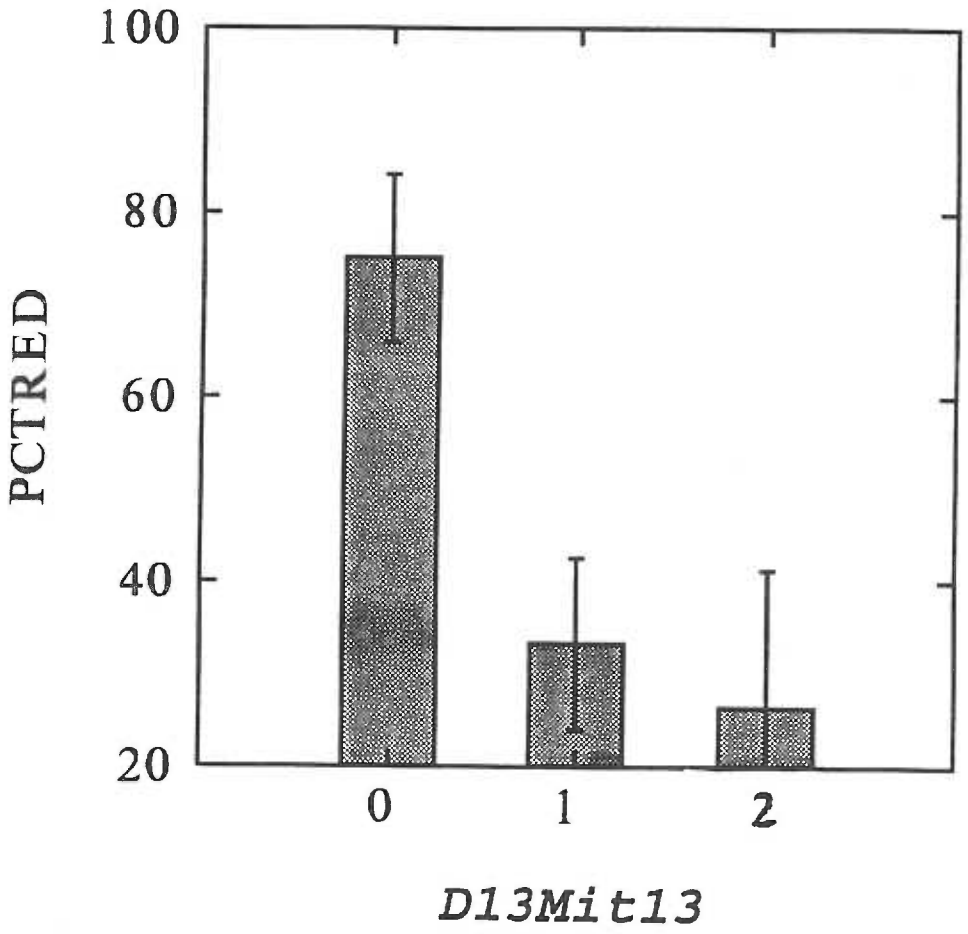

D13Mit3
1 2 3 4 5 6 7 8 9 10 11 12 13
B6
D2

The MAPMAKER/QTL analysis of the F_2 data revealed a maximum LOD score of additive and dominance effects of thechromosome 13 QTL to be 2.86 ($p< 0.0014$, df=2) for PCTRED using a one-tailed test. Since the direction of effect is known from the BXD data (B6 allele confers higher anorexia scores) a one-tailed test was appropriate. MAPMAKER identified the location of the QTL to be between the *D13Mit23* and *D13Mit13* markers and accounts for 20.5% of the phenotypic variance. In addition, MAPMAKER/QTL indicated a dominance effect of the D2 allele for this QTL. This is illustrated by the data presented in Figure 11. The built-in error check routine threw-out the *D13Mit21* marker data because of too many suspected genotyping errors. The consensus map distances as defined by Mouse Genome (1995) were 19 cM for *D13Mit3*, 44 cM for *D13Mit13* and 44 cM for *D13Mit23*. The QTL, *Tpmt*, was designated to be at 40 cM. Interestingly, the dopamine D1 receptor subtype, *Drd1a*, was designated to be at 39 cM.

To increase statistical power, *p* values from the BXD RI study and the F_2 study were combined using Fisher's joint probability. For PCTRED, the combined *p* value was 0.00016 (LOD=3.10, df=1) for the additive effect of the QTL using *D13Mit13* for both the BXD and F_2 data. These data fall short of statistical significance (lod scores >3.5) based on the recommendations of Lander and Schork (1994). However, these data are highly suggestive that there probably is a QTL affecting PCTRED in this chromosome region.

Figure 11. F_2 anorexia scores expressed as PCTRED (±SEM) plotted as a function of genotype at marker locus *D13Mit13*. The number 0,1, and 2 are the number of DBA/2J alleles at this marker. As illustrated, there was a dominance effect of the D2 allele for this QTL. Those animals homozygous for the B6 allele conferred higher anorexia scores.

Gene Dosage for *D13Mit13*



Because of their close proximity to suspected QTLs and the ease in using coat color identification, both the dilute (dd, d-) and brown (bb, B-) coat color loci were used to confirm the candidate QTLs identified on chromosome 4 (*Hf3-3*) and chromosome 9 (*Fv2*), respectively. Neither correlation of PCTRED and coat color loci or one-way ANOVA on coat color were significant for the dilute locus on chromosome 4 or the brown locus on chromosome 9. In addition, the two-way ANOVA for treatment group and coat color was not significant.

SUMMARY

In this study, as in Experiment 1, the B6 allele was associated with the greater anorectic effect of methamphetamine. Chi square analysis of the genotypic data indicated the significance of the chromosome 13 (*Tpmt*) QTL.

MAPMAKER/QTL analysis of the F_2 data revealed a maximum LOD score of additive and dominance effects of the chromosome 13 QTL to be 2.86 (p, 0.0014, df=2) for MA-induced anorexia effect. Furthermore, using Fisher's joint probability the combined p value of the BXD RI and F_2 study was 0.00016 with a LOD score of 3.1 (df=1). This falls short of Lander and Schork's (1995) standard of significance, LOD= 3.5.

Nevertheless, the confirmation process of the F_2 data indicated *Tpmt* on chromosome 13 is a probable QTL, in that it is much more likely to be a true positive than a false positive. According to Lander and Kruglyak (1995), this QTL would be described as suggestive, since it is within 1.0 lod of the significance threshold.

PART II. MICROSTRUCTURE ANALYSIS

EXPERIMENT 3: FEEDING AND BEHAVIORAL STUDY

METHODS

To study the microstructure of MA's effect on feeding behavior, male mice from six BXD recombinant inbred mouse strains (BXD-1, BXD-11, BXD-12, BXD-13, BXD-21 and BXD-27) and their progenitor strains, B6 and D2, were used in this study. Mice were obtained from a breeding colony at the Veterans Affairs Medical Center, Portland, Oregon. These strains were chosen because of their sensitivity (high, average, or low) to MA on food consumption in the Experiment 1 RI feeding study. High responding strains, BXD-1 & BXD-27 showed the greatest decrease in food consumption; average strains, BXD-13 & BXD-21, were in the middle of the strain distribution; and the low responding strains, BXD-11 & BXD-12, showed minimal reduction in food consumption (see Figure 2). Animals were transported to another building for behavioral testing at least one week before the start of each experiment. Mice, 60-100 days old at the beginning of each experimental pass, were weighed (baseline weight), individually housed in standard shoebox cages and maintained on a 12h light-dark cycle (lights on at 0730 h) with tap water available ad lib. Behavioral testing occurred as previously described in both Experiment 1 and Experiment 2 feeding studies except there was only one drug

day (Day 6). Day 7 was not included in this study. On Day 6, half the animals per strain received saline s.c. (Group 1), while the other half received 1 mg/kg MA, s.c. (Group 2). In addition, animals were videotaped. To videotape behaviors, home cages were moved to procedure tables where animals were normally weighed and injected. As before, each animal received a s.c. injection 15 minutes prior to food access. Food consumption was measured after the 30 minute food access period. Behaviors were videotaped using both an 8-mm Sony videocamera and a Burle videosystem during this 30 minute test period. Each camera was placed in front and to the left of a test cage. This allowed the best view of the animal's behavior in the test cage. Data were stored on videotapes for later viewing and quantification. Two raters (Sandra Angeli-Gade and Susan Richards) independently viewed the videotapes, quantifying rearing and line crossing as independent measures. A line crossing constituted crossing from the front of the cage to the back of the cage or vice versa. An imaginary line at the food bin separated the cage into front and back. In addition, the behaviors eating, sitting, drinking, sniffing, climbing, grooming and a general behavior category for behavior not fitting any other previous category were measured in seconds. Raters watched the videotapes and recorded a behavior for each second of the 30 minutes test period for every animal tested. This was possible because the video apparatus was equipped with internal clocks that displayed the

time throughout a test session. The microstructure data were organized by a computer program designed by Dr. Steve Mitchell. The program allowed for statistical analysis of the data. The microstructure behaviors were previously described in Experiment 1. However, a brief description is given in Table 4 showing each behavioral category and the criteria used to classify a behavior. Since animals had to be taped individually, only four animals per experimental pass were tested during one morning three-hour session. The four animals usually consisted of mice from two different strains. To efficiently test a large number of animals, an experimental pass was started every two days. Over a 6-week period, 10 mice per BXD-1,BXD-11, BXD-12, BXD-13, BXD-21, and BXD-27, and 8 D2 and 12 B6 mice were tested across 18 different experimental passes. Drug was prepared fresh approximately every three days.

DATA ANALYSIS

All statistical analyses and graphical representations were performed with the IBM version of SYSTAT and SYGRAPH software packages. Percent reduction in food intake (MICROPCTRED) was calculated by subtracting the 30 minute food consumption (g) on Day 6 from the average baseline consumption (MICROAVGBASE), and then dividing by MICROAVGBASE. This number was then multiplied by 100 to create a percentage

DEFINITIONS OF MICROSTRUCTURE BEHAVIOR

BEHAVIOR	DEFINITION
1). Sitting	Stationary and not engaged in other behaviors.
2). Grooming	Washing, licking and/or scratching involving the body surface.
3). Eating	Mouse on food cup and actively obtaining and consuming food.
4). Drinking	Actively obtaining water from water bottle.
5). Sniffing	Up/down or side/side head movement accompanied by vibrissae movement.
6). Rearing	Front limbs raised with head & body raised.
7). Climbing	Actively hanging or moving on bottom of cage lid.
8). Line Crossing	Locomotor activity assessed by crossing a point between front and back of cage.
9). Other Behaviors	Any behavior not previously defined.

Table 4. Definitions of the Microstructure behaviors quantified during the 30 minute food access period in Experiment 3. Animals received either saline or MA (1 mg/kg) s.c. 15 minutes prior to access period.

score. For Group 1, Day 6 was a drug day, while for Group 2 Day 6 was a saline day. Average baseline feeding (MICROAVGBASE) was calculated by averaging 30 minute food consumption across Day 3, Day 4, and Day 5, similar to AVGBASE in Experiment 1 and Experiment 2. Baseline body weight was taken at the beginning of each experiment when animals were individually housed. Percent reduction in body weight (MICROPCTWRED) was calculated as the ratio of the difference of the baseline weight and Day 6 body weight to baseline body weight. A two-way ANOVA grouped on strain and treatment group was calculated for each of the three indices (MICROPCTRED, MICROAVGBASE, and MICROPCTWRED). Behavioral data from Experiment 1 RI feeding study were recalculated to match the microstructure measurements and genotypic correlations between these dependent measures were calculated.

Microstructure behavioral data were calculated by determining the percent of time engaged in each of the nine behaviors measured over the 30 minute testing period per observer on Day 6. Data from four randomly chosen experimental passes of different strains from each rater were correlated to assess the interrater reliability ratio. Data from both raters for each behavior measured were averaged to produce the percent time an animal engaged in each behavior over the 30 minute test period. Strain means were calculated for each of the nine microstructure measures. Two-way ANOVAs for strain and group were run for each of the nine

microstructure measures. In addition, numerous correlations between microstructure indices were assessed between and within both treatment groups.

RESULTS

MICROSTRUCTURE FEEDING COMPONENT

As in Experiment 1, animals' daily food consumption during the 30 minute test period appeared to be stabilized by Day 5, the last baseline day. In fact, Day 4 & 5 consumption was similar. Figure 12 shows food consumption across baseline days (Day 1-Day 5) collapsed on strain. As in the previous feeding studies, MICROAVGBASE was calculated for Days 3, 4 & 5 as the index of pretreatment food consumption. There was no significant difference between treatment groups or strain by group interaction on this measure. However, when collapsed on group there was a significant strain effect [$(7,64) = 6.212$, $p < 0.0001$] for MICROAVGBASE (Figure 13).

Figure 12 also shows methamphetamine's effect on food consumption on drug day (Day 6) for the two treatment groups. There was a significant decrease in food consumption in the MA group (Group 1) as compared to the Saline group (Group 2) on drug day [$(1,64) = 77.885$, $p < 0.0001$]. There was a significant strain difference in consumption on Day 6 [$(7,64) = 3.007$, $p < 0.01$] but no interaction between strain and group. No significant correlation between the average baseline consumption indices for the eight strains tested in Experiment

Figure 12. Daily food consumption during 30 minute food access period for Group 1 and Group 2 in Microstructure study. On Drug day animals received either saline (Group 1) or 1 mg/kg MA s.c. (Group 2) 15 minutes prior to drug access period. As shown, food consumption steadily increased and was stabilized by Day 5. On Drug Day, administration of MA reduced food intake in Group 2 as compared to Group 1.

Consumption Across Days

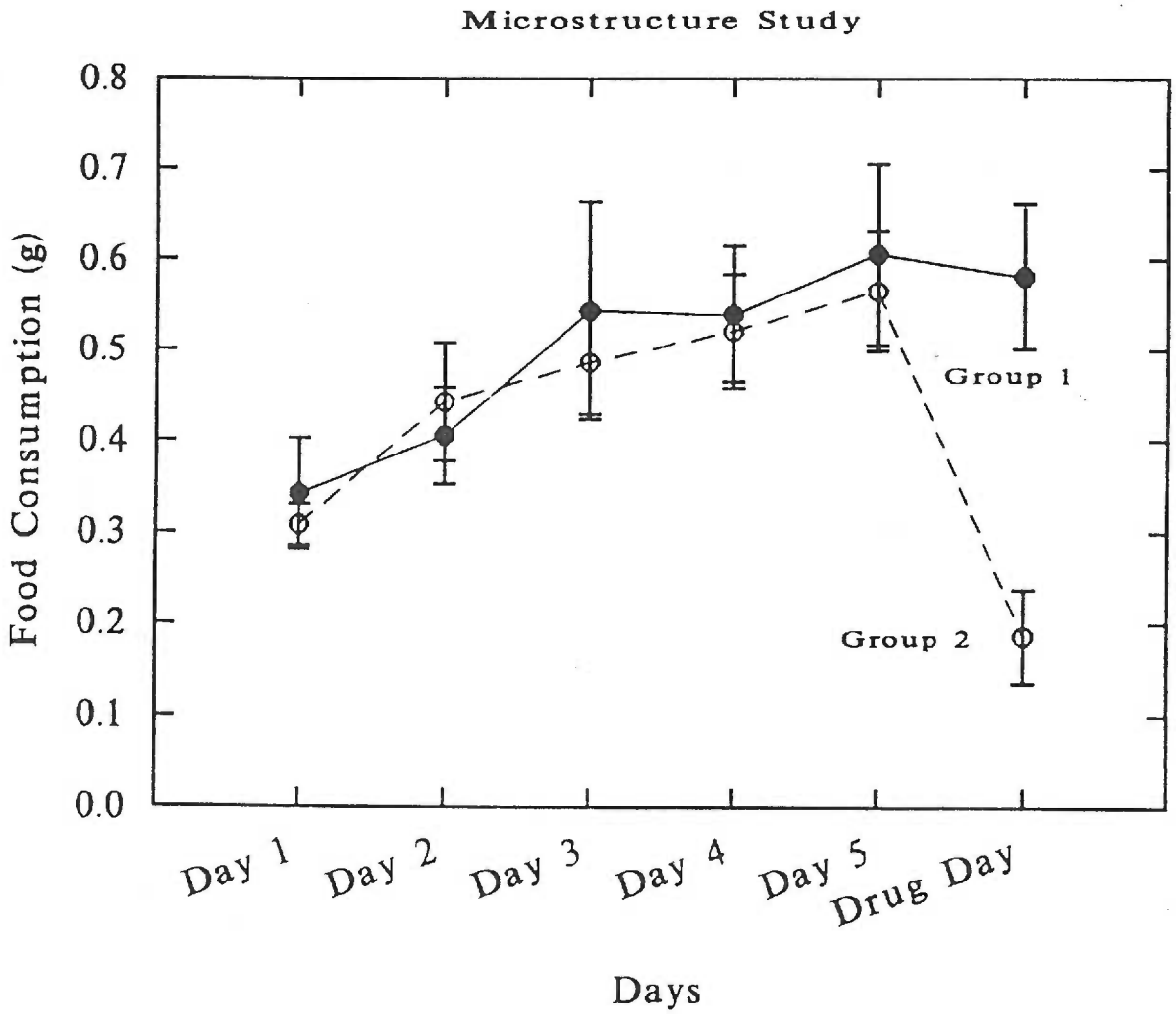
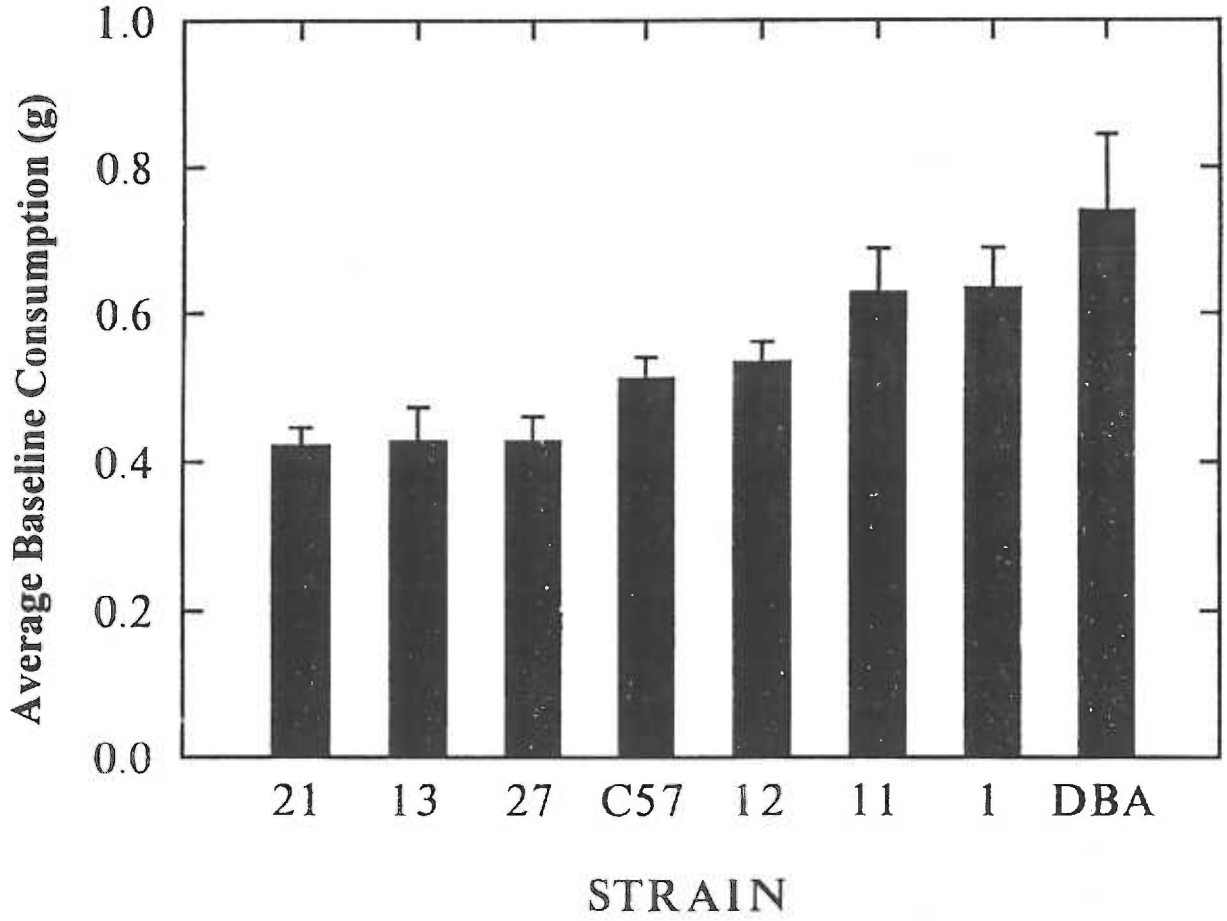


Figure 13. Strain means (±SEM) for 8 strains tested in Experiment 3 for average baseline consumption (MICROAVGBASE). MICROAVGBASE was calculated by averaging the consumption during the 30 minute food access period across Day 3, Day 4 and Day 5. As shown, there was a significant strain effect for MICROAVGBASE.

Microstructure Study



1 and this study was seen.

The dependent variable used to assess drug effect in this study was MICROPCTRED (percent reduction in food consumption). There was a significant group effect [(1,64)= 95.163, p< 0.0001] but no strain or strain by group effect for percent reduction in consumption (MICROPCTRED). However, the pattern of strain sensitivity to MA's effect on feeding behavior was similar to that seen in Experiment 1 for percent reduction in consumption (Figure 14).

Percent reduction in body weight (MICROPCTWRED) was also assessed in this study. There was a significant strain effect [(7,64)= 4.131, p< 0.001] but no group or interaction effect for MICROPCTWRED (Figure 15). Only one strain (BXD-27) had a mean loss of more than 20% of baseline body weight. This strain appeared healthy and active. Similar results for this strain were seen in Experiment 1. There was a significant correlation between strains for MICROPCTWRED and PCTWRED from Experiment 1 (r=0.736, p< 0.038).

There were no significant genotypic correlations (Pearson's r correlations for strain means) between the different indices (MICROPCTRED, MICROPCTWRED & MICROAVGBASE) measured during Experiment 3.

Figure 14. Strain means (±SEM) for MA-induced anorexia expressed as MICROPCTRED. MICROPCTRED was calculated by subtracting the 30 minute food consumption on Day 6 from MICROAVGBASE and then dividing by MICROAVGBASE. Animals received 1 mg/kg MA s.c. 15 minutes prior to the food access period. There was no strain effect for MICROPCTRED. However, the pattern of strain sensitivity to MA's effect on feeding behavior was similar to that seen in Experment 1.

Microstructure Study

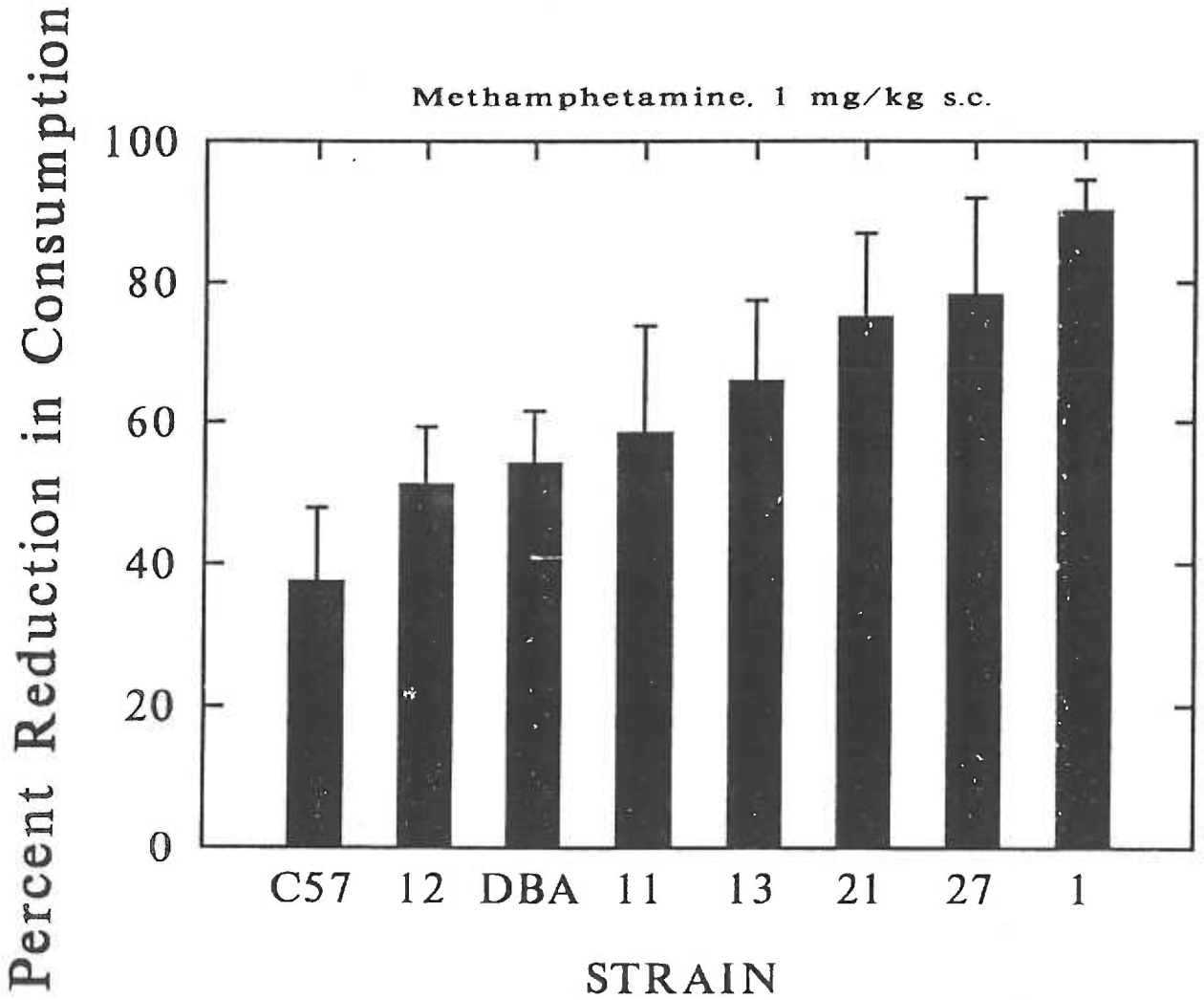
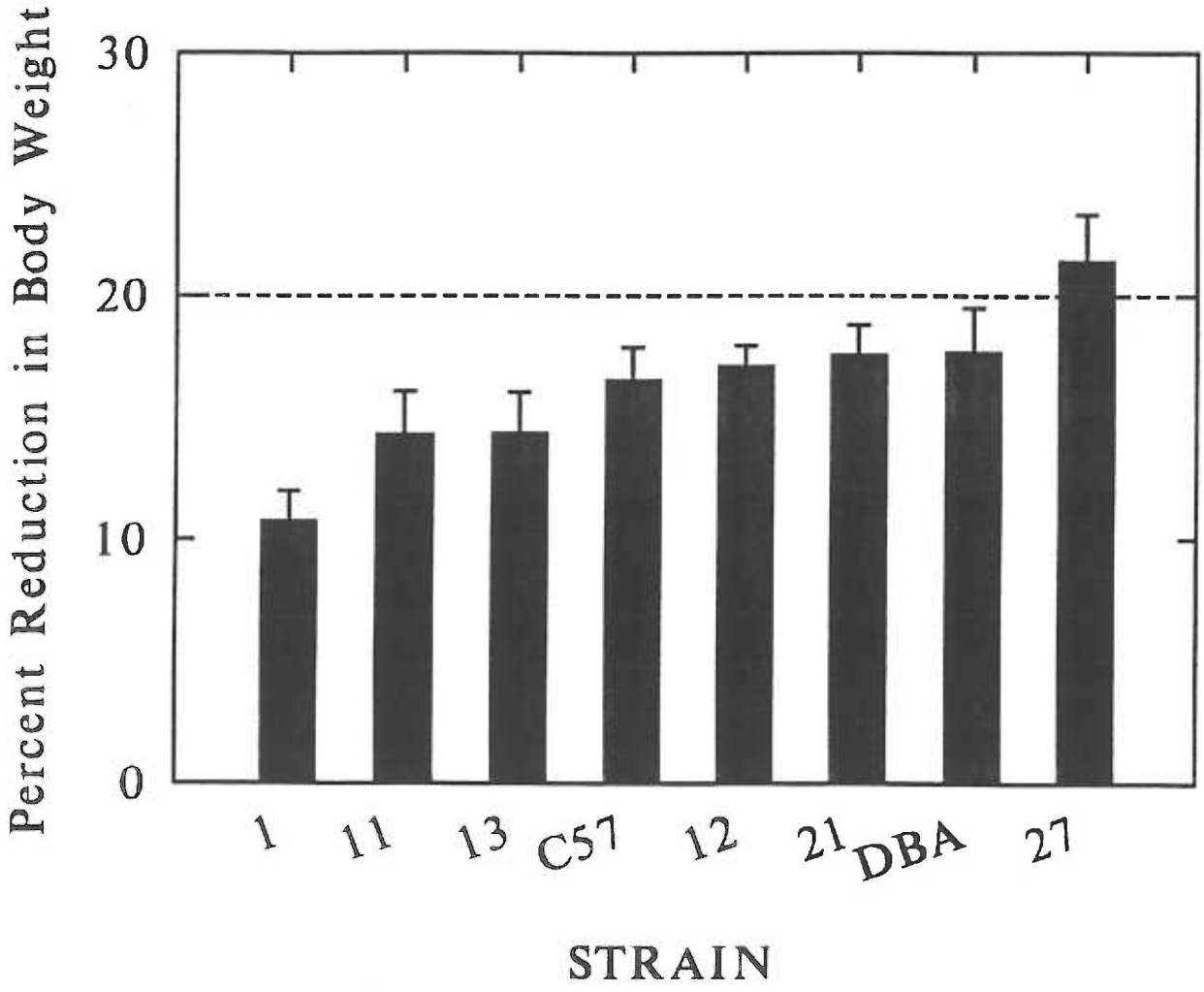


Figure 15. Strain means (±SEM) for percent reduction in body weight (MICROPCTWRED). MICROPCTWRED was calculated by subtracting Day 6 body weight from the baseline body weight and dividing by baseline body weight. The dashed lines indicate the threshold of 20% or more loss of initial body weight. As shown, there was a significant strain effect for PCTWRED. Only one strain loss more than 20% of baseline body weight. This strain appeared healthy and active.

Microstructure Study



MICROSTRUCTURE BEHAVIORAL COMPONENT

The highly significant interrater reliability of the observed microstructure behaviors assessed by correlating four random experimental passes of different strains from each of the raters ($r= 0.986$, $p< 0.001$) allowed both rater's microstructure data to be combined across strains. Figure 16 & 17 show the microstructure behaviors expressed as percentage of total behavior for the Saline and MA groups, respectively, across the different strains. Animals receiving saline were predominantly observed eating. The mean percentage of time eating for the Saline group was 68.09% (±S.E.M. 7.73). In the extreme strains tested, the percentage of time spent eating ranged from 76% (D2) to 53% (BXD-11). Data collapsed on strain showed behaviors such as sniffing ($\bar{x}= 13.50$, ±S.E.M. 4.00) and drinking ($\bar{x}= 7.12$, ±S.E.M. 1.84) constituted most of the remaining test period (Figure 16). The mean percentage of time eating for the MA group was 49.03% (±S.E.M. 10.02). In the extreme strains tested, the percentage of time spent eating ranged from 62% (B6) to 36% (BXD1). Data collapsed on strain showed sniffing ($\bar{x}= 25.87$, ±S.E.M. 9.70) as the single behavior dominating the remaining test period (Figure 17). Figures 18 and 19 show pictures of some of the microstructure behaviors. Still-life photographs were taken from videotaped sessions.

A two-way ANOVA for strain and treatment group on each microstructure behavior detected numerous significant

Figure 16. Microstructure behaviors expressed as a percent of behaviors during the 30 minute food access period for each of the strains tested. Each animal received saline s.c. 15 minutes prior to the food access period. Refer to Table 4 for definition of behaviors. Figure shows that animals ate the majority of the food access period. Sniffing and drinking constituted most of the remaining test period.

Microstructure Behaviors

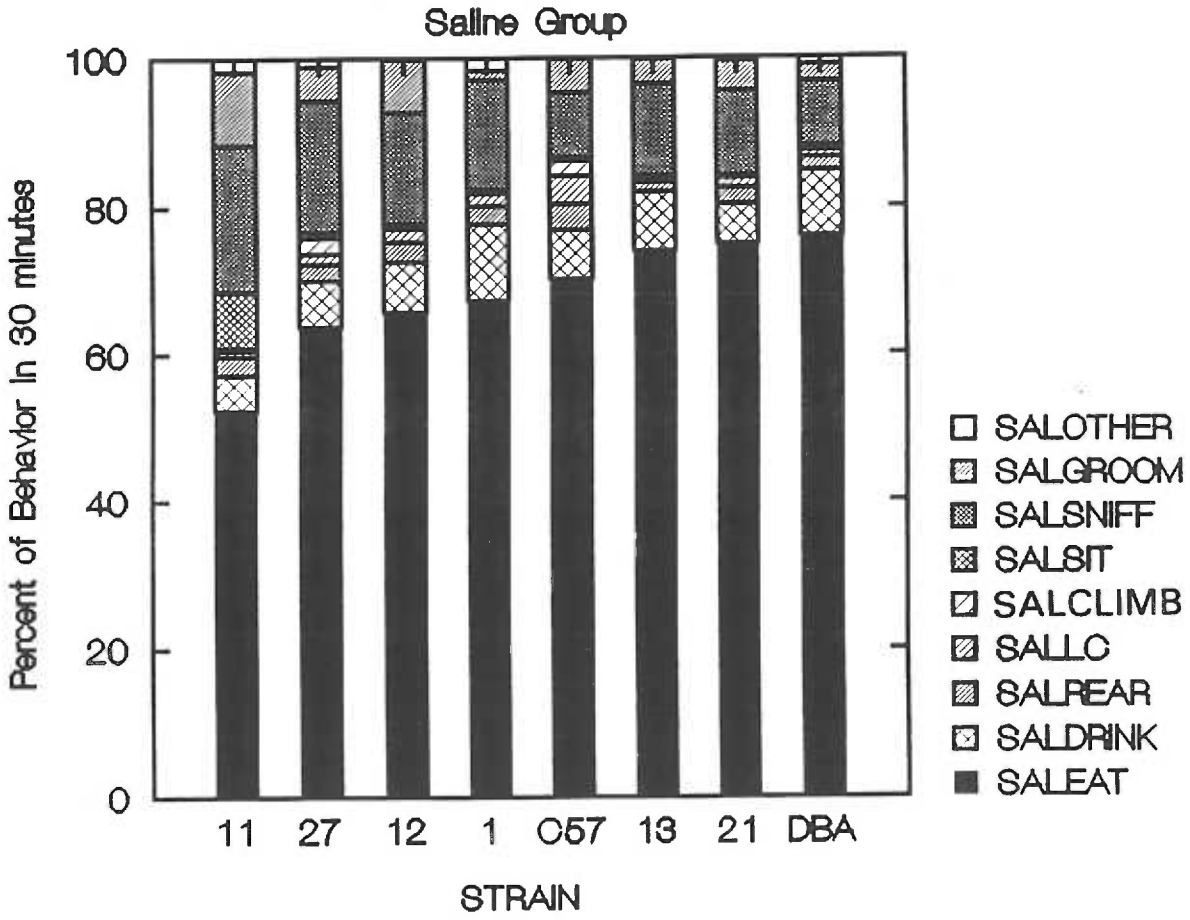


Figure 17. Microstructure behaviors expressed as a percent of behaviors during the 30 minute food access period for each of the strains tested. Each animal received 1 mg/kg MA s.c. 15 minutes prior to the food access period. Refer to Table 4 for definition of behaviors. Animals ate 49% with sniffing beings the single behavior dominating the remaining test period.

Microstructure Behaviors

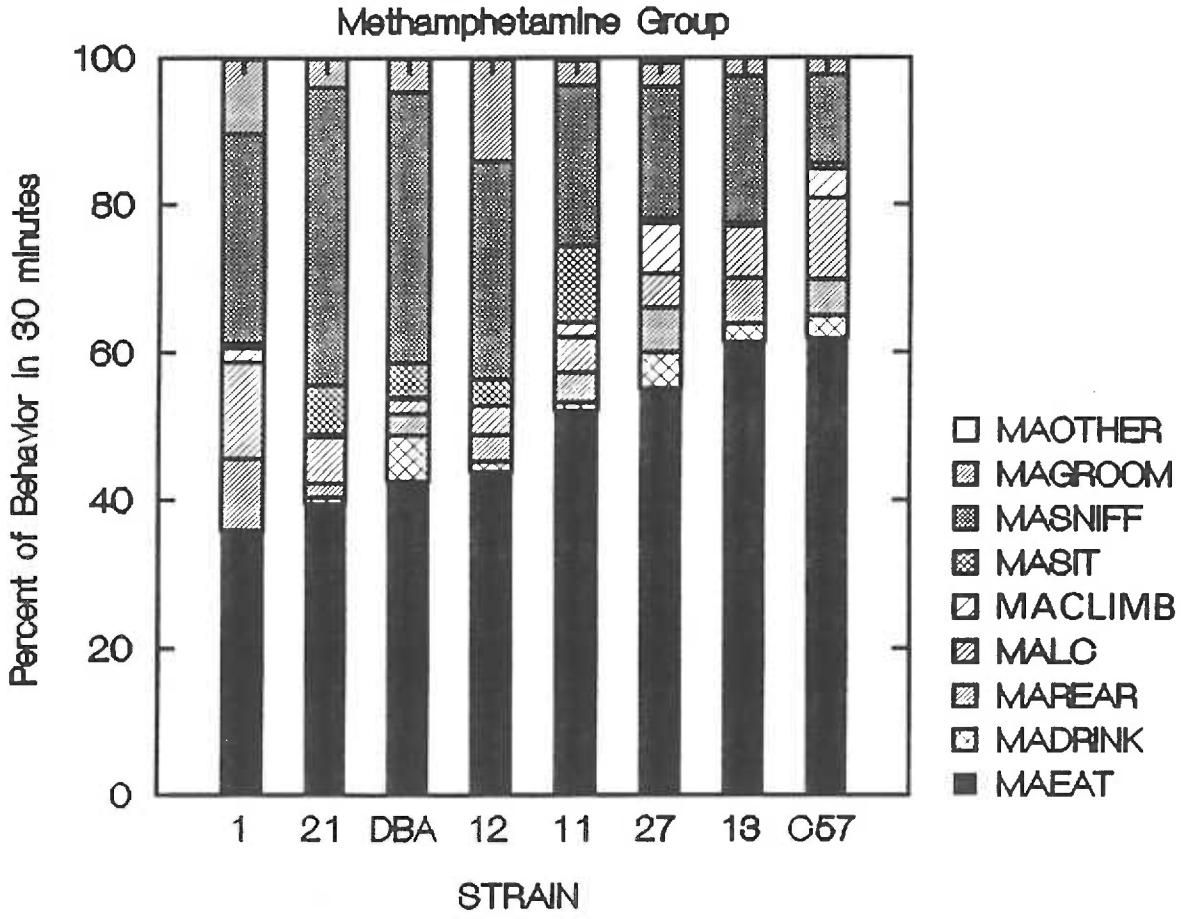


Figure 18. Representative photographs of the microstructure behaviors (a) eating, (b) line crossing and (c) stationary. Pictures were taken from videotaped sessions.

a

b

c

Figure 19. Representative photographs of the microstructure behaviors (d) drinking, (e) rearing and (f) sniffing. Pictures were taken from videotaped sessions.

d

e

f

findings. Examination of the effect of treatment group showed a decrease in eating [(1,60)= 35.902, p< 0.0001], and drinking [(1,60)=73.470, p< 0.0001] in the MA group as compared to the Saline group. In addition, there was an increase in sniffing [(1,60)= 21.241, p< 0.0001], rearing [(1,60)= 24.635, p< 0.0001], linecrossing [(1,60)= 55.231, p< 0.0001] and a decrease in the other behavior category [(1,60)= 18.602, p< 0.0001] in the MA group as compared to the Saline group. There was a significant main effect of strain for drinking [(7,60)= 3.516, p< 0.01], rearing [(7.60)= 3.174, p< 0.01], linecrossing [(7,60)= 4.307, p< 0.001], climbing [(7,60)= 2.725, p< 0.05], sitting [(7,60)= 2.796, p< 0.05] and the other behavior index [(7,60)= 2.842, p< 0.05]. Significant strain by group interactions were seen for drinking [(7,60)= 2.454, p< 0.05], sniffing [(7,60)= 2.681, p< 0.05] rearing [(7,60)= 3.032, p< 0.01], and linecrossing [(7,60)= 2.508, p< 0.05].

Pearson's correlation of strain means among the nine microstructure measures within the Saline group identified numerous correlations. Table 5 shows the significant correlations identified within the Saline group itself for the microstructure behaviors. Eating (Saleat) was negatively correlated with sitting (Salsit), sniffing (Salsniff) and grooming (Salgroom). Sitting (Salsit) and grooming (Salgroom) were positively correlated, as were the two measures of locomotor activity (line crossing, Sallc and rearing,

CORRELATIONS FOR MICROSTRUCTURE BEHAVIORS

WITHIN THE SALINE GROUP

BEHAVIOR 1	BEHAVIOR 2	r	p<
SALSIT	SALEAT	-0.840	0.009
SALSIT	SALGROOM	0.784	0.021
SALLC	SALREAR	0.879	0.004
SALSNIFF	SALEAT	-0.878	0.004
SALGROOM	SALEAT	-0.773	.025

Table 5. The significant correlations and their p values for microstructure behaviors within the saline group.

Salrear).

Pearson's correlations of strain means among the nine microstructure measures within the MA group identified four significant correlations. Sniffing after MA administration was negatively correlated with eating after MA administration ($r= -0.847$, $p< 0.001$), i.e., if sniffing, an animal tended not to eat. Line crossing after MA administration was significantly correlated with rearing after methamphetamine ($r= 0.707$, $p< 0.05$), i.e., if animal was moving back and forth in the cage, it tended to also show rearing behavior.

Pearson's correlation among behaviors between the two treatment groups identified two significant correlations. Sitting ($r= 0.759$, $p< 0.05$) and climbing ($r=0.914$, $p< 0.01$) after both saline and MA administration were significantly correlated. The level of sitting and climbing behavior was similar whether animals were drugged or not.

SUMMARY

In the microstructure study, as in Experiment 1, methamphetamine significantly decreased food consumption in a genetically diverse population of mice. No significant strain difference was found for percent reduction in consumption in Experiment 3. However, a similar pattern of sensitivity to the effect of MA was seen in Experiment 1

and Experiment 3 for the strains tested.

The breakdown of behaviors into various categories provided a wealth of information. Overall, animals in both the drug (49%) and saline (68%) groups spent the majority of their time eating, while sniffing dominated the majority of the remaining time. A decrease in eating behavior was accompanied by an increase in sniffing, linecrossing and rearing.

Correlations of strain means within both the saline and drug group showed similar patterns. If sniffing, an animal tended not to eat. However, this pattern of behavior was more pronouced in the MA treated group. In addition, if an animal moved about in the cage it was likely to display rearing behavior.

CONCLUSIONS AND SIGNIFICANCE OF RESULTS

The aim of this study was to utilize both genetics and behavioral techniques to identify the underlying mechanism(s) involved in the effect of methamphetamine on food consumption. It has long been known that amphetamines decrease food consumption, producing an anorectic state in both animals and humans. What is less clear is the systems responsible for this action. In this thesis, a powerful new experimental method that combines behavioral research and modern genetic techniques was used to pinpoint the system(s) mediating amphetamines, namely methamphetamine's anorectic effect. In addition, to rule-out possible alternative explanations for MA's influence on food consumption, a microstructure study was performed. In the following pages, the results of the two-phase behavioral genetic study (Experiment 1 and Experiment 2) as well as the microstructure study (Experiment 3) will be summarized and their significance discussed.

PART I. EXPERIMENT 1

Methamphetamine decreases food consumption in a panel of BXD mice. A scheduled feeding paradigm was employed to study the anorectic effect of methamphetamine in mice. This design was chosen instead of a total deprivation model, gavage, sweetened mash or free-feeding model for a number of reasons. First, since a large number of animals of different inbred strains were being tested it was important to control environmental factors as much as possible and obtain baseline behavioral measurements. A scheduled feeding paradigm allowed consumption to stabilize prior to drug administration and animals to experience the effect of only partial deprivation. Therefore, food reduction would more likely be a measure of drug effect and not a consequence of sudden removal of food or starvation as in a total deprivation model. Blake (1970) previously showed a difference in the response of the progenitors, B6 and D2, to total food deprivation. D2 mice survived continuous food deprivation much longer than B6 mice, which were more sensitive to repeated days of deprivation and died. In this same study, tyrosine aminotransferase (TA) activity remained low in D2 mice, while TA activity increased four-fold within 60 hours of food deprivation in B6 mice (Blake, 1970).

Therefore, a total food deprivation design would create differential behavior and physiological results in the progenitors independent of drug action which could confound the interpretation of the effect of methamphetamine on food consumption. In this thesis, the animals experienced food deprivation between scheduled feeding periods. This partial deprivation could cause a baseline physiological difference but nothing as dramatic as seen in a total food deprivation model. Another reason a scheduled feeding paradigm was used was previous experimental success studying the effect on consumption in mice utilizing the schedule feeding paradigm (Duterte-Boucher et al., 1990). A scheduled feeding paradigm minimizes changes in hunger, food preference, response to taste and macronutrient preference and physiology in animals (Blundell, 1991; LeMagnen, 1992). In addition, scheduled feeding decreases meal pattern variation and periodicity seen in free-feeding paradigms (LeMagnen, 1992). Finally, this design was used because it was most appropriate for large sample sizes required in a RI study.

Other indices measured in this study were important for ruling out alternative explanations of food reduction after MA administration. The measurement of baseline consumption as the average consumption of days 3 thru 5 was chosen because it provided a good index of basal consumption and, when used in the calculation of percent reduction, gave the largest heritability value. There was a strain difference

in basal consumption but heritability was very low. More importantly, overall baseline consumption stabilized before drug exposure. This is important since it assures animals had adapted to the feeding schedule and suggests that intake reduction was due to administration of drug rather than to variability in food consumption. In addition, lack of correlation between baseline consumption and food reduction indicated the previous pattern of food consumption was not associated with the drug effect.

The measurement of body weight loss was chosen because it was a good monitor of the animals' health. Percent reduction in body weight indicated strains could maintain body weight and stay healthy on a scheduled feeding paradigm. However, a group difference for percent body weight loss indicates there was a drug effect on weight loss. Body weights were measured at the beginning of each day and weight loss was the percent score of the difference between the beginning weight and the end (Day 7) weight. Day 7 body weight for Group 1 measured body weight after a saline injection while Group 2 day 7 body weight measured weight after a MA injection. Therefore, the reduction in food consumption was reflected in body weight. This phenotype has a large genetic component as seen by the large heritability score. Forty-three percent of the variance seen among the strains could be accounted for by strain (genetic) differences in weight loss. The feeding schedule

had a differential effect on strains' weight loss, but lack of correlation with food reduction and weight reduction rules out the possibility of weight loss contributing to drug effect or explaining the decrease in food consumption (i.e., if an animal was too sick from severe weight loss it would not be able to consume food regardless of drug exposure).

A reduction in food consumption after MA administration was seen only in the 30 minute food access period. Total food consumed was not affected and no rebound hyperphagia was seen in the afternoon food access period. A previous report showed a rebound effect in Swiss albino mice 60-120 minutes after presentation of food (Duterte-Boucher et al., 1990). In this study, animals were given 1.25 mg/kg of amphetamine 15 minutes prior to food presentation. The dissimilarity in results could be explained by the different strains and dose used in each study. In addition, there was a difference in the time of food availability and measurement of drug effect in the two studies. Nevertheless, at this dose food intake was decreased in albino mice similar to that seen in the BXD RI mice.

Administration of methamphetamine was subcutaneous instead of intraperitoneal so as not to cause gastro-intestinal irritation, which in turn could affect food consumption. Drug was administered 15 minutes prior to the 30 minute food consumption period to obtain the optimal drug

effect. Previous work had shown peak amphetamine concentration in mice to be within the first hour after administration (Hicks et al., 1980). In addition, Duterte-Boucher et al. (1990) had successfully used a similar design in mice.

Melega et al. (1995) have recently shown the pharmacokinetics of amphetamine and methamphetamine to be equivalent, and both to have a similar effect on dopamine terminals in the striatum. This finding is important since the majority of studies completed have used amphetamine. Therefore, with this study in mind, results from amphetamine and methamphetamine studies can be confidently compared. In addition, Melega et al. concluded that the change in extracellular DA levels were derived from the actions of amphetamine and methamphetamine and not their metabolites.

The majority of studies examining drug-induced anorexia have concentrated on the action of amphetamine in a rat animal model; little or no research has been done in mice. This study is the first to investigate the anorectic effect of methamphetamine in a large panel of recombinant inbred mice. Since we were interested in mapping the genes involved in the anorectic effect of MA, we used the BXD RI strains. The BXD mouse strains are used extensively in behavior genetic studies since a wealth of information concerning their genetic makeup exists (Gora-Maslak et al., 1991). In this study, results showed a genetic variance in

a panel of BXD RI strains for reduction in food consumption after methamphetamine administration. Heritability was moderate-to-low and no difference between the progenitors was seen, ruling out a single gene effect. However, differences among strains indicate a polygenic model of inheritance making this phenotype suitable for QTL analysis.

QTL analysis identified five putative QTLs for MA-induced anorexia. Three of the five QTLs were within 10 cM of candidate genes for the serotonin (*Fv2*), dopamine (*Tpmt*), and adrenergic receptor systems (*D19Byu1*). All three of these neurochemical systems have been shown to play key roles in the mediation of feeding behavior (Samanin & Garattini, 1993; Hoebel et al., 1989). Evidence from pharmacological studies indicate that 5-HT agonists cause satiety while 5-HT receptor antagonists and uptake inhibitors reduce food intake in rats. It has been suggested that the serotonergic system affects the state of satiety and in fact serotonergic drugs have been proposed in the treatment of bulimia and clinical obesity (Samanin & Garattini, 1990; McGuirk & Silverstone, 1990). As previously discussed, the dopamine receptor system has been thought to play a key role in anorexia induced by amphetamines. Numerous pharmacological studies using receptor agonists and antagonists have shown the dopamine receptor system to influence feeding behavior (Samanin et al., 1975; Leibowitz, 1978; Blundell & Latham, 1980; Hoebel

et al., 1989; Duterte-Boucher et al., 1990). Finally, pharmacological studies have suggested that the adrenergic system plays a part in amphetamine-induced anorexia (Wellman, 1992). In fact, Hoebel et al. (1981) have suggested that the β-adrenergic and dopaminergic systems within the lateral perifornical region of the hypothalamus function in concert to suppress food intake (reviewed by Wellman, 1992). The literature shows that all three of these receptor systems have some involvement in the mediation of feeding behavior. It is of interest that the three main neuroreceptor systems studied in drug-induced anorexia were identified by QTL analysis.

Part I. EXPERIMENT 2

A two-phase mapping strategy identified and confirmed a putative QTL for the anorectic effect of methamphetamine. In Experiment 2, five putative QTLs were detected, however multiple regression analysis suggested that the data only supported the possibility of no more than two QTLs. Nevertheless, all markers with linked polymorphic *Mit* microsatellite markers were pursued with PCR. This was done with two things in mind; first, to confirm the statistical findings and second, to perform the confirmation process of genotyping F_2 mice with PCR. Unfortunately, at the time of this experiment no polymorphic *Mit* microsatellite markers

were available for confirmation of *Hcf-3* on chromosome 4 or *D19Byu1* on chromosome 19. The latter was within 10 cM of the beta- adrenergic receptor kinase-1 loci.

Results of the confirmation process suggested linkage of the locus, *Tpmt*, per Lander and Kruglyak's standards (1995). Lander and Kruglyak have proposed new standards for reporting the results from genetic mapping studies. Instead of the traditional lod score of 3 as obligatory for significant linkage, these authors propose categorizing results of mapping studies into four different groups depending on the probability of linkage to occur in a study. The four groups include 1) suggestive linkage, 2) significant linkage, 3)highly significant linkage and a 4) confirmed linkage. For example, a QTL is considered suggestive in a mouse or rat intercross model with dominance when a *p*-value of 2.4×10^{-3} and lod=2.0 is found. It would be considered a significant linkage when a *p*-value of 7.2×10^{-4} and lod=3.4 is detected. The authors have proposed such a system to standardize the numerous studies being pursued and to ensure that any possible linkage that could be informative is not ignored (Lander and Kruglyak, 1995).

In this study, the relatively small number of F_2 mice phenotyped reduced the probability of confirming a QTL. The number of F_2 mice tested allowed the detection of QTLs only down to about 23% of the phenotypic variance with a power of 0.9, or about 15% when power is 0.5. (Soller et al., 1976;

Lander & Botstein, 1989). However, due to time constraints, availability of mice and the extensive paradigm used for the measurement of this phenotype, the number was deemed adequate to begin the confirmation process for the larger QTLs. In this study, a lod score of 3.1 and a *p* value of 1.6×10^{-4} was found; this is just below the significant linkage level suggested by Lander and Kruglyak (1995). If more animals were phenotyped and in turn genotyped, the power would be increased and the QTL might be classified as significant or even highly significant. In addition, other QTLs could also be confirmed with the addition of more animals.

Thiopurine methyltransferase (*Tpmt*) is an enzyme that catalyzes the S-methylation of thiopurine drugs which are used in treatment of neoplastic diseases (Weinshilbom et al., 1992). The identification of a suggestive linkage for *Tpmt* could be important since this locus is only 1 cM from the murine dopamine D1 receptor subtype (Drd1a) on chromsome 13. This QTL shows extensive linkage homology with the human chromsome 5q34-q35 where the human dopamine D1 receptor subtype (Drd1a) is located (Wilke et al., 1993). It has been proposed that the D1 dopamine receptor is involved in the inhibition of feeding in animals and humans (Terry & Katz, 1992; Terry & Katz, 1994; Martin-Iverson & Dourish, 1988; Gilbert & Cooper, 1985; McGuirk et al, 1991). Pharmacological studies have shown that D1 receptor agonists

suppress feeding in rats using a variety of paradigms. Using a highly-palatable diet, Cooper et al. (1990) found that SK&F 38393 significantly reduced food consumption in non-deprived rats adapted to a scheduled feeding paradigm with sweetened mash. Also, SK&F 38393 decreased food pellet intake in a free-feeding situation (Martin-Iverson & Dourish, 1988) and in a food-deprivation model (Rusk & Cooper, 1989). Additionally, SK&F 38393 and CY 208-243, another D1 agonist, produced dose-related reductions in sucrose sham feeding in rats (Cooper et al., 1993). The role of the D1 receptor system in the inhibition of feeding behavior is further supported by the absence of stereotypical behavior after administration of D1 agonists. (Martin-Iverson & Dourish, 1988; Terry & Katz, 1992). Therefore, the finding from this dissertation adds support to the previously reported pharmacology studies, suggesting the dopamine D1 receptor has a key role in the control of feeding behavior.

The QTL analysis did not identify any putative loci near either the *anx* or *Adra-2b* genes on chromosome 2. Feeding behavior is a complex phenotype. The MA-induced anorexia phenotype measured in this thesis appears to be under a different genetic control than the phenotype controled by the *anx* mutation.

PART II. EXPERIMENT 3

Microstructure analysis verified the anorectic effect of methamphetamine. One goal of this study was to reanalyze the sensitivity of MA-induced anorexia in a select panel of BXD RI strains and progenitors. As in Experiment 1, administration of MA decreased food consumption similarly in the strains tested. However, in Experiment 1, a strain difference in MA-induced anorexia was seen while in Experiment 3, there was no strain difference. One explanation for this could be a difference in the number of animals tested. In Experiment 1, 14-29 animals per strain were tested while in this experiment only 8-10 animals per strain were tested. Unlike Experiment 1, where data from animals in each treatment group were combined to calculate PCTRED, in this experiment the number of animals per treatment group was half of the animals per strain tested. Therefore, in Experiment 1, data from 29 animals per strain were used in the analysis while in this study data from only 4 animals per strain were used. This difference in experimental size could result in increased variability and decreased power in the latter study. Additionally, the use of the videotaping equipment required a slight change in cage location on drug day. It is possible that this change could have altered the expression of the phenotype being measured. Nevertheless, the pattern of drug sensitivity

seen in both experiments indicates that it was only a difference in magnitude and not direction of drug effect.

In addition, a major aim of this study was to see if the anorectic effect of methamphetamine seen orginally could be attributed to a gross change in behavior. This consideration is based upon studies which have demonstrated that amphetamines stimulate locomotor activity and produce stereotypy (Segal, 1975; Eichler et al., 1980; Mueller et al., 1989). It might, therefore, follow that the anorectic effect of amphetamines might purely be a side effect of the drug's locomotor stimulant effect (Lyons & Robbins, 1975, Blundell & Latham, 1982). Amphetamine could thus alter non-consummatory behaviors which could in turn compete with eating behaviors and cause a decrease in food consumption. In fact, microstructure studies using D1 and D2 receptor agonists which are putative anorectic agents have shown different effects on consummatory behaviors, as well as on non-consummatory behaviors. The D2 receptor agonist, N-0437, has been shown to affect various behavioral indices measured during a microstructure study. At low doses, this agonist reduced locomotor activity and exploratory sniffing, while increasing yawning, and having no effect on grooming or rearing activity. At higher doses, N-0437, produced an increase in sniffing and oral behaviors, while suppressing grooming (Rusk & Cooper, 1989). In contrast, the D1 receptor agonist, SK&F 38393, has been shown to cause

excessive grooming and reduced locomotor activity while not affecting rearing, sniffing or resting (Cooper, Francis & Rusk, 1990). The different effect on grooming has led to the speculation that grooming behavior would be a good index of D1 vs. D2 receptor function (Cooper et al., 1990). In this thesis, however, no change in grooming was seen. In another study, the D2 receptor agonist PHNA increased both consumption and oral behaviors, while the SK&F 38393 decreased consumption and produced no change in oral behaviors (Martin-Iverson & Dourish, 1988). Cooper et al. (1990, 1993) have seen similar patterns of decrease in consumption after D1 receptor administration with no effect on non-consummatory behaviors.

Studies examining D1 and D2 receptor involvment in the production of stereotypic behaviors have suggested that the expression of the D2 receptor agonist motor stimulant effect requires concurrent activation of D1 receptors (Martin-Iverson & Dourish, 1988; Starr & Starr, 1987; and Vasse et al., 1988). Perhaps there is an interaction of the D1 and D2 dopamine receptor subtypes to produce drug-induced anorexia. D1 and D2 dopamine receptor subtypes may work in concert and/or independently to produce a change in food intake and non-consummatory behaviors which alter feeding behavior.

In the present study, there were differences between the two treatment groups on some of the behavioral indices.

Examination of the results indicate that MA decreased food consumption, but not without affecting non-consummatory behaviors such as sniffing, line crossing and rearing. It is possible that in the MA-treated group in the microstructure study and also in the BXD and F_2 studies, a change in food intake was the result of alteration in these non-consummatory behaviors. However, there was no genetic correlation of MA-induced anorexia with stimulated locomotor activity as measured in an open-field apparatus after administration of 1 mg/kg MA in another set of progenitors and BXD RI strains (Phillips, 1994). This difference may be due to the difference in activity in an open-field environment vs closed environment seen in the homecage or even the satiation status of the animals. In the Phillips study, animals were not food deprived as animals were in this thesis.

Sniffing was the single behavior of consequence besides eating that dominated the test period in each treatment group. A similar effect has been seen with GBR 12909, a dopamine uptake inhibitor. This drug showed a striking behavioral effect of increasing sniffing activity while reducing food intake in a microstructure study (van der Hoek & Cooper, 1994). These authors concluded that drug-induced anorexia and intense sniffing are closely related to dopamine uptake inhibition. Therefore, perhaps the pattern of behavior seen here could be attributed to the ability of

MA to block the uptake of dopamine.

Another explanation for the pronounced sniffing seen in this study could be explained by the appetitive motivational hypothesis. Ikemoto and Panksepp (1994) have hypothesized that self-stimulation "reward" is caused by activation of brain systems that mediate psychobehavioral processes necessary for survival. These behavior processes include eating, drinking, rearing, sniffing, defense, and reproduction. This ethologically based theory is derived from observation of brain-stimulation studies. Electrical stimulation at the lateral hypothalamic-medial forebrain bundle (LH-MFB) induces drinking and increase rearing, sniffing and locomotion. In fact, there is such a strong relationship between exploratory sniffing and self-stimulation that sniffing is used as an index for appropriate electrode placement in lateral hypothalamic-medial forbrain bundle (LH-MFB) implanation surgery. When electrical stimulation is presented at fixed intervals, rats will exhibit vigorous sniffing just prior to stimulation. Ikemoto and Panskepp (1994) theorize that exploratory/investigatory behaviors such as sniffing, rearing and locomotion are necessary to obtain food and are good predictors of appetitve motivation. Interestingly, sniffing, rearing and line crossing were all increased in the MA-treated group. Perhaps in partially food-deprived animals, exploratory sniffing is increased in anticipation

of food presentation and this is enhanced by administration of methamphetamine. This could explain why in addition to eating, sniffing was the one predominate behavior in both the MA-treated and saline groups.

Ikemoto and Panskepp (1994) have modeled a system comprised of sub-systems which are comprised of nodes that are interconnected and mediate appettitive/consummatory behavior. Examples of the sub-systems are drive, sensory-motor integration, motor systems and perceptual/appraisal. Destruction or artifical stimulation of a single node can affect one sub-system while having little effect on another. A similar behavior system approach organized into a hierarchical model has been previously presented by Timberlake and Lucas (1989). These models identify motivational processes and perceptual-motor structures as critical features of behavior systems.

Ikemoto and Panskepp speculate that the dopamine system arouses and coordinates many subfunctions of the appetitive motivation system. Therefore, administration of methamphetamine may influence this general motivation system by affecting one node or sub-system to produce increased sniffing. This is in turn reflected in a change in feeding behavior.

SUMMATION AND FUTURE DIRECTIONS

Using a two-phase genetic mapping strategy, this study was able to detect and confirm a QTL with suggestive linkage on chromosome 13 for the anorectic effect of methamphetamine. This QTL near *Tpmt* is within 1 cM of the dopamine D1 receptor subtype locus. Previously reported pharmacological studies complement this finding, suggesting mediation of the anorectic effect of amphetamines by the dopamine D1 receptor system. Finally, Experiment 3 showed that MA at this dose reduced food intake, but did alter some non-consummatory behaviors. This raises the possibility that the phenotype measured was a reflection of alteration of both consummatory and non-consummatory behaviors by the D1 dopamine receptor.

Future research in this area could again combine genetics and pharmacology. For instance, the role of the D1 receptor subtype in MA-induced anorexia could be studied by examining the action of dopamine D1 agonists and antagonists on consumption in selected BXD RI strains. The sensitivity of MA-induced anorexia is now known in the BXDs, therefore, would the same profile exist for sensitivity to selected agonists and antagonists? Similar profiles would further support the involvement of the D1 receptor system in this phenotype. Also, it would be interesting to find out if the localization and densities of the D1a dopamine receptor

in the progenitors and BXD strains fall in line with the MA-induced anorexia.

Confirmation of the QTL near *Tpmt* could be done by pursuing other genetic models. Belknap et al. (in press) have listed many possible independent experimental designs that can be used to support a previously detected QTL. For instance, one could use other RI sets, recombinant congenic sets, standard inbred strains, selectively inbred lines originating from a F_2, congenic strains or by linkage analysis in a backcross or F_2 population using the same progenitor strains as the RI set

In this study, the microstructure design was used to investigate the consequences of non-consummatory behaviors. An exciting aspect of the microstructure design is that it allows the detailed analysis of eating behavior. Therefore, in future studies, microstructure measures such as frequency, duration, and rate of eating could be calculated to provide further insight into the action(s) of amphetamine on the consummatory and non-consummatory aspects of feeding behavior in different genetic strains.

Can the results of this thesis support the importance of the dopaminergic system in food and drug reward processes? The central dopaminergic system has been implicated in reward-related and incentive motivation processes (Fibiger & Phillips, 1986; Gray & Wise, 1980), as well as motor activity (Ungerstedt, 1971). Specifically,

the mesolimbic DA system (MDS) has been suggested to play a role in the general rewarding properties of stimuli (Wise, 1980; Martel & Fantino, 1996). DA release in the MDS has been shown to be potentiated by a variety of stimuli such as drug administration, psychostimulant reward, sexual behavior and self-stimulation (Parada et al., 1988; Louilot et al., 1991l; Pettit et al., 1991). In addition, feeding behavior has been shown to enhance dopamine turnover in scheduled feeding paradigms (Radhakishun et al., 1988) and food-deprivation models (Hernandez & Hoebel, 1990). In the scheduled-feeding paradigm, an increase in DA release in the nucleus accumbens (NA) was seen immediately after onset of the feeding period, while basal levels of DA release were the same in both the satiated and treatment group (Radhakishun et al., 1988). The nucleus accumbens, specifically the shell, has been implicated in the modulation of oral behaviors in rats (Prinssen et al., 1994). Feeding has also been shown to induce dopamine turnover in the prefrontal cortex similar to turnover seen after cocaine self-injection and self-stimulation (Goeders & Smith, 1983; Mora & Meyers, 1977; Hernandez & Hoebel, 1990). These researchers have suggested that the prefrontal cortex plays a role in the cognitive aspects of searching for food reward. The neural circuits that have been suggested to play important roles in the mediation in reinforcement have similar activity with feeding behavior. Therefore, these

systems could play an important role in feeding behavior and the rewarding properties of feeding behavior.

Pothos et al. (1995), studying the relationship of chronic food deprivation and weight loss with extracellular dopamine activity in the NA, have suggested that animals, including humans, who are food deprived may eat food and take drugs when their MDS is depressed and extracellular DA is low in the NA. After feeding or administration of drug, the levels of DA will increase to near basal levels. Therefore, in the paradigm used for this thesis, the dopaminergic system role might be as follows. Animals under a scheduled-feeding paradigm would have decreased extracellular DA levels. In saline animals, feeding would have increased extracellular DA levels, while in drug treated animals MA would have increased extracellular DA levels taking the place of feeding behavior. If this is true then the dopamergic D1 receptor system could have an important role in the mediation of food reward as well as drug reinforcement.

REFERENCES

Angel, I., Stivers, J.A., Paul, S.M., & Crawley, J.N. (1987). Site of action of anorectic drugs: glucoprivic- versus food deprivation-induced feeding. Pharmacology, Biochemistry and Behavior, 27, 291-297.

Bailey, D.W. (1981). Recombinant inbred strains and bilineal congenic strains. In H.L. Foster, J.D. Small & J.G. Fox (Eds.). The Mouse in Biochemical Research, Vol. 1 (pp. 223-239). New York: Academic Press

Bartley, G.H., & Leibowitz, S.F. (1981). Brain monoamines in the modulation of self-stimulation, feeding, and body weight. In H. Weiner, M.A. Hofer and A.J. Stunkard (Eds.). Brain, Behavior and Bodily Disease (pp. 103-142). New York: Raven Press

Belknap, J.K., Dubay, C., Crabbe, J.C., & Buck, K.J. (In press). Mapping quantitative trait loci for behavioral traits in the mouse. In K. Blum & E.P. Noble (Eds.). Handbook of Psychiatric Genetics. CRC Press.

Belknap, J.K., Mogil, J.S., Helms, M.L., O'Toole, L.A., Bergen, S.E., & Buck, K.J. (1995). Localization to chromosome 10 of a locus influencing morphine analgesia in crosses derived from C57BL/6 and DBA/2 strains. Life Sciences (Pharmacology Letter), 57, 117-124.

Belknap, J.K., Mitchell, S.R., O'Toole, L.A., Helms, M.L., & Crabbe, J.C. (1996). Type I and Type II error rates for quantitative trait loci (QTL) mapping studies using recombinant inbred mouse strains. Behavior Genetics, 26, 149-160.

Belknap, J.K. (1993). Personal communication.

Belknap, J.K. (1992). Empirical estimates of Bonferroni corrections for use in chromosome mapping studies with the BXD recombinant inbred strains of mice. Behavior Genetics, 22, 677-684.

Belknap, J.K. & Crabbe, J.C. (1992). Chromosome mapping of gene loci affecting morphine and amphetamine responses in BXD recombinant inbred mice. In P.W. Kalivas & H.H. Samson (Eds.). The Neurobiology of Drug and Alcohol Addiction (pp. 311-323). Ann. N.Y. Acad. Sci.

Belknap, J.K., Crabbe, J.C., Riggan, J., & O'Toole, L.A. (1993). Voluntary consumption of morphine in 15 inbred mouse strains. Psychopharmacology, 112, 352-358.

Belknap, J.K., Crabbe, J.C., & Young, E.R. (1993). Voluntary consumption of ethanol in 15 inbred mouse strains. Psychopharmacology, 112, 503-510.

Berrettini, W.H., Ferraro, T.N., Alexander, R.C., Buchberg, A.M., & Vogel, W.H. (1994). Quantitative trait loci mapping of three loci controlling morphine preference using inbred mouse strains. Nature Genetics, 7, 54-58.

Bhakthavatsalam, P., Kamatchi, G.L., & Ghosh, M.N. (1992). Dissociation of receptor sensitivity changes in rat perifornical hypothalamus; a role for dopaminergic receptors in amphetamine anorexic tolerance. Journal Pharmacology and Experimental Therapeutics, 60(3-4), 250-251.

Blake, R.L. (1970). Regulation of liver tyrosine aminotransferase activity in inbred strains and mutant mice, I.strain variance in fasting enzyme levels. International Journal of Biochemistry, 1, 361-370.

Blizard, D.A. (1992). Recombinant-inbred strains: general methodological considerations relevant to the study of complex characters. Behavior Genetics, 22, 621-634.

Blum, K., Noble, E.P., Sheridan, P.J., Montgomery, A., Ritchie, T., Kagadeeswaran, P., Nogami, H., Briggs, A.H., & Cohn, J.B. (1990). Allelic association of human dopamine D2 receptor gene in alcoholism. Journal of the American Medical Association, 263, 2055-2060.

Blundell, J. E., & Latham, C.J. (1982). Behavioral pharmacology of feeding. In T. Silverstone (Ed.). Drugs and appetite (pp. 41-80). London: Academic Press

Blundell, J. (1991). Pharmacological approaches to appetite suppression. Trends in Pharmacological Sciences, 12, 147-157.

Blundell, J.E., & Latham, C.J. (1980). Characterisation of adjustments to the structure of feeding behavior following pharmacological treatment: effects of amphetamine and fenfluramine and the antagonism produced by pimozide and methergoline. Pharmacology, Biochemistry and Behavior, 12, 717-722.

Blundell, J. E., & Barridge, S.L. (1979). Control of feeding and the pharmacology of anorexic drugs. In J. Munro (ed.). The Treatment of Obesity (pp. 1-3). Lancaster: M.T.P. press

Blundell, J.E., & Barridge, S.L. (1979). Serotonergic influences on food intake: effect of 5-hydroxtryptophan on parameters of feeding behavior in deprived and free-feeding rats. Pharmacology, Biochemistry and Behavior, 11, 341-438.

Botstein, D., White, R.L., Skolnick, M., & Davis, R.W. (1980). Construction of a genetic linkage map in man using restriction fragment length polymorphisms. American Journal of Human Genetics, 32, 314-331.

Buck, K.J., Metten, P., Belknap, J.K., & Crabbe, J.C. (submitted). Loci influencing genetic predispositions to acute alcohol dependence and withdrawal mapped to murine chromsome 1, 4, and 11.

Cadoret, R.S., O'Gorman, T., Troughton, E., & Heywood, E. (1986). An adoption study of genetic and environmental function in drug abuse. Archive of General Psychiatry, 43, 1131-1136.

Carr, G.D. & White, N.M. (1986). Contributions of dopamine terminal areas to amphetamine-induced anorexia and adipsia. Pharmacology Biochemistry and Behavior, 25, 17-22.

Caul, W.F., Jones, J.R., & Barrett, R.J. (1988). Amphetamine's effects on food consumption and body weight: the role of adaptive processes. Behavioral Neuroscience, 102, 441-450.

Cho, A.K. (1990). Ice: a new dosage form of an old drug. Science, 249, 631-634.

Cole, S.O. (1980). Deprivation-depedent effects of amphetamine on concurrent measures of feeding and activity. Pharmacology, Biochemistry and Behavior, 12, 723-727.

Cooper, S.J., Francis, J., & Barber, D.J., (1993). Selective dopamine d-1 receptor agonists, SK&F 38393 and CY 208-243 reduce sucrose sham-feeding in the rat. Neuropharmacology, 32, 101-102.

Cooper, J.R., Bloom, F.E. & Roth, R.H. (1992). Dopamine. In J.R. Cooper, F.E. Bloom & R.H. Roth (Eds.). The Biochemical of Neuropharmacology 6th ed. New York: Oxford University Press, Inc.

Cooper, S.J., Frances, J., & Rusk, I.N. (1990). The anorectic effect of SK&F 38393, a selective dopamine D1 receptor agonist: a microstructural analysis of feeding and related behavior. Psychopharmacology, 100, 182-187.

Copeland, N.G., Jenkins, N.A., Gilbert, D.J., Eppig, J.T., Maltais, L.J., Miller, J.C., Dietrich, W.F., Weaver, A., Lincoln, S.E., Steen, R.G., Stein, L.D., Nadeau, J.H., & Lander, E.S. (1993). A genetic linkage map of the mouse: current applications and future prospects. Science, 262, 57-66.

Crabbe, J.C., Kosobud, A., & Young, E.R. (1983). Genetic selection for ethanol withdrawal severity: difference in replicate mouse lines. Life Sciences, 33, 955-962.

Crabbe, J.C., Jarvik, L.F., Liston, E.H., & Jenden, D.J. (1983). Behavioral responses to amphetamines in identical twins. Acta Genetica Medical Gemellol, 32, 139-149.

Crabbe, J.C. & Harris, R.A. (Eds.). The genetic basis of alcohol and drug actions. New York: Plenum Press, 1991

de Wit, H., Uhlenhuth, E.H., & Johanson, C.E. (1986). Individual differences in the reinforcing and subjective effects of amphetamine and diazepam. Drug and Alcohol Dependence, 16, 341-360.

Devor, E.J. & Cloninger, C.R. (1992). Genetics of Alcoholism. Annual Review of Genetics, 23, 19-36.

Dietrich, W., Katz, H., Lincoln, S.E., Shin, H.-S., Friedman, J., Dracopoli, N.C., & Lander, E.S. (1992). A genetic map of the mouse suitable for typing intraspecific crosses. Genetics, 131, 423-427.

Dobrzanski, S., & Doggett, N.S. (1976). The effect of (+)-amphetamine and fenfluramine on feeding in starved and satiated mice. Psychopharmacology, 48, 283-286.

Duterte-Boucher, D., Kamenka, J.M., & Costentin, J. (1990). Comparison of the effects of three indirect dopamine agonists, GK 13, GBR 12783 and dexamphetamine on behavioral tests involving central catecholminergic transmissions. Psychopharmacology, 101, 344-353.

Eichler, A.J., Antelman, S.M., & Black, C.A. (1980). Amphetamine stereotypy is not a homogeneous phenomenon: sniffing and licking show distinct profiles of sensitization and tolerance. Psychopharmacology, 68, 287-290.

Fibiger, H.C., & Phillips, A.G. (1986). Reward, motivation, cognition: psychobiology of mesotelencephalic dopamine systems. In F.E.Bloom (ed.). Handbook of physiology, Vol 4 (pp 647-675). Bethesda. American Physiology Society

Fischer, R.A. (1958). Statistical Methods for research workers. New York: Hafner Publishing Co.

Flint J., Corley, R., DeFries, J.C., Fulker, D.W., Gray, J.A., Miller, S., & Collins, A.C. (1995). A simple genetic basis for a complex psychological trait in laboratory mice. Science, 269, 1432-1435.

Gannon, K.S., Smith, J.C. Henderson, R., & Hendrick, P. (1992). A system for studying the microstructure of ingestive behavior in mice. Physiology and Behavior, 51, 515-521.

Gannon, K.S. (1995). Personnal communication

Gilbert, D.B. & Cooper, S.J. (1985). Analysis of dopamine D1 and D2 receptor involvement in d- and l-amphetamine-induced anorexia in rats. Brain Research Bulletin, 15, 385-389.

Goodall, E., Trenchard, E., & Silverstone, T. (1987). Receptor blocking drugs and amphetamine anorexia in human subjects. Psychopharmacology, 92, 484-490.

Gora-Maslak, G., McClearn, G.E., Crabbe, J.C., Phillips, T.J., Belknap, J.K., & Plomin, R. (1991). Use of recombinant inbred strains to identify quantitative trait loci in psychopharmacology. Psychopharmacology, 104, 413-424.

Green, E.L. (1981). Genetics and Probability in Animal Breeding Experiments. New York: Oxford Press.

Gray, T., & Wise, R. (1980) Effect of pimozide on lever pressing behavior maintained on an intermittent reinforcement schedule. Pharmacology Biochemistry and Behavior, 12, 931-935.

Goeders, N. E., & Smith J. (1983). Cortical dopaminergic involvement in cocaine reinforcement. Science, 221, 773-775.

Hernandez, L., & Hoebel, B.G. (1988). Food reward and cocaine increases dopamine metabolism in the rat brain. Physiology Behavior, 55, 541-546.

Hernandez, L., & Hoebel, B.G. (1990). Feeding can enhance dopamine turnover in the prefrontal cortex. Brain Research Bulletin, 25, 975-979.

Hicks, P., Straong, J.C., & Samarojski, T. (1980). Aging alters amphetamine-induced stereotyped gnawing and neostriatl elimination of amphetamine ine mice. Life Sciences, 27, 715-722.

Hilbert, P., Lindpainter, K., Beckmann, J.S., Serikawa, T., Soubrier, F., Dubay, C., Cartwright, P., DeGouyon, B., Julier, C., Takahasi, S., Vincent, M., Ganten, D., Georges, M., & Lathrop, G.M. (1991). Chromosomal mapping of two genetic loci associated with blood-pressure regulation in hereditary hypertensive rats. Nature, 353, 521-529.

Hoebel, B.G., Hernandez, L., Schwartz, D.H., Mark, G.P. & Hunter, G.A. (1989). Microdialysis studies of brain norepinephrine, serotonin, and dopamine release during ingestive behavior: theoretical and clinical implications. In L.H. Schneider & S.J. Cooper (Eds.). The Psychobiology of Human Eating Disorders: Preclinical and Clinical Perspectives (pp. 171-193). New York: Annals of the New York Academy of Science

Hoebel, B.G., & Leibowitz. S.F. (1981). Brain monoamines in the modulation of self-stimulation, feeding, and body weight. In H. Weiner, M.A. Hofer, & A.J. Stunkard (Eds.). Brain, Behavior, and Bodily Disease (pp. 103-142). New York: Raven Press

Hooks, M.S., Jones, G.H., Neill, D.B., & Justice, J.R. (1991). Individual differences in amphetamine sensitization: dose-dependent effects. Pharmacology Biochemistry and Behavior, 41, 203-210.

Ikemoto, S., & Panksepp, J. (1994). The relationship between self-stimulation and sniffing in rats: does a common brain system mediate these behaviors? Behavioral Brain Research, 61, 143-162.

Inoue, K., Kiriike, N., Okuno, M., Ito, H., Fujisaki, Y. Matsui, T., & Kawakita, Y. (1993). Scheduled feeding caused activation of dopamine metabolism in the striatum of rats. (1993). Physiology and Behavior, 53, 177-181.

Jacob, H.J., Lindpainter, K., Lincoln, S.E., Kusumi, K., Bunker, R.K., Mao, V.P., Ganten, D., Dzau, V.J., & Lander, E.S. (1991). Genetic mapping of a gene causing hypertension in the stroke-prone spontaneously hypertensive rat. Cell, 67, 213-224.

Jhanwar-Uniyal, M., Darwish, M., Levin, B., & Leibowitz, S.F. (1987). Alterations in catecholamine levels and turnover in discrete brain areas after food deprivation. Pharmacology, Biochemistry and Behavior, 26, 271-275.

Jones, J.R., & Caul, W.F. (1989). Time course of the effect of amphetamine on eating is biphasic. Behavioral Neuroscience, 103, 1047-1052.

Justice, M.J., & Stephenson, D.A. (1994). Mouse chromosome 13. Mammalian Genome, 5, s196-s206.

Kitahama, K. & Valatx, J. (1979). Strain differences in amphetamine sensitivity in mice; I. A diallel analysis of open field activity. Psychopharmacology, 66, 189-194.

Koob, G.F., & Bloom, F.E. (1988). Cellular and molecular mechanisms of drug dependence. Science, 242, 715-723.

Lander, E., Green, R., Abrahamson, J., Barlow, A., Daley, M., Lincoln, S., & Newburg, L. (1987). MAPMAKER: An interactive computer package for constructing primary genetic linkage maps of experimental and natural populations. Genomics, 1, 174-181.

Lander, E.S., & Botstein, D. (1989). Mapping mendelian factors underlying quantitative traits using RFLP linkage maps. Genetics, 121, 185-189.

Lander, E., & Kruglyak, L. (1995). Genetic dissection of complex traits: guidelines for interpreting and reporting linkage results. Nature Genetics, 11, 241-247.

Lander, E., & Schork, N.J. (1994). Genetic dissection of complex traits. Science, 265:2037-2048.

Leibowitz, S.F. (1975). Catecholaminergic mechanisms of the lateral hypothalamus: their role in the mediation of amphetamine anorexia. Brain Research, 98, 529-545.

Leibowitz, S.F. (1975). Amphetamine: possible site and mode of action for producing anorexia in the rat. Brain Research, 84, 160-167.

Leibowitz, S.F. (1978). Identification of catecholamine receptor mechanisms in the perifornical lateral hypothalamus and their role in mediating amphetamine and L-Dopa anorexia. In S. Garattini and R. Samanin (Eds.). Central mechanisms of anorectic drugs (pp. 39-82). New York: Raven Press

LeMagnen, J. The sensory stimulation to eat and not to eat. Neurobiology of feeding and nutrition. San Diego: Academic Press, Inc

Lewin, B. (1994). Genes V. New York: Oxford Unversity Press

Levitsky, D.A., Strupp, B.J., & Lupoli, J. (1981). Tolerance to anorectic drugs: pharmacological or artifactual. Pharmacology Biochemistry and Behavior, 14, 661-667.

Link, R.E., Kobilka, B.K., & Barsh, G.S. (1993). Linkage mapping of α-2 adrenergic receptor genes to mouse chromosomes 2 and 5. Mammalian Genome, 4, 650-655.

Lincoln, S., Daly, M. & Lander, E. (1988). Constructing Genetic Maps with MAPMAKER/EXP 3.0.. Whitehead Institute Technical Report. 3rd edition.

Lincoln, S., Daly, M. & Lander, E. (1992). Mapping Genes Controlling Quantitative Traits ith MAPMAKER/QTL 1.1. Whitehead Institute Technical Report. 2nd edition.

Logan, L., Seale, T.W., Cao, W., & Carney, J.M. (1989). Effects of chronic amphetamine in BALB/cBy mice, a strain that is not stimulated by acute administration of amphetamine. Pharmacology Biochemistry and Behavior, 31, 675-682.

Louilot, A., Gpmza;es-Mora, J.L., Guadalupe, L., Mas, M. (1991). Sex-related olfactory stimuli induce a selective increase in dopamine release in the nucleus accumbens of male rats. A voltametric study. Brain Research, 553, 313-317.

Lynch, J. & House, M.A. (1992). Cardiovascular effects of methamphetamine. Journal Cardiovascular Nursing, 6(2), 12-18.

Lyons, M., & Robbins, T.W. (1975). The action of central nervous system stimulant drugs: a general theory concerning amphetamine effects. In W. Essman, L. Valzelli (eds.). Current Developments in psychopharamcology, Vol 2. (Pp. 79-163). New York: Spectrum

McClearn, G.E. & Kakihana, R. (1973). Selective breeding for ethanol sensitivity in mice. Behavior Genetics, 3, 409-410.

McGuirk, J., Goodall, E., Silverstone, T., & Willner, P. (1991). Differential effects of *d*-fenfluramine, *l*-fenfluramine and *d*-amphetamine on microstructure of human eating behavior. Behavioral Pharmacology, 2, 113-119.

McGuirk, J., Silverstone, T. (1990). The effect of the 5-HT re-uptake inhibitor fluoxetine on food intake and body weight in healthy male subjects. International Journal of Obesity, 14, 361-372.

Maltias, L.J., Lane, P.W., & Beamer, W.G. (1984). Anorexia, a recessive mutation causing starvation in preweanling mice. The Journal of Heredity, 75, 468-472.

Martel, P., & Fantino, M. (1996). Mesolimbic dopaminergic system activity as a function of food reward: a microdialysis study. Pharmacology, Biochemistry and Behavior, 53, 221-226.

Martin-Iverson, M.T. & Dourish, C.T. (1988). Role of dopamine D-1 and D-2 receptor subtypes in mediating dopamine agonist effects on food consumption in rats. Psychopharmacology, 96, 370-374.

Melega, W.P., Williams, A.E.,Schmitz, D.A., Distefano, E.W., & Cho, A.K. (1995). Pharmacokinetic and pharmacodynamic analysis of the actions of *D*-amphetamine and *D*-methamphetamine on the dopamine terminal. The Journal of Pharmacology and Experimental Therapeutics, 274, 90-96.

Mora, F., & Meyers, R.D. (1977). Brain self-stimulation: direct evidence of the involvement of dopamine in prefrontal cortex. Science, 197, 1387-1389.

Mueller K., Hollingsworth, E.M., & Cross, D.R. (1989). Another look at amphetamine-induced stereotyped locomotor activity in rats using a new statistic to measure locomotor stereotopy. Psychopharmacology, 97, 74-79.

Nurnberger, J.L., Gershon, E.S., Simmons, S., Ebert, M., Kessler, L.R., Dibble, E.D., Jimerson, S.S., Broun, G.M., Gold, P., Jimerson, D.C., Guroff, J., & Storch, F.I. (1982). Behavioral, biochemical and neuroendocrine response to amphetamine in normal twins, and "well-state" bipolar patients. Psychoneuroendocrinology, 7, 163-176.

Parada, M., Hernandez, L., Schwartz, D., & Hoebel, B.G. (1988). Hypothalamic infusion of amphetamine increases serotonin, dopamine and norepinephrine. Physiology Behavior, 44, 607-610.

Paulus, M.P., Callaway, C.W., & Geyer, M.A. (1993). Quantitative assessment of the microstructure of rat behavior: II. Distinctive effect of dopamine releasers and uptake inhibitors. Psychopharmacology, 113, 187-198.

Petit, H. O. & J.B. Justice (1991). Effect of dose on cocaine self-administration behavior and dopamine levels in the nucleus accumbens. Brain Research, 539, 94-102.

Phillips, T.J., Belknap, J.K., & Crabbe, J.C. (1992). Use of voluntary morphine consumption in recombinant inbred strains to access vulnerability to drug abuse at the genetic level. Journal of Addiction Disorders, 10, 29-44.

Phillips, T.J. & Crabbe J.C. (1991). Behavioral studies of genetic differences in alcohol action. In Crabbe, J.C. & R.A. Harris (Eds.). The genetic basis of alcohol and drug actions (pp 25-104). New York: Plenum Press

Phillips, T.J. (1994). Personal communication.

Plomin, R., DeFries, J.C. & McClearn, G.E. (1990). Behavioral Genetics: A Primer. New York: W.H. Freeman.

Plomin, R., McClearn, G.E., & Gora-Maslak, G. (1991). Quantitative trait loci and psychopharmacology. Journal of Psychopharmacology, 5, 1-9.

Pothos, E.N., Creese, I., & Hoebel, B.G. (1995). Restricted eating with weight loss selectively decreases extracellular dopamine in the nucleus accumbens and alters dopamine response to amphetamine, morphine and food intake. The Journal of Neuroscience, 15(10), 6640-6650.

Prinssen, E., Balestra, W., Bemeimans, F., & Cools, A.R. (1994). Evidence of a role of the shell of the nucleus accumbens in oral behavior of freely moving rats. The Journal of Neuroscience, 14(3), 1555-1562.

Radhakishum, F.S., van Ree, J.M., & Westerink, B.H. (1988). Scheduled eating increases dopamine release in the nucleus accumbens of food-deprived rats as assessed with on-line dialysis. Neuroscience Letters, 85, 351-356.

Rise, M.L., Frankel, W.N., Coffin, J.M., & Seyfried, T.N. (1991). Genes for epilepsy mapped in the mouse. Science, 253, 669-673.

Rose, E.A. (1991). Applications of the polymerase chain reaction to genome analysis. FASEB Journal, 5, 46-54.

Rusk, I.N. & Cooper, S.J. (1989). Microstructural analysis of the anorectic effect of N-0437, a highly selective dopamine D2 agonist. Brain Research, 494, 350-358.

Saiki, R.K., Scharf, S., Faloona, F., Mullis, K.B., Horn, G.T., Erlich, H.A., & Arnheim, N. (1985). Enzymatic amplification of B-globin genomic sequences and restriction site analysis for diagnosis of sickle cell anemia. Science, 230, 1350-1354.

Samanin, R., Bernasconi, S., & Garattini, S. (1975). The effect of selective lesioning of brain catecholamine-containing neurons on the activity of various anorectics in the rat. European Journal of Pharmacology, 34, 373-375.

Samanin, R., & Garattini, S. (1990). The pharmacology of serotoninergic drugs affecting appetite. In R.J. Wurtman and J.J. Wurtman (Eds.). Nutrition and the brain (pp. 163-188). New York: Raven Press

Samanin, R., & Garattini, S. (1993). Neurochemical mechanisms of action of anorectic drugs. Pharmacology and Toxicology, 73, 63-68.

Sannerud, C.A., Brady, J.V., & Griffiths, R.R. (1989). Self-injection in baboons of amphetamines and related designer drugs, NIDA Research Monogram, 94, 30-42.

Seale, T.W., Carney, J.M., Johnson, P., & Rennert, O.M. (1985). Inheritance of amphetamine-induced thermoregulatory response in inbred mice. Pharmacology, Biochemistry and Behavior, 23, 373-377.

Seale, T.W. (1991). Genetic differences in response to cocaine and stimulant drugs. In J.C. Crabbe & R.A. Harris (Eds.). The Genetic Basis of Alcohol and Drug Actions (pp. 279-321). New York: Plenum Press

Seale, T.W., McLanahan, K., Johnson, P., Carney, J.M., & Rennert, O.M. (1984). Systematic comparison of apomorphine-induced behavioral changes in two mouse strains with inherited differences in brain dopamine receptors. Pharmacology Biochemistry and Behavior, 21, 237-244.

Segal, D. (1975). Behavioral characterization of *d*- and l-amphetamine: neurochemical implications. Science, 190, 475-477.

Sills, T.L., Baird, J.P., & Vaccarino, F.J. (1993). Individual differences in the feeding effects of amphetamine: role of nucleus accumbens dopamine and circadian factors. Psychopharmacology, 112, 211-218.

Sills, T.L., & Vaccarino, F.J. (1991). Facilitation and inhibition of feeding by a single dose of amphetamine: relationship to baseline intake and accumbens cholecystokinin. Psychopharmacology, 105, 329-334.

Silver, L.M. (1992). Bouncing off microsatellites. Nature Genetics, 2, 8-9.

Silver, L.M. (1995). Mouse Genetics, Concepts and Applications. New York: Oxford University Press

Soller, M., Brody, T., & Genizi, A. (1976). On the power of experimental designs for the dection of linkage between marker loci and quantitative loci in crosses between inbred lines. Theoretical Applied Genetics, 47, 35-39.

Sokal, R.R. & Rohl, F. (1981). Biometry. San Francisco: Freeman Press

Starr, B.S., & Starr, M.S. (1987). Behavioral interactions involving D1 and D2 dopamine receptors in no-habituated mice. Neuropharmacology, 26, 613-619.

Surgue, M.F. (1987). Neuropharmacology of drugs affecting food intake. Pharmacologic Therapeutics, 32, 145-182.

Taylor, B. & Reifsnyder, P.C. (1993). Typing recombinant inbred mouse strains for microsatellite markers. Mammalian Genome, 4, 239-242.

Taylor, B.A. (1978). Recombinant inbred strains: use in gene mapping. In H.C. Morse (Ed.). Origins of Inbred Mice (pp. 423-438). New York: Academic Press

Terry, P., & Katz, J.L. (1994). A comparison of the effects of the D_1 receptor antagonists SCH 23390 and SCH39166 on suppression of feeding behavior by the D_1 agonists SKF38393. Psychopharmacology, 113, 328-333.

Terry, P., & Katz, J.L. (1992). Differential antagonism of the effects of dopamine d_1-receptor agonists on feeding behavior in the rat. Psychopharmacology, 109, 403-409.

Thompson, M.W., McInnes, R.R. & Willard, H.F. (1991). Genetics In Medicine. Philadelphia: W.B. Saunders Co..

Timberlake, W., & Lucas, G.A. (1989). Behavior systems and learning: from misbehavior to general principles. In S.B. Klein and R.R. Mowrer (Eds.). Contemporary Learning Theories. New York: Academic Press

Todd, J.A, Aitman, T.J., Cornall, R.J., Ghosh, S., Hall, J.R.S., Hearne, C.M., Knight, A.M., Lo, J.M., McAleer, M.A., Prins, J., Rodrigues, N., Lathrop, M., Pressey, A., DeLarato, N.H., Peterson, L.B., & Wic, L.S. (1991). Genetic analysis of autoimmune type 1 diabetes mellitus in mice. Nature, 351, 542-547.

Tolstoi, L.G. (1989). The role of pharmacotherapy in anorexia nervosa and bulimia. Perspectives in Practice, 89, 1640-1646.

Towell, A., Muscat, R., & Willner, P. (1988). Behavioral microanalysis of the role of dopamine in amphetamine anorexia. Pharmacology Biochemistry and Behavior, 30, 641-648.

Towell, A., Willner, P. & Muscat, R. (1988). Apomorphine anorexia: the role of dopamine receptors in the ventral forebrain. Psychopharmacology, 96, 135-141.

Uhl, G.R., Persico, A.M., & Smith, S. (1992). Current excitement with D_2 dopamine receptor gene alleles in substance abuse. Archives General Psychiatry, 49, 157-160.

Ujike, H., Akiyama, K., & Otsuki, S. (1990). D-2 but not D-1 dopamine agonists produce augmented behavioral response in rats after subchronic treatment with methamphetamine or cocaine. Psychopharmacology, 102, 459-464.

Ungerstedt, U. (1971). Adipsia and aphagia after 6-hydroxydopamine induced generation of the nigrostriatal dopamine system. Acta Physiology Scandinavian Supplement, 367, 95-122.

Van der Hoek, G.A., & Cooper S.J. (1994). The selective dopamine uptake inhibitor GBR 12909: its effects on the microstructure of feeding in rats. Pharmacology, Biochemistry and Behavior, 48, 135-140.

Vasse, M., Chagraoui, A., & Protais, P. (1988). Climbing and stereotyped behaviors in mice require the stimulation of D1 dopamine receptors. European Journal of Pharamcology, 148, 221-229.

Weber, J.L. & May, P.E. (1989). Abundant class of human DNA polymorphisms which can be typed using the polymerase chain reaction. American Journal of Human Genetics, 44, 388-396.

Weinshilbom, R.M., Otterness, D.M., Nobuhiko, T., & Taylor, B.A. (1992). *Tpmt* and *Ly-28*: location on mouse chromosome 13. Mouse Genome, 90, 446-448.

Wellman, P.J. (1992). Overview of adrenergic anorectic agents. American Journal of Clinical Nutrition, 55, 193S-198S.

Wilke, T.M., Chen, Y., Gilbert, D.J., Moore, K.J., Yu, L., Simon, M.I., Copeland, N.G., & Jenkins, N.A. (1993). Identifcation, chromosomal location, and genome organization of mammalian G-protein-coupled receptors. Genomics, 18, 175-184.

Willner, P., & Towell, A. (1982). Microstructural analysis of the involvement of beta-receptors in amphetamine anorexia. Pharmacology, Biochemistry and Behavior, 17, 255-262.

Wise, R.A. (1982). Common nerual basis for brain stimulation reward, drug reward and food reward. In B.G. Hoebel & D. Novin (Eds.). Neural basis of feeding and reward (pp 445-454). Brunswick. Haer Institute

Woolverton, W.L. & Virus, R.M. (1989). The effects of a D1 and a D2 dopamine antagonist on behavior maintained by cocaine and food. Pharmacology Biochemistry and Behavior, 32, 691-697.

APPENDIX. QTL ANALYSIS

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N		PCTWRED	P2ALPHA	P2N		AVGBASE1	P3ALPHA	P3N	
One	1	D1Ncvs72	B457		-0.249	0.2637	22		0.023	0.919	22		-0.2366	0.2892	22	
One	2	D1Mit1	L33		-0.5145	0.0289	18	X	-0.406	0.0945	18		0.2319	0.3545	18	
One	3	Odc-rs10			-0.3777	0.0688	24		-0.0488	0.8208	24		-0.0833	0.6987	24	
One	4	Uqtla1-rs			-0.1937	0.3644	24		-0.02	0.9261	24		-0.0277	0.8976	24	
One	5	D0Byu25			0.0916	0.6702	24		-0.1887	0.3771	24		0.0055	0.9797	24	
One	6	Gst2-rs			-0.0266	0.9019	24		-0.2459	0.2468	24		0.0781	0.7168	24	
One	7	D1Ncvs35	D40		0.352	0.0995	23		-0.0304	0.8905	23		0.007	0.9749	23	
One	8	Aox1	Aox-1		0.0724	0.7367	24		0.24	0.2586	24		-0.039	0.8565	24	
One	9	D1Ncvs36	B260		0.1016	0.6447	23		0.2361	0.2782	23		-0.0228	0.9177	23	
One	10	D1Ncvs37	D165		0.0341	0.8772	23		0.1946	0.3736	23		0.0371	0.8667	23	
One	11	D1Ncvs38	D360		0.1029	0.6571	21		0.2456	0.2832	21		0.0657	0.7774	21	
One	12	Idh1	Idh-1		0.0045	0.9832	24		0.1996	0.3498	24		0.0178	0.9343	24	
One	13	Gln3-5			0.0045	0.9832	24		0.1996	0.3498	24		0.0178	0.9343	24	
One	14	D1Rti2			0.018	0.9383	21		0.2114	0.3577	21		0.0416	0.8578	21	
One	15	Cryg	Len-1		0.0045	0.9832	24		0.1996	0.3498	24		0.0178	0.9343	24	
One	16	Iap1s1-2			0.0045	0.9832	24		0.1996	0.3498	24		0.0178	0.9343	24	
One	17	D1Mit5	L20		0.1191	0.617	20		0.0337	0.8877	20		0.1416	0.5516	20	
One	18	D1Ncvs73	B564		0.0538	0.8027	24		0.1913	0.3706	24		-0.0186	0.9313	24	
One	19	D1Ncvs74	D563		0.0538	0.8027	24		0.1913	0.3706	24		-0.0186	0.9313	24	
One	20	Mtap2	Mtap-2		-0.0433	0.8562	20		0.1362	0.5671	20		-0.0448	0.8511	20	
One	21	D1Ncvs39	D100		0.082	0.7167	22		0.2103	0.3475	22		-0.111	0.6229	22	
One	22	D1Ncvs40	B101		0.1211	0.5915	22		0.2138	0.3394	22		-0.2032	0.3644	22	
One	23	Mylf	Mlc-f, MLC1,3F		0.1533	0.4958	22		0.1855	0.4086	22		-0.1303	0.5632	22	
One	24	D1Byu1			0.1247	0.5614	24		0.2235	0.2939	24		-0.0971	0.6516	24	
One	25	D1Mit19	L86		0.2954	0.206	20		0.2419	0.3041	20		0.0051	0.9829	20	
One	26	Tnp1	Tp-1		0.3491	0.0945	24		0.0899	0.6761	24		0.0692	0.748	24	
One	27	D1Mcg2			0.2992	0.1556	24		0.0778	0.7178	24		0.0643	0.7654	24	
One	28	Des	D1Mcg7		0.2992	0.1556	24		0.0778	0.7178	24		0.0643	0.7654	24	
One	29	Lsh			0.2992	0.1556	24		0.0778	0.7178	24		0.0643	0.7654	24	
One	30	Vil			0.2992	0.1556	24		0.0778	0.7178	24		0.0643	0.7654	24	
One	31	D1Mit7	A80		0.2992	0.1556	24		0.0778	0.7178	24		0.0643	0.7654	24	
One	32	Inha			0.2992	0.1556	24		0.0778	0.7178	24		0.0643	0.7654	24	
One	33	D1Ncvs41	D67		0.2754	0.2537	19		0.2658	0.2714	19		-0.1855	0.447	19	
One	34	D1Ncvs75	D688		0.2392	0.2604	24		0.2198	0.3022	24		-0.2145	0.3142	24	
One	35	D1Ncvs42	B110		0.2239	0.3045	23		0.2236	0.3051	23		-0.1626	0.4585	23	
One	36	D1Ncvs43	D64		0.2346	0.3336	19		0.0539	0.8267	19		0.1534	0.5306	19	
One	37	Uqtla1	UDPGT-K39/Udpgt		0.0992	0.6448	24		0.1016	0.6366	24		-0.2278	0.2843	24	
One	38	Ncl	DONds28, D1Nds2		0.1879	0.3793	24		0.2277	0.2845	24		-0.1761	0.4105	24	
One	39	Acrg	Achr-3		0.1717	0.4691	20		0.5138	0.0205	20	X	-0.3315	0.1533	20	
One	40	DMit11	M17		0.0339	0.8873	20		-0.0161	0.9461	20		-0.4736	0.0349	20	X
One	41	Bcl2	Bcl-2		-0.1087	0.6131	24		0.246	0.2466	24		-0.4576	0.0245	24	X
One	42	Rnu1-ps1	Rnu1-1/Rnu-1/Ui		-0.0368	0.8645	24		0.3717	0.0737	24		-0.5112	0.0107	24	X
One	43	Emv17	Emv-17		-0.1875	0.3803	24		0.161	0.4523	24		-0.3967	0.0549	24	
One	44	D1Ncvs44	D334		-0.1753	0.4237	23		0.136	0.5361	23		-0.3785	0.0749	23	
One	45	D1Ncvs45	D122		-0.1071	0.6531	20		0.2776	0.2361	20		-0.4255	0.0614	20	
One	46	Inhbb			-0.0645	0.7756	22		0.4122	0.0566	22		-0.39	0.0727	22	
One	47	En1	En-1		-0.107	0.6188	24		0.3641	0.0803	24		-0.3953	0.0559	24	
One	48	D1Ncvs67	D559		0.1392	0.5941	17		0.3488	0.17	17		-0.658	0.0041	17	X
One	49	D1Ncvs68	D658		-0.1445	0.58	17		0.2696	0.2954	17		-0.459	0.0639	17	
One	50	D1Ncvs69	D493		-0.135	0.5492	22		0.3056	0.1667	22		-0.5216	0.0128	22	X
One	51	D1Ncvs76	B492		0.0002	0.9993	21		0.2528	0.2688	21		-0.4363	0.048	21	X
One	52	Iap1s3-1			0.0052	0.9808	24		0.3668	0.0779	24		-0.4992	0.013	24	X
One	53	D0Byu26			0.1618	0.45	24		0.2915	0.167	24		-0.3622	0.082	24	
One	54	D1Ncvs46	D118		0.0348	0.8875	19		0.33	0.1676	19		-0.6265	0.0041	19	X
One	55	Mpmv6	Mpmv-6		-0.0627	0.771	24		0.3244	0.122	24		-0.4408	0.0311	24	X
One	56	D1Byu2			-0.0627	0.771	24		0.3244	0.122	24		-0.4408	0.0311	24	X
One	57	Mpmv16	Mpmv-16		-0.0627	0.771	24		0.3244	0.122	24		-0.4408	0.0311	24	X
One	58	Ubq2	Ubq-2		-0.0627	0.771	24		0.3244	0.122	24		-0.4408	0.0311	24	X
One	59	Ren1	Ren-1		-0.0385	0.8615	23		0.3263	0.1287	23		-0.4718	0.023	23	X
One	60	Ren2	Ren-2		-0.0385	0.8615	23		0.3263	0.1287	23		-0.4718	0.023	23	X
One	61	Pep3	Pep-3		-0.0627	0.771	24		0.3244	0.122	24		-0.4408	0.0311	24	X
One	62	Cfh	Sas-1		0.0321	0.8817	24		0.3229	0.1238	24		-0.3939	0.0569	24	
One	63	D1Ncvs47	B247		-0.032	0.8964	19		0.3359	0.1597	19		-0.2927	0.224	19	
One	64	D1Ncvs48	B268		0.0329	0.8937	19		0.3087	0.1985	19		-0.295	0.2201	19	
One	65	D1Ncvs54	B84		0.0854	0.6984	23		0.4642	0.0257	23	X	-0.3319	0.1218	23	
One	66	D1Ncvs56	D298		0.0575	0.8207	18		0.4331	0.0726	18		-0.4591	0.0553	18	
One	67	D1Ncvs11	B294		0.0854	0.6984	23		0.4642	0.0257	23	X	-0.3319	0.1218	23	
One	68	D1Ncvs57	D109		0.0854	0.6984	23		0.4642	0.0257	23	X	-0.3319	0.1218	23	
One	69	D1Ncvs55	D236		0.1828	0.4155	22		0.4378	0.0416	22	X	-0.2816	0.2042	22	
One	70	D1Ncvs71	D665		0.1189	0.58	24		0.3762	0.07	24		-0.3663	0.0783	24	
One	71	D1Ncvs70	D446		0.145	0.5093	23		0.4933	0.0168	23	X	-0.3032	0.1596	23	
One	72	D1Ncvs77	B447		0.2591	0.2325	23		0.4977	0.0157	23	X	-0.2785	0.1982	23	
One	73	D1Ncvs78	B507		0.1014	0.6451	23		0.4272	0.042	23	X	-0.3929	0.0636	23	
One	74	D1Ncvs51	D103		0.1388	0.5278	23		0.4563	0.0286	23	X	-0.3703	0.082	23	
One	75	Lamb2			0.0735	0.7329	24		0.3465	0.0972	24		-0.4785	0.018	24	X
One	76	D1Pas2			0.128	0.5605	23		0.4595	0.0274	23	X	-0.4352	0.038	23	X
One	77	D1Ncvs52	B265		0.0515	0.8292	20		0.5331	0.0155	20	X	-0.3451	0.1362	20	
One	78	D1Byu7			0.0688	0.7609	22		0.4188	0.0524	22		-0.3338	0.1289	22	
One	79	D1Ncvs12	B139		0.1275	0.6029	19		0.3571	0.1333	19		-0.3228	0.1776	19	
One	80	D1Byu4			0.0142	0.9476	24		0.5046	0.0119	24	X	-0.2934	0.164	24	
One	81	D1Ncvs58	D85		0.0854	0.6984	23		0.4642	0.0257	23	X	-0.3319	0.1218	23	

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N	PCTWRED	P2ALPHA	P2N	AVGBASE1	P3ALPHA	P3N	
One	82	P1Ehs1	E		0.0088	0.969	22	0.4887	0.021	22 X	-0.3953	0.0686	22	
One	83	Ltw4	Ltw-4		0.0786	0.7282	22	0.4396	0.0406	22 X	-0.3844	0.0773	22	
One	84	At3	At-3		0.0856	0.7198	20	0.4759	0.0339	20 X	-0.4556	0.0435	20	X
One	85	D1Byu5			0.0567	0.7923	24	0.4629	0.0227	24 X	-0.3403	0.1037	24	
One	86	D1Byu6			0.0567	0.7923	24	0.4629	0.0227	24 X	-0.3403	0.1037	24	
One	87	D1Ncvs15	B78		-0.0786	0.7281	22	0.4823	0.023	22 X	-0.3195	0.1472	22	
One	88	D1Ncvs50	B283		-0.0144	0.9479	23	0.4676	0.0245	23 X	-0.3829	0.0714	23	
One	89	D1Ncvs53	D284		-0.0144	0.9479	23	0.4676	0.0245	23 X	-0.3829	0.0714	23	
One	90	Lnhr	Ly-22;Sell		-0.0381	0.8597	24	0.4671	0.0214	24 X	-0.3899	0.0596	24	
One	91	Xmmv9	Ehv-9, Xp-20, X		-0.0381	0.8597	24	0.4671	0.0214	24 X	-0.3899	0.0596	24	
One	92	Xmv32	Xmv-32		-0.0381	0.8597	24	0.4671	0.0214	24 X	-0.3899	0.0596	24	
One	93	D1Bir1			-0.0381	0.8597	24	0.4671	0.0214	24 X	-0.3899	0.0596	24	
One	94	P40-rs2	P40-2		-0.0381	0.8597	24	0.4671	0.0214	24 X	-0.3899	0.0596	24	
One	95	Otf1	Oct-1, Otf-1		-0.0381	0.8597	24	0.4671	0.0214	24 X	-0.3899	0.0596	24	
One	96	D1Ncvs49	D13		0.0983	0.6887	19	0.2315	0.3403	19	-0.2137	0.3797	19	
One	97	Cd3z	Tcrz		0.0311	0.9056	17	0.4651	0.06	17	-0.5834	0.014	17	X
One	98	D1Mit16	L46		0.0787	0.7416	20	0.3846	0.0941	20	-0.3528	0.1271	20	
One	99	Pmv24	Pmv-24		0.0598	0.7815	24	0.5213	0.009	24 X	-0.1851	0.3866	24	
One	100	Xmv41	Xmv-41		-0.0916	0.6777	23	0.401	0.0579	23	0.1269	0.564	23	
One	101	Fcgr2	Cd32, Ly-17		-0.0389	0.8707	20	0.3659	0.1126	20	0.0695	0.771	20	
One	102	Mpp			-0.1961	0.3585	24	0.3957	0.0556	24	-0.0435	0.84	24	
One	103	Apoa2	Apoa-2		-0.0389	0.8707	20	0.3659	0.1126	20	0.0695	0.771	20	
One	104	Tal2	Nscl		-0.0859	0.7188	20	0.3611	0.1177	20	0.2238	0.3429	20	
One	105	Ly9	Ly-9		-0.1064	0.6376	22	0.3877	0.0747	22	0.0984	0.6632	22	
One	106	D1Byu3			-0.0722	0.7375	24	0.3967	0.055	24	0.1545	0.4709	24	
One	107	Fcer1g			-0.0964	0.7129	17	0.2381	0.3574	17	-0.0271	0.9179	17	
One	108	D1Byu8			-0.0368	0.8643	24	0.3115	0.1384	24	0.1115	0.6039	24	
One	109	Sap			-0.1217	0.5711	24	0.4106	0.0463	24 X	0.1207	0.5743	24	
One	110	Spna1	Spna-1		0.0289	0.9039	20	0.3756	0.1027	20	0.0703	0.7682	20	
One	111	Mtv7	Mtv-7		-0.0703	0.7442	24	0.4215	0.0402	24 X	0.1254	0.5594	24	
One	112	Adprp			0.0667	0.7801	20	0.2857	0.2221	20	-0.3865	0.0923	20	
One	113	Eph1	Eph-1		0.0333	0.8771	24	0.4191	0.0415	24 X	-0.1042	0.6281	24	
One	114	Xmmv36	Ehv-36, Xmmv-36		0.051	0.8172	23	0.3957	0.0616	23	-0.127	0.5637	23	
One	115	Mpmv25	Xmmv-6, Mpmv-25		0.0333	0.8771	24	0.4191	0.0415	24 X	-0.1042	0.6281	24	
One	116	Lsd			0.0244	0.9098	24	0.406	0.049	24 X	-0.0984	0.6475	24	
One	117	Mls1	Mls-1		0.0079	0.9721	22	0.3337	0.129	22	0.1198	0.5954	22	
One	118	D1Ncvs79	B449		0.0197	0.9445	15	0.626	0.0125	15 X	-0.2735	0.3239	15	
One	119	D1Ncvs80	B613		0.3593	0.0847	24	0.2501	0.2385	24	-0.2041	0.3389	24	
One	120	Mpmv22	Mpmv-22		0.3118	0.138	24	0.2613	0.2174	24	-0.1686	0.4309	24	
One	121	Pmv21	Pmv-21		0.3118	0.138	24	0.2613	0.2174	24	-0.1686	0.4309	24	
One	122	Gstp-rs1			0.3118	0.138	24	0.2613	0.2174	24	-0.1686	0.4309	24	
One	123	D1Byu9			0.3118	0.138	24	0.2613	0.2174	24	-0.1686	0.4309	24	
One	124	D1Mit17	M41		0.505	0.0231	20	X 0.0992	0.6773	20	-0.2055	0.3848	20	
One	125	D1Ncvs59	D113		0.3527	0.1272	20	0.3614	0.1174	20	-0.2352	0.3183	20	
One	126	D1Ncvs60	B34		0.2933	0.1744	23	0.2791	0.1972	23	-0.1924	0.379	23	
One	127	D1Ncvs61	D33		0.2933	0.1744	23	0.2791	0.1972	23	-0.1924	0.379	23	
One	128	D1Ncvs62	B27		0.2933	0.1744	23	0.2791	0.1972	23	-0.1924	0.379	23	
One	129	D1Ncvs63	D25		0.4146	0.0871	18	0.1913	0.447	18	-0.1753	0.4866	18	
Two	130	D2Mit6	L18		0.3431	0.1505	19	0.2147	0.3773	19	-0.1292	0.598	19	
Two	131	D2Ncvs31	B250		0.1429	0.5258	22	-0.1243	0.5815	22	-0.0061	0.9786	22	
Two	132	D2Ncvs32	D303		0.1914	0.3816	23	-0.0678	0.7585	23	-0.048	0.828	23	
Two	133	D2Ncvs46	D670		0.3375	0.1068	24	-0.2444	0.2498	24	-0.0041	0.9849	24	
Two	134	D2Ncvs50	B671		0.3444	0.0994	24	-0.1373	0.5223	24	0.0122	0.955	24	
Two	135	Ass1	Ass-1		0.1728	0.4193	24	0.0075	0.9723	24	-0.156	0.4668	24	
Two	136	Hc	C5		0.0293	0.892	24	0.0279	0.8972	24	-0.3249	0.1214	24	
Two	137	Rmp2			-0.0105	0.9671	18	0.1741	0.4895	18	-0.1182	0.6404	18	
Two	138	Rmp3			0.0386	0.8754	19	0.1098	0.6546	19	-0.051	0.8359	19	
Two	139	Brb13			0.0575	0.8208	18	0.2663	0.2855	18	-0.284	0.2533	18	
Two	140	D2Ncvs51	B506		0.2918	0.1876	22	-0.1939	0.3874	22	-0.2981	0.1779	22	
Two	141	D2Ncvs33	D82		0.2784	0.1984	23	-0.2049	0.3483	23	-0.3072	0.1539	23	
Two	142	D2Mit7	L44		0.2967	0.2039	20	-0.038	0.8737	20	-0.4062	0.0756	20	
Two	143	D2Byu1			0.2881	0.1722	24	-0.2276	0.2847	24	-0.2769	0.1903	24	
Two	144	Neb			0.3562	0.0876	24	-0.2663	0.2085	24	-0.268	0.2055	24	
Two	145	Pmv7	Pmv-7		0.2966	0.1593	24	-0.2363	0.2662	24	-0.3	0.1544	24	
Two	146	D2Mit9	M85		0.0247	0.9177	20	-0.3279	0.1582	20	-0.0157	0.9477	20	
Two	147	Scn2a			-0.0565	0.793	24	-0.4516	0.0267	24 X	-0.0598	0.7815	24	
Two	148	D2Kyo1	Scn2a		-0.0819	0.7102	23	-0.4598	0.0273	23 X	-0.0131	0.9527	23	
Two	149	D2Byu2			-0.0565	0.793	24	-0.4516	0.0267	24 X	-0.0598	0.7815	24	
Two	150	D2Ncvs7	B145		0.0775	0.7251	23	-0.4279	0.0416	23 X	-0.2142	0.3263	23	
Two	151	D2Ncvs34	D117		0.0266	0.9113	20	-0.335	0.1488	20	0.0047	0.9842	20	
Two	152	Iap1s2-8			-0.1103	0.6079	24	-0.293	0.1647	24	-0.0071	0.9739	24	
Two	153	Hoxd	Hox4, Hox4.4, H		-0.0422	0.8447	24	-0.2559	0.2275	24	-0.0657	0.7602	24	
Two	154	D2Mit14	M163		-0.1676	0.4336	24	0.0035	0.987	24	-0.4401	0.0314	24	X
Two	155	D2Ncvs52	B633		-0.1819	0.4701	18	0.3455	0.1602	18	-0.4679	0.0502	18	X
Two	156	D2Mc2	KB1, D2McC2		-0.1516	0.4794	24	0.1061	0.6216	24	-0.416	0.0432	24	X
Two	157	Mdk			-0.0464	0.8334	23	0.1539	0.4832	23	-0.5345	0.0086	23	X
Two	158	D2Nds1	T19		-0.0556	0.7965	24	0.3435	0.1003	24	-0.3037	0.1491	24	
Two	159	D2Byu19	D0Byu19		0.1874	0.3805	24	0.3548	0.0889	24	-0.1405	0.5127	24	
Two	160	D2Mit12	M179		0.1611	0.4975	20	0.0755	0.7519	20	0.0754	0.7521	20	
Two	161	Cd44	Pgp-1, Ly-24		-0.0052	0.9807	24	0.3438	0.1	24	-0.0703	0.7439	24	
Two	162	D2Ncvs36	DI32		-0.1685	0.5483	15	0.2499	0.369	15	0.1519	0.5891	15	

SAG QTL w/ 1994 marker set

analysis run 12-16-94 by SRM

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N	PCTWRED	P2ALPHA	P2N	AVGBASE1	P3ALPHA	P3N	
Two	163	D2Ncvs35	B133		-0.1585	0.5299	18	0.332	0.1783	18	0.1855	0.461	18	
Two	164	D2Ncvs47	D693		0.0037	0.9862	24	0.2789	0.187	24	-0.0291	0.8925	24	
Two	165	D2Ncvs53	B485		0.0519	0.814	23	0.2816	0.1931	23	-0.1615	0.4617	23	
Two	166	D2Mit30	Trh1. D111		0.0518	0.8382	18	0.2011	0.4237	18	-0.1497	0.5532	18	
Two	167	D2Mit17	M246		0.0177	0.9411	20	0.1879	0.4276	20	-0.1493	0.5299	20	
Two	168	Ebp4.2			-0.1	0.6838	19	0.2683	0.2668	19	-0.2079	0.3931	19	
Two	169	B2m	Ly-4. Lym11		-0.1014	0.6373	24	0.3705	0.0747	24	-0.2333	0.2725	24	
Two	170	D2Byu4			-0.1014	0.6373	24	0.3705	0.0747	24	-0.2333	0.2725	24	
Two	171	D2Byu3			-0.0503	0.8154	24	0.3534	0.0902	24	-0.2657	0.2096	24	
Two	172	Hdc			0.1335	0.5341	24	0.3785	0.0682	24	-0.2074	0.3309	24	
Two	173	D2Ncvs37	D243		0.1194	0.6062	21	0.2819	0.2157	21	-0.2119	0.3564	21	
Two	174	D2Ncvs38	B337		0.0769	0.7542	19	0.3346	0.1615	19	-0.0557	0.8209	19	
Two	175	D2Ncvs39	D339		0.1618	0.4608	23	0.3772	0.076	23	-0.1962	0.3695	23	
Two	176	D2Ncvs40	B244		-0.0095	0.9702	18	0.3789	0.121	18	-0.0362	0.8867	18	
Two	177	Il-1a			0.1063	0.6292	23	0.2873	0.1837	23	-0.1963	0.3693	23	
Two	178	D2Ncvs41	D41/B41?		0.1474	0.502	23	0.2765	0.2016	23	-0.2157	0.323	23	
Two	179	Mpmv-28			0.1036	0.6464	22	0.3551	0.1049	22	-0.2079	0.3532	22	
Two	180	D2Bir1			0.2316	0.2761	24	0.2834	0.1795	24	-0.3314	0.1136	24	
Two	181	D2Ncvs54	B490		0.1711	0.435	23	0.2831	0.1905	23	-0.2093	0.3379	23	
Two	182	D2Ncvs55	B540		0.3317	0.1419	21	0.0063	0.9784	21	0.0652	0.7789	21	
Two	183	D2Ncvs56	D541		0.1495	0.5178	21	-0.0079	0.9728	21	0.1656	0.4731	21	
Two	184	D2Byu9	D0Byu9		0.3021	0.1514	24	-0.0491	0.8199	24	-0.0387	0.8576	24	
Two	185	Iapls2-4			0.3021	0.1514	24	-0.0491	0.8199	24	-0.0387	0.8576	24	
Two	186	D2Mc1	KR235, D2McC1		0.3434	0.15	19	-0.1109	0.6513	19	0.124	0.6131	19	
Two	187	D2Ncvs42	B59		0.2647	0.2594	20	0.0766	0.7482	20	0.0872	0.7148	20	
Two	188	D2Ncvs43	D60		0.2647	0.2594	20	0.0766	0.7482	20	0.0872	0.7148	20	
Two	189	D2Ncvs44	D137		0.1646	0.4528	23	-0.0329	0.8814	23	0.2098	0.3365	23	
Two	190	Odc-rs2			0.1976	0.3547	24	-0.047	0.8275	24	0.2241	0.2926	24	
Two	191	Psp			0.1976	0.3547	24	-0.047	0.8275	24	0.2241	0.2926	24	
Two	192	D2Ncvs48	D479		0.1468	0.5143	22	-0.0472	0.8348	22	0.2131	0.341	22	
Two	193	Src			0.2074	0.3308	24	0.147	0.4932	24	0.2257	0.289	24	
Two	194	D2Ncvs49	D608		0.2318	0.2992	22	-0.0227	0.92	22	0.1325	0.5568	22	
Two	195	Svp1	Svp-1		0.1749	0.4137	24	-0.0176	0.9348	24	0.0453	0.8335	24	
Two	196	D2Ncvs45	D177		0.1201	0.5852	23	0.0734	0.7394	23	0.073	0.7405	23	
Two	197	D2Ncvs57	D624		0.0904	0.6745	24	0.0811	0.7065	24	0.0544	0.8005	24	
Two	198	Iapls3-3			0.0904	0.6745	24	0.0811	0.7065	24	0.0544	0.8005	24	
Two	199	Pmv33	Pmv-33		0.0904	0.6745	24	0.0811	0.7065	24	0.0544	0.8005	24	
Three	200	Car2	Car-2		-0.0242	0.9106	24	-0.0642	0.7656	24	0.1326	0.5369	24	
Three	201	Ap2			-0.0327	0.8823	23	-0.037	0.8671	23	0.1755	0.4231	23	
Three	202	D3Byu1			-0.0242	0.9106	24	-0.0642	0.7656	24	0.1326	0.5369	24	
Three	203	D3Byu2			-0.0242	0.9106	24	-0.0642	0.7656	24	0.1326	0.5369	24	
Three	204	D3Ncvs38	D662		-0.0642	0.7766	22	-0.0333	0.8829	22	0.2024	0.3663	22	
Three	205	D3Byu3			0.2343	0.2704	24	-0.0102	0.9621	24	0.2216	0.298	24	
Three	206	D3Mc1	KAB4		0.1576	0.4725	23	-0.0037	0.9865	23	0.0739	0.7374	23	
Three	207	D3Ncvs24	B68		0.2088	0.3512	22	-0.0052	0.9816	22	0.0975	0.6661	22	
Three	208	D3Ncvs42	D547		-0.0208	0.9233	24	-0.0646	0.7644	24	0.0186	0.9311	24	
Three	209	D3Ncvs1	D74, 009		-0.123	0.5953	21	0.0006	0.9979	21	0.0422	0.8558	21	
Three	210	D3Mit21	I1-2. D31		-0.23	0.3293	20	0.1446	0.543	20	0.0081	0.9729	20	
Three	211	Il2	Il-2		-0.1072	0.6182	24	-0.0388	0.8573	24	-0.0026	0.9903	24	
Three	212	Evi1	Evi-1		0.1169	0.5864	24	-0.0678	0.753	24	0.2349	0.2693	24	
Three	213	D3Pas1			-0.1533	0.4851	23	0.2223	0.3079	23	0.0436	0.8432	23	
Three	214	D3Mit5	M123		-0.1544	0.5158	20	0.0299	0.9004	20	0.1591	0.5028	20	
Three	215	D3Ncvs25	B245		0.0035	0.9875	23	-0.0531	0.8097	23	0.1638	0.4551	23	
Three	216	D3Ncvs26	D246		-0.0489	0.8288	22	-0.0526	0.8163	22	0.1424	0.5272	22	
Three	217	Rnu1b1	U1-8		-0.2055	0.3588	22	0.2219	0.321	22	-0.0166	0.9416	22	
Three	218	Iapls3-13			-0.2136	0.3164	24	0.3651	0.0794	24	-0.1461	0.4958	24	
Three	219	D0Byu17			-0.1857	0.3849	24	0.331	0.1141	24	-0.1429	0.5052	24	
Three	220	Cnp2	Cnp-2		-0.3633	0.081	24	0.321	0.1261	24	-0.3477	0.0959	24	
Three	221	D3Byu5			-0.4615	0.0232	24 X	0.1023	0.6342	24	-0.3485	0.0951	24	
Three	222	D3Ncvs27	B75		-0.4477	0.0322	23 X	0.0937	0.6705	23	-0.3405	0.1119	23	
Three	223	D3Mit22	Rpl32-ps. D122		-0.4959	0.0262	20 Y	-0.0778	0.7446	20	-0.2614	0.2656	20	
Three	224	D3Byu4			-0.2051	0.3363	24	0.1453	0.4982	24	-0.4535	0.026	24 X	
Three	225	Iapls2-9			-0.1744	0.4262	23	0.0444	0.8406	23	-0.3299	0.1242	23	
Three	226	D3Ncvs28	B266		-0.3069	0.1544	23	0.0401	0.8559	23	-0.3605	0.0911	23	
Three	227	D3Ncvs29	D263		-0.2763	0.2132	22	-0.0585	0.7959	22	-0.3352	0.1273	22	
Three	228	D3J1			-0.3762	0.0769	23	0.0592	0.7884	23	-0.3269	0.1278	23	
Three	229	Xmmv65	Xp-24, Xmmv-65		-0.3279	0.1178	24	0.0516	0.8109	24	-0.367	0.0777	24	
Three	230	D3Byu6			-0.3279	0.1178	24	0.0516	0.8109	24	-0.367	0.0777	24	
Three	231	D3Mit9	A85		-0.342	0.14	20	-0.1343	0.5724	20	-0.284	0.2249	20	
Three	232	Iapls1-7			-0.3279	0.1178	24	0.0516	0.8109	24	-0.367	0.0777	24	
Three	233	Fgg			-0.35	0.1198	21	0.0212	0.9272	21	-0.3721	0.0967	21	
Three	234	Cap1			-0.2849	0.2106	21	0.1185	0.6091	21	-0.3369	0.1353	21	
Three	235	Call1			-0.3078	0.153	23	0.0908	0.6802	23	-0.2959	0.1704	23	
Three	236	D3Tu51			-0.182	0.4425	20	0.1736	0.4642	20	-0.4361	0.0546	20	
Three	237	Pmv38	Pmv-38		-0.2992	0.1555	24	0.0884	0.6813	24	-0.2911	0.1676	24	
Three	238	D3Mit10	A34		-0.1565	0.51	20	-0.067	0.7791	20	-0.0275	0.9085	20	
Three	239	D3Mit12	A60		-0.3972	0.0829	20	-0.1139	0.6326	20	0.3988	0.0815	20	
Three	240	D3Ncvs43	B495		-0.3947	0.0623	23	0.0047	0.9829	23	0.0507	0.8182	23	
Three	241	D3Ncvs30	B328		-0.4098	0.0521	23	0.126	0.5666	23	0.1273	0.5627	23	
Three	242	D3Ncvs31	D329		-0.4098	0.0521	23	0.126	0.5666	23	0.1273	0.5627	23	
Three	243	D3Ncvs32	B143		-0.3926	0.0639	23	0.1419	0.5184	23	0.1156	0.5994	23	

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N		PCTWRED	P2ALPHA	P2N		AVGBASE1	P3ALPHA	P3N	
Three	244	Amy1	Amy-1		-0.3774	0.069	24		0.251	0.2368	24		0.0093	0.9656	24	
Three	245	Amy2			-0.3774	0.069	24		0.251	0.2368	24		0.0093	0.9656	24	
Three	246	D3Nds5			-0.3774	0.069	24		0.251	0.2368	24		0.0093	0.9656	24	
Three	247	D3Ncvs45	B508		-0.38	0.0984	20		0.2281	0.3335	20		0.0516	0.8289	20	
Three	248	D3Ncvs44	B527		-0.4841	0.0192	23	X	0.1143	0.6035	23		0.1016	0.6446	23	
Three	249	D3J3			-0.4788	0.0242	22	X	0.241	0.2799	22		0.2183	0.329	22	
Three	250	Fabp1			-0.4638	0.0297	22	X	0.1924	0.391	22		0.1077	0.6334	22	
Three	251	D3Ncvs33	D125		-0.4058	0.0547	23		0.2296	0.2919	23		0.1714	0.4343	23	
Three	252	D3Ncvs8	B126, D126?		-0.3408	0.1206	22		0.2549	0.2524	22		0.2146	0.3376	22	
Three	253	Adh1-ps	Adh-rs		-0.3698	0.1085	20		0.259	0.2702	20		0.1123	0.6375	20	
Three	254	Oat-rs2			-0.2831	0.1801	24		0.2354	0.2682	24		0.0992	0.6445	24	
Three	255	Pmv39			-0.2831	0.1801	24		0.2354	0.2682	24		0.0992	0.6445	24	
Three	256	D3Mit19	M141		-0.4025	0.0785	20		0.0254	0.9153	20		-0.0398	0.8678	20	
Three	257	cdm			-0.195	0.3611	24		0.1854	0.3857	24		0.1136	0.597	24	
Three	258	D3Ncvs34	B206		-0.2278	0.2959	23		0.1854	0.3969	23		0.0843	0.7023	23	
Three	259	D3Ncvs35	D207		-0.2278	0.2959	23		0.1854	0.3969	23		0.0843	0.7023	23	
Three	260	D3Ncvs36	D22		-0.1571	0.474	23		0.1445	0.5107	23		0.0903	0.6819	23	
Three	261	D3Ncvs37	B21		-0.1571	0.474	23		0.1445	0.5107	23		0.0903	0.6819	23	
Three	262	D3Nds2	T21		-0.0974	0.6828	20		-0.0429	0.8576	20		0.0896	0.7072	20	
Three	263	D3Mit15	A55		-0.0974	0.6828	20		-0.0429	0.8576	20		0.0896	0.7072	20	
Three	264	D3Nds3			-0.1786	0.4037	24		0.1505	0.4826	24		0.0713	0.7406	24	
Three	265	Egf			-0.1786	0.4037	24		0.1505	0.4826	24		0.0713	0.7406	24	
Three	266	Adh1			-0.1786	0.4037	24		0.1505	0.4826	24		0.0713	0.7406	24	
Three	267	Adh3			-0.1972	0.3671	23		0.1021	0.643	23		0.1187	0.5895	23	
Three	268	Bmn			-0.2521	0.2703	21		0.1451	0.5302	21		0.0692	0.7655	21	
Three	269	D3Jkn1			-0.1305	0.5434	24		0.1408	0.5118	24		0.0354	0.8696	24	
Three	270	D3Ncvs47	B589		-0.1305	0.5434	24		0.1408	0.5118	24		0.0354	0.8696	24	
Three	271	D3Ncvs48	D686		-0.1165	0.5964	23		0.1062	0.6298	23		0.016	0.9423	23	
Three	272	D3Ncvs40	D590		-0.1305	0.5434	24		0.1408	0.5118	24		0.0354	0.8696	24	
Three	273	D3Ncvs41	D647		-0.1074	0.6256	23		0.0964	0.6616	23		0.0388	0.8603	23	
Three	274	D3Mit17	M235		0.1406	0.5543	20		-0.1394	0.5577	20		-0.2738	0.2427	20	
Three	275	D3Ncvs49	D521		-0.0745	0.7294	24		0.2863	0.175	24		-0.0612	0.7764	24	
Three	276	Pmv26			-0.2485	0.2416	24		0.1906	0.3722	24		0.0636	0.7679	24	
Three	277	Iapls2-14			-0.2515	0.247	23		0.4163	0.0482	23	X	0.0554	0.8017	23	
Three	278	P40-rs4	P40-4		-0.106	0.622	24		0.1686	0.4311	24		0.1212	0.5728	24	
Four	279	Mos			-0.157	0.4638	24		0.2379	0.2629	24		-0.3584	0.0855	24	
Four	280	Carp			0.0537	0.8031	24		0.4775	0.0183	24	X	-0.4749	0.019	24	X
Four	281	D4Bir3			0.0537	0.8031	24		0.4775	0.0183	24	X	-0.4749	0.019	24	X
Four	282	Gln3-6			-0.0723	0.7369	24		0.3755	0.0706	24		-0.3904	0.0593	24	
Four	283	Gln3-2			-0.0723	0.7369	24		0.3755	0.0706	24		-0.3904	0.0593	24	
Four	284	Mtv14	Mtv-14		0.339	0.1051	24		0.1486	0.4882	24		-0.3164	0.1319	24	
Four	285	D4Byu1			0.339	0.1051	24		0.1486	0.4882	24		-0.3164	0.1319	24	
Four	286	D4Byu2			0.339	0.1051	24		0.1486	0.4882	24		-0.3164	0.1319	24	
Four	287	D4Bir4			0.1676	0.4337	24		0.1114	0.6043	24		-0.0306	0.8873	24	
Four	288	D4Ncvs56	D317		0.511	0.0127	23	X	0.29	0.1794	23		-0.2271	0.2973	23	
Four	289	D4Ncvs57	B318		0.5017	0.0174	22	X	0.2833	0.2014	22		-0.255	0.2521	22	
Four	290	D4Ncvs78	B478		0.428	0.0416	23	X	0.0434	0.8442	23		-0.2289	0.2935	23	
Four	291	Lyb4	Lyb-4		0.4247	0.0434	23	X	-0.0635	0.7735	23		-0.1213	0.5814	23	
Four	292	Scl2			0.5208	0.0825	12		-0.195	0.5437	12		-0.0004	0.9989	12	
Four	293	Xmmv8	Env-8, Xmmv-8		0.3832	0.0646	24		-0.0077	0.9714	24		-0.2491	0.2405	24	
Four	294	D4Bir2			0.445	0.0293	24	X	-0.0188	0.9303	24		-0.294	0.1631	24	
Four	295	Pmv-30			0.445	0.0293	24	X	-0.0188	0.9303	24		-0.294	0.1631	24	
Four	296	D4Nds3			0.5288	0.0114	22	X	-0.0275	0.9032	22		-0.2559	0.2503	22	
Four	297	Ly32	Ly-32		0.4418	0.0307	24	X	0.0511	0.8125	24		-0.214	0.3153	24	
Four	298	Cd72	Lyb-2		0.4319	0.0447	22	X	0.0203	0.9286	22		-0.212	0.3437	22	
Four	299	D4Nds8			0.4418	0.0307	24	X	0.0511	0.8125	24		-0.214	0.3153	24	
Four	300	D4Nds6			0.3438	0.1	24		0.0236	0.9129	24		-0.1545	0.471	24	
Four	301	D4Ncvs58	D223		0.3326	0.121	23		0.0299	0.8923	23		-0.1673	0.4455	23	
Four	302	Gln3-1			0.3438	0.1	24		0.0236	0.9129	24		-0.1545	0.471	24	
Four	303	Iapls3-22			0.3438	0.1	24		0.0236	0.9129	24		-0.1545	0.471	24	
Four	304	Mup1	Mup-1		0.3438	0.1	24		0.0236	0.9129	24		-0.1545	0.471	24	
Four	305	Lv			0.2274	0.2966	23		-0.0699	0.7515	23		0.0153	0.9448	23	
Four	306	Orm1	Orm-1		0.4254	0.0382	24	X	0.2534	0.2322	24		-0.1547	0.4704	24	
Four	307	D4Byu3			0.3438	0.1	24		0.0236	0.9129	24		-0.1545	0.471	24	
Four	308	D4Ncvs79	B460		0.2067	0.3687	21		0.3603	0.1086	21		-0.3989	0.0733	21	
Four	309	D4Ncvs74	D459		0.2067	0.3687	21		0.3603	0.1086	21		-0.3989	0.0733	21	
Four	310	D4Ncvs81	B410		0.3198	0.1576	21		-0.0662	0.7755	21		-0.2512	0.2721	21	
Four	311	D4Mit17	Alqp. D1		0.3467	0.1342	20		0.5824	0.0071	20	X	-0.3511	0.1291	20	
Four	312	D4Nds7			0.3473	0.0964	24		0.276	0.1917	24		-0.2825	0.1811	24	
Four	313	Tyrp	b;Trp		0.3473	0.0964	24		0.276	0.1917	24		-0.2825	0.1811	24	
Four	314	b			0.3473	0.0964	24		0.276	0.1917	24		-0.2825	0.1811	24	
Four	315	Adfp			0.4317	0.0448	22	X	0.204	0.3626	22		-0.3166	0.1511	22	
Four	316	Iapls1-10			0.3646	0.0798	24		0.221	0.2993	24		-0.3643	0.0801	24	
Four	317	D4Bir5			0.3646	0.0798	24		0.221	0.2993	24		-0.3643	0.0801	24	
Four	318	D4Mc2	D4McC2		0.4068	0.0541	23		0.2167	0.3207	23		-0.3565	0.095	23	
Four	319	Ms6hm			0.1612	0.4625	23		0.2504	0.2491	23		-0.2496	0.2507	23	
Four	320	D4Ncvs82	B513		0.2323	0.3109	21		0.2094	0.3623	21		-0.3627	0.1061	21	
Four	321	Ifa			0.2257	0.289	24		0.2707	0.2007	24		-0.3544	0.0893	24	
Four	322	Iapls3-10			0.1959	0.359	24		0.0266	0.9018	24		-0.1036	0.63	24	
Four	323	Mls2	Mls-2		0.1021	0.7523	12		-0.0622	0.8477	12		-0.1859	0.5629	12	
Four	324	Pmv19	Pmv-19		0.1609	0.4527	24		-0.016	0.9409	24		-0.2312	0.277	24	

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N	PCTWRED	P2ALPHA	P2N	AVGBASE1	P3ALPHA	P3N
Four	325	D4Ncvs75	D470		0.1963	0.3812	22	-0.0258	0.9093	22	-0.1209	0.592	22
Four	326	D4Ncvs83	B480		0.0896	0.6844	23	-0.1174	0.5936	23	-0.1749	0.4248	23
Four	327	D4Ncvs84	D481		0.0714	0.7462	23	-0.0196	0.9293	23	-0.1599	0.4661	23
Four	328	D4Ncvs59	D319		0.1157	0.5992	23	-0.041	0.8528	23	-0.1454	0.5079	23
Four	329	Mtv13	Mtv-13		0.0451	0.8341	24	-0.0238	0.9122	24	-0.1213	0.5722	24
Four	330	Cycb10	Cycb-10		0.0321	0.8844	23	-0.0217	0.9217	23	-0.0964	0.6617	23
Four	331	Cyp4a10	Cyp4a		0.0451	0.8341	24	-0.0238	0.9122	24	-0.1213	0.5722	24
Four	332	Glut1	Glut-1		0.0451	0.8341	24	-0.0238	0.9122	24	-0.1213	0.5722	24
Four	333	D4Ncvs60	D341		0.063	0.7753	23	-0.0301	0.8915	23	-0.112	0.611	23
Four	334	D4Ncvs10	B340		0.063	0.7753	23	-0.0301	0.8915	23	-0.112	0.611	23
Four	335	Msl5-1			0.0225	0.9187	23	-0.0107	0.9612	23	-0.1251	0.5694	23
Four	336	D4Ncvs14	B152		0.063	0.7753	23	-0.0301	0.8915	23	-0.112	0.611	23
Four	337	D4Ncvs61	D105		0.0533	0.8092	23	-0.0485	0.8262	23	-0.0993	0.6521	23
Four	338	D4Ncvs13	B102		0.0533	0.8092	23	-0.0485	0.8262	23	-0.0993	0.6521	23
Four	339	D4Ncvs62	B65		0.1683	0.4427	23	0.0921	0.676	23	-0.0154	0.9443	23
Four	340	D4Mit12	M15		0.1319	0.5793	20	0.1015	0.6702	20	-0.0774	0.7457	20
Four	341	Mpmv19	Mpmv-19		0.0885	0.6809	24	0.0447	0.8356	24	-0.0433	0.8408	24
Four	342	Ckb-rs1	Ck-1		0.1507	0.4822	24	0.0968	0.6529	24	-0.0249	0.9079	24
Four	343	D4Ncvs85	B641		0.2904	0.1687	24	0.0753	0.7266	24	-0.0099	0.9633	24
Four	344	D4Mit16	A65		0.1317	0.58	20	0.2339	0.3209	20	0.077	0.7471	20
Four	345	Lck			0.1438	0.5028	24	-0.0601	0.7802	24	0.1458	0.4968	24
Four	346	D4Ncvs63	B205		0.1888	0.3884	23	-0.0608	0.7828	23	0.0048	0.9827	23
Four	347	Nhe1	Nhe-1		0.1213	0.6106	20	-0.06	0.8017	20	-0.1273	0.5927	20
Four	348	D4Ncvs87	B586		0.2078	0.33	24	-0.0611	0.7767	24	-0.0467	0.8284	24
Four	349	Xmmv23	Env-23, Xmmv-23		0.1848	0.3987	23	-0.1014	0.6451	23	-0.0352	0.8733	23
Four	350	D4Byu4			0.0078	0.972	23	-0.2162	0.3218	23	0.0798	0.7173	23
Four	351	Ela1-ps	Ela-1rs, Ela-1p		0.0502	0.8159	24	-0.0894	0.6778	24	0.0855	0.691	24
Four	352	D4Mc1	KAB1, D4McC1		-0.0015	0.9944	24	-0.1009	0.639	24	0.0795	0.7119	24
Four	353	Ahd1	Ahd-1		-0.0015	0.9944	24	-0.1009	0.639	24	0.0795	0.7119	24
Four	354	D4Ncvs64	B153		-0.0728	0.7539	21	-0.1365	0.5553	21	0.3198	0.1576	21
Four	355	D4Ncvs65	D154		-0.0728	0.7539	21	-0.1365	0.5553	21	0.3198	0.1576	21
Four	356	D4Ncvs66	B66		-0.0041	0.9872	18	-0.0571	0.822	18	0.0662	0.7942	18
Four	357	Akp-2			-0.0015	0.9944	24	-0.1009	0.639	24	0.0795	0.7119	24
Four	358	Ly31	Ly-31		-0.0015	0.9944	24	-0.1009	0.639	24	0.0795	0.7119	24
Four	359	D4Ncvs88	B543		-0.0562	0.8038	22	-0.1282	0.5697	22	0.1911	0.3943	22
Four	360	D4Ncvs89	D627		-0.0369	0.8673	23	-0.205	0.3481	23	-0.0015	0.9947	23
Four	361	D4Ncvs90	D529		-0.0233	0.9222	20	-0.252	0.2839	20	-0.064	0.7886	20
Four	362	D4Mit13	M169		-0.1567	0.5094	20	-0.1307	0.5827	20	-0.0034	0.9887	20
Four	363	Pdi			-0.2354	0.3043	21	-0.0563	0.8083	21	0.0085	0.9709	21
Four	364	D4Ncvs68	D171		-0.0291	0.8976	22	-0.1143	0.6125	22	0.0717	0.7511	22
Four	365	D4Ncvs67	D99		-0.0574	0.7946	23	-0.137	0.5332	23	0.0595	0.7874	23
Four	366	D4Ncvs19	B287		-0.0574	0.7946	23	-0.137	0.5332	23	0.0595	0.7874	23
Four	367	Hspg2	Plc		-0.1455	0.4976	24	-0.1667	0.4362	24	0.1006	0.6398	24
Four	368	Gpd1	Gpd-1		-0.0515	0.8112	24	-0.0881	0.6824	24	0.0839	0.6966	24
Four	369	D4Nds5			-0.0515	0.8112	24	-0.0881	0.6824	24	0.0839	0.6966	24
Four	370	D4Mit14	A69		-0.1366	0.5658	20	-0.0301	0.8997	20	0.0382	0.8731	20
Four	371	Ms6-2			-0.0774	0.7322	22	-0.1132	0.6158	22	0.2735	0.2182	22
Four	372	D4Ncvs94	B525		-0.0761	0.7239	24	-0.119	0.5798	24	-0.1169	0.5865	24
Four	373	Ly39	Ly-39		-0.1671	0.5215	17	-0.3463	0.1733	17	-0.0387	0.8828	17
Four	374	D4Ncvs93	D524		0.0315	0.8837	24	-0.179	0.4025	24	0.0067	0.9753	24
Four	375	D4Ncvs92	B669		-0.0092	0.9666	23	-0.1551	0.4797	23	0.0412	0.8519	23
Four	376	D4Ncvs77	D421		-0.0428	0.8578	20	-0.2273	0.3351	20	0.0399	0.8673	20
Four	377	D4Ncvs91	B491		-0.1867	0.4054	22	-0.1729	0.4415	22	0.0478	0.8327	22
Four	378	D4Ncvs76	D668		-0.0595	0.7874	23	-0.1489	0.4976	23	0.0774	0.7256	23
Four	379	D4Ncvs69	D234		0.0629	0.7755	23	0.1236	0.5741	23	0.0963	0.662	23
Four	380	D4Ncvs70	B48		0.0629	0.7755	23	0.1236	0.5741	23	0.0963	0.662	23
Four	381	D4Ncvs71	B357		0.0629	0.7755	23	0.1236	0.5741	23	0.0963	0.662	23
Four	382	D4Ncvs72	B241		0.0629	0.7755	23	0.1236	0.5741	23	0.0963	0.662	23
Four	383	Fv1	Fv-1		0.0552	0.8073	22	0.0754	0.7389	22	0.0563	0.8034	22
Four	384	Xmv8	Xmv-8		0.0372	0.8629	24	0.1299	0.5453	24	0.0788	0.7144	24
Four	385	Xmv9	Xmv-9		0.0372	0.8629	24	0.1299	0.5453	24	0.0788	0.7144	24
Four	386	Xmv14	Xmv-14		0.0372	0.8629	24	0.1299	0.5453	24	0.0788	0.7144	24
Four	387	Xmv44	Xmv-44		0.0372	0.8629	24	0.1299	0.5453	24	0.0788	0.7144	24
Four	388	Ssm1	Ssm-1		0.0372	0.8629	24	0.1299	0.5453	24	0.0788	0.7144	24
Four	389	Iap1s2-7;3-17			0.0372	0.8629	24	0.1299	0.5453	24	0.0788	0.7144	24
Four	390	Pmd	Anf		0.0372	0.8629	24	0.1299	0.5453	24	0.0788	0.7144	24
Four	391	D4Ncvs95	B651		0.0178	0.934	24	0.0782	0.7164	24	0.0995	0.6436	24
Four	392	D4Ncvs96	B652		0.0594	0.783	24	0.0155	0.9428	24	0.1242	0.5631	24
Four	393	D4Smh6b	D4Pas1		-0.062	0.7789	23	-0.0822	0.7092	23	0.3109	0.1488	23
Four	394	D4Ncvs73	D17		0.0566	0.8075	21	0.0648	0.7802	21	0.3484	0.1216	21
Four	395	D4Ncvs25	B18		0.0967	0.6686	22	0.1733	0.4406	22	0.3169	0.1508	22
Four	396	D4Bir1			0.0186	0.9313	24	0.1775	0.4067	24	0.3413	0.1026	24
Four	397	Dv1			0.0186	0.9313	24	0.1775	0.4067	24	0.3413	0.1026	24
Four	398	D4Ncvs97	B618		0.0671	0.7553	24	0.1653	0.4401	24	0.3015	0.1522	24
Four	399	D4Ncvs25	B018, 184		-0.0079	0.9736	20	0.1208	0.6118	20	0.3366	0.1468	20
Four	400	Tel4q			-0.0867	0.687	24	0.1626	0.4478	24	0.2993	0.1554	24
Five	401	D5Mit1	A82		-0.3923	0.0966	19	-0.0516	0.8338	19	0.2468	0.3085	19
Five	402	Pmv40	Pmv-40		-0.3231	0.1235	24	-0.2553	0.2285	24	0.3003	0.154	24
Five	403	D5Ncvs52	D660		-0.4717	0.056	17	-0.3207	0.2094	17	0.4496	0.0702	17
Five	404	D5Ncvs45	B218		-0.3895	0.0732	22	-0.2925	0.1866	22	0.2967	0.18	22
Five	405	D5Ncvs55	B636		-0.2547	0.2409	23	-0.2266	0.2984	23	0.289	0.181	23

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CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N		PCTWRED	P2ALPHA	P2N		AVGBASE1	P3ALPHA	P3N	
Five	406	D5Ncvs56	B683		-0.4302	0.0457	22	X	-0.1654	0.4619	22		0.2641	0.235	22	
Five	407	Xmv17	Xmv-17		-0.4602	0.0236	24	X	-0.1696	0.4283	24		0.3054	0.1468	24	
Five	408	Xmv45			-0.4602	0.0236	24	X	-0.1696	0.4283	24		0.3054	0.1468	24	
Five	409	Mpmv23	Xmmv-53/Xp-11/M		-0.4602	0.0236	24	X	-0.1696	0.4283	24		0.3054	0.1468	24	
Five	410	D5Ncvs3	B187, 269		-0.4239	0.0555	21		-0.123	0.5954	21		0.3313	0.1423	21	
Five	411	Xmv34	Xmv-34		-0.4495	0.0275	24	X	-0.1517	0.4791	24		0.2975	0.158	24	
Five	412	Mpmv13			-0.4315	0.0353	24	X	0.211	0.3223	24		0.0647	0.7639	24	
Five	413	D5H4S43	D4S43h		-0.439	0.0319	24	X	-0.0426	0.8432	24		-0.0464	0.8296	24	
Five	414	Mpmv7	Xmmv-5/Env-5/Mp		-0.3097	0.1409	24		0.1688	0.4305	24		-0.1355	0.5279	24	
Five	415	Pmv5	Pmv-5		-0.3097	0.1409	24		0.1688	0.4305	24		-0.1355	0.5279	24	
Five	416	D5Nds1			-0.2776	0.2109	22		0.0506	0.823	22		-0.0178	0.9373	22	
Five	417	D5Ncvs57	B554		-0.41	0.052	23		0.2287	0.2939	23		-0.1818	0.4063	23	
Five	418	D5Mit11	M97		-0.0652	0.7908	19		-0.115	0.6393	19		-0.1943	0.4254	19	
Five	419	D5H4S76	D4S76h		-0.1454	0.4977	24		-0.0601	0.7803	24		-0.0664	0.758	24	
Five	420	D5Byu1			-0.3292	0.1162	24		-0.0891	0.679	24		-0.1221	0.5698	24	
Five	421	D5Byu2			-0.1454	0.4977	24		-0.0601	0.7803	24		-0.0664	0.758	24	
Five	422	D5Byu3			-0.2233	0.2941	24		-0.1863	0.3833	24		0.0548	0.7993	24	
Five	423	Pqm1	Pqm-1		-0.2841	0.1784	24		-0.0097	0.9643	24		-0.0579	0.788	24	
Five	424	D5Ncvs46	BI79		-0.1627	0.4582	23		-0.036	0.8706	23		-0.182	0.4058	23	
Five	425	D5Ncvs47	D181		-0.1627	0.4582	23		-0.036	0.8706	23		-0.182	0.4058	23	
Five	426	D5Ncvs58	B616		-0.4516	0.0349	22	X	-0.0448	0.8431	22		-0.158	0.4826	22	
Five	427	D5Mn125	D5SC25		-0.2257	0.2889	24		-0.0761	0.7239	24		-0.044	0.8383	24	
Five	428	Ms15-6			-0.3022	0.1611	23		-0.0514	0.8157	23		0.0385	0.8616	23	
Five	429	Pmv11	Pmv-11		-0.2056	0.3352	24		0.1893	0.3758	24		-0.0578	0.7886	24	
Five	430	D5Nds3			-0.2056	0.3352	24		0.1893	0.3758	24		-0.0578	0.7886	24	
Five	431	Nmyc2	Nmyc-2		-0.1724	0.4315	23		0.1733	0.429	23		0.0131	0.9527	23	
Five	432	Els1	Els-1		-0.2072	0.3312	24		0.2363	0.2663	24		0.1422	0.5073	24	
Five	433	Iapls1-4			-0.1675	0.4339	24		0.2303	0.279	24		0.0591	0.784	24	
Five	434	D5Bir1			-0.1675	0.4339	24		0.2303	0.279	24		0.0591	0.784	24	
Five	435	Afp	D5Nds4		-0.2842	0.1888	23		0.0159	0.9426	23		0.1127	0.6087	23	
Five	436	D5Mit7	M154		-0.1787	0.4511	20		-0.0936	0.6946	20		0.2189	0.3538	20	
Five	437	D5Mit10	M207		-0.2803	0.2451	19		-0.1005	0.6822	19		0.1076	0.6611	19	
Five	438	Ric			-0.3587	0.0852	24		0.1262	0.5567	24		-0.0319	0.8825	24	
Five	439	Spp1	Spp-1		-0.3587	0.0852	24		0.1262	0.5567	24		-0.0319	0.8825	24	
Five	440	D5Ncvs48	B220		-0.4508	0.0403	21	X	0.1809	0.4327	21		-0.0599	0.7964	21	
Five	441	Tsz1	Tsz-1		-0.333	0.1636	19		0.5014	0.0287	19	X	0.1125	0.6464	19	
Five	442	D5Ncvs13	B161		-0.3876	0.0677	23		0.2233	0.3057	23		0.1021	0.6431	23	
Five	443	Ms15-5			-0.1069	0.6274	23		-0.0493	0.8233	23		0.2889	0.1812	23	
Five	444	D5Ncvs49	D316		-0.2638	0.248	21		-0.0839	0.7176	21		0.2101	0.3608	21	
Five	445	D5Ncvs50	B164		-0.3612	0.1077	21		0.0959	0.6792	21		0.0535	0.8177	21	
Five	446	D5Ncvs51	D163		-0.3612	0.1077	21		0.0959	0.6792	21		0.0535	0.8177	21	
Five	447	Bcd1	Bcd-1		-0.3177	0.1497	22		0.0739	0.7437	22		0.0526	0.8161	22	
Five	448	D5Ncvs59	B569		-0.0351	0.9011	15		0.0802	0.7764	15		0.1687	0.5479	15	
Five	449	Tcf1	Hnf-1/Tcf-1		-0.3166	0.1317	24		0.1285	0.5495	24		0.0753	0.7267	24	
Five	450	D5Ehs1	C		-0.3177	0.1497	22		0.0739	0.7437	22		0.0526	0.8161	22	
Five	451	D5Ncvs60	B435		-0.2591	0.2326	23		0.2022	0.3547	23		-0.062	0.7786	23	
Five	452	Fla	Flp		-0.2578	0.2592	21		0.2003	0.384	21		-0.0917	0.6926	21	
Five	453	D5Byu4			-0.154	0.4724	24		0.2916	0.1669	24		-0.2628	0.2147	24	
Five	454	Zp3	Zp-3		-0.1803	0.3991	24		0.1385	0.5186	24		-0.1418	0.5086	24	
Five	455	Ache			-0.1879	0.3792	24		0.168	0.4327	24		-0.2471	0.2443	24	
Five	456	Mr66-1			-0.1879	0.3792	24		0.168	0.4327	24		-0.2471	0.2443	24	
Five	457	D5Ncvs53	D675		-0.0513	0.8163	23		-0.1069	0.6275	23		0.0081	0.9706	23	
Five	458	D5Ncvs61	B674		-0.0513	0.8163	23		-0.1069	0.6275	23		0.0081	0.9706	23	
Five	459	D5Ncvs54	D596		-0.2579	0.2236	24		-0.0102	0.9623	24		0.04	0.8527	24	
Five	460	Pmv12	Pmv-12		-0.3099	0.1405	24		0.0001	0.9995	24		0.077	0.7206	24	
Five	461	D5Byu5			-0.3099	0.1405	24		0.0001	0.9995	24		0.077	0.7206	24	
Six	462	D6Mit86			0.011	0.9595	24		0.2427	0.2531	24		-0.0894	0.6779	24	
Six	463	Met			-0.03	0.8895	24		0.3912	0.0587	24		-0.1405	0.5127	24	
Six	464	D6Nds3			-0.03	0.8895	24		0.3912	0.0587	24		-0.1405	0.5127	24	
Six	465	D6Ncvs31	D353		0.0885	0.7108	20		0.3217	0.1667	20		-0.0673	0.7781	20	
Six	466	D6Ncvs32	B354		-0.0099	0.9642	23		0.3894	0.0662	23		-0.1296	0.5556	23	
Six	467	Iapls2-2			-0.0935	0.664	24		0.1749	0.4136	24		-0.182	0.3947	24	
Six	468	D6Ncvs33	B151		-0.0713	0.7465	23		0.1696	0.4391	23		-0.1703	0.4372	23	
Six	469	Hbnf			0.1508	0.4922	23		0.3328	0.1208	23		-0.3487	0.1029	23	
Six	470	D6Ncvs47	D661		-0.0327	0.8824	23		0.2802	0.1953	23		-0.2627	0.2258	23	
Six	471	Hoxa	Hox-1		0.1011	0.6382	24		0.2667	0.2078	24		-0.3107	0.1395	24	
Six	472	D6Ncvs34	D190		0.3311	0.1228	23		0.2969	0.1689	23		-0.2465	0.2568	23	
Six	473	D6Ncvs35	B274		0.1182	0.6004	22		0.3286	0.1354	22		-0.2854	0.1979	22	
Six	474	D6Ncvs36	B30		0.1341	0.542	23		0.2636	0.2243	23		-0.3013	0.1624	23	
Six	475	D6Ncvs37	D169		0.2832	0.2263	20		0.1554	0.513	20		-0.3052	0.1908	20	
Six	476	D6Ncvs38	B170		0.2285	0.3063	22		0.1921	0.3918	22		-0.254	0.254	22	
Six	477	D6Ncvs39	B189		0.1341	0.542	23		0.2636	0.2243	23		-0.3013	0.1624	23	
Six	478	Iapls3-16			0.1011	0.6382	24		0.2667	0.2078	24		-0.3107	0.1395	24	
Six	479	Gpc			0.1011	0.6382	24		0.2667	0.2078	24		-0.3107	0.1395	24	
Six	480	D6Ncvs3	B55		0.1341	0.542	23		0.2636	0.2243	23		-0.3013	0.1624	23	
Six	481	D6Ncvs40	D56		0.1341	0.542	23		0.2636	0.2243	23		-0.3013	0.1624	23	
Six	482	D6Ncvs49	B405		-0.0815	0.7255	21		0.2497	0.2749	21		-0.4376	0.0472	21	X
Six	483	D5Ncvs50	B614		0.4392	0.0318	24	X	0.0821	0.7031	24		-0.114	0.5957	24	
Six	484	D6Ncvs41	B77		0.3462	0.1056	23		0.0683	0.7569	23		-0.1703	0.4372	23	
Six	485	Cd8a	D6Mit16, Ly-2		0.3621	0.0821	24		0.0573	0.7902	24		-0.1493	0.4864	24	
Six	486	D6Mit16	Ly-2. 511		0.2474	0.3224	18		0.2546	0.308	18		-0.2325	0.3532	18	

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CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N		PCTWRED	P2ALPHA	P2N		AVGBASE1	P3ALPHA	P3N	
Six	487	Xmmv27	Env-27, Xmmv-27		0.3621	0.0821	24		0.0573	0.7902	24		-0.1493	0.4864	24	
Six	488	Lvp1	Lvp-1		0.3621	0.0821	24		0.0573	0.7902	24		-0.1493	0.4864	24	
Six	489	Brp1	Brp-1		0.3506	0.1192	21		0.0521	0.8225	21		-0.0786	0.735	21	
Six	490	Ms6-4			0.3781	0.0827	22		0.04	0.8597	22		-0.0255	0.9102	22	
Six	491	D6Mit9	L23		0.1944	0.4114	20		0.2983	0.2014	20		-0.3034	0.1935	20	
Six	492	Tgfa	wa-1		0.2815	0.1827	24		0.0962	0.6547	24		-0.1276	0.5525	24	
Six	493	D6Nds2			0.1623	0.4486	24		0.0911	0.6719	24		-0.3333	0.1115	24	
Six	494	D6Ncvs51	B486		0.1616	0.4612	23		0.0877	0.6908	23		-0.1947	0.3734	23	
Six	495	Gln3-3			0.2258	0.2886	24		0.0934	0.6641	24		-0.013	0.9519	24	
Six	496	Il5ra			0.1968	0.3568	24		0.1484	0.4888	24		0.057	0.7912	24	
Six	497	D6Nds5	D4Nds1		0.1968	0.3568	24		0.1484	0.4888	24		0.057	0.7912	24	
Six	498	D6Bir1			0.008	0.9704	24		0.1167	0.587	24		-0.0001	0.9998	24	
Six	499	D6Bir2			0.0312	0.8875	23		0.1242	0.5724	23		0.0022	0.9919	23	
Six	500	Cyx			-0.0004	0.9987	20		-0.2804	0.2311	20		0.1167	0.6243	20	
Six	501	Qu1			0.1758	0.4585	20		-0.234	0.3208	20		-0.131	0.582	20	
Six	502	D6Ncvs42	D237		-0.0777	0.731	22		-0.153	0.4966	22		-0.1571	0.485	22	
Six	503	D6Ncvs43	B210		0.0036	0.987	23		-0.0782	0.7228	23		-0.2033	0.3523	23	
Six	504	Rua			0.0746	0.7547	20		-0.2285	0.3325	20		-0.1931	0.4147	20	
Six	505	Glb			0.1636	0.4906	20		-0.2065	0.3825	20		-0.0133	0.9556	20	
Six	506	Rho			0.0762	0.7233	24		-0.097	0.652	24		-0.2259	0.2885	24	
Six	507	Tpi1	Tpi-1		-0.0276	0.9082	20		0.1062	0.656	20		-0.3957	0.0842	20	
Six	508	Tpi-rs11			0.1269	0.5835	21		-0.0694	0.7651	21		-0.1895	0.4107	21	
Six	509	Kap			0.0834	0.7194	21		-0.1519	0.511	21		-0.2755	0.2268	21	
Six	510	D6Mit13	Prp. D34		-0.0572	0.8107	20		0.041	0.8639	20		-0.4291	0.059	20	
Six	511	Tnfrl			-0.1128	0.5996	24		-0.0333	0.8772	24		-0.1747	0.4143	24	
Six	512	Ly55	mNKR-P1, Ly-55		0.0256	0.9054	24		-0.0848	0.6934	24		-0.1853	0.3861	24	
Six	513	Prp	D6Mit16		0.0256	0.9054	24		-0.0848	0.6934	24		-0.1853	0.3861	24	
Six	514	Ly49	Ly-49		0.0256	0.9054	24		-0.0848	0.6934	24		-0.1853	0.3861	24	
Six	515	D6Ncvs44	B138		-0.0119	0.9571	23		-0.1805	0.41	23		-0.2195	0.3142	23	
Six	516	D6Ncvs45	B148		0.0558	0.8002	23		-0.1375	0.5315	23		-0.2791	0.1972	23	
Six	517	D6Byu1			0.0803	0.7091	24		-0.1437	0.5028	24		-0.2553	0.2285	24	
Six	518	Nmdar2b			0.0803	0.7091	24		-0.1437	0.5028	24		-0.2553	0.2285	24	
Six	519	D6Mit10	M78		0.0557	0.8155	20		0.341	0.1412	20		-0.4251	0.0617	20	
Six	520	Eal0	Ea-10		0.3017	0.1961	20		0.4324	0.0569	20		-0.2447	0.2985	20	
Six	521	D6Rp1			0.1177	0.5839	24		-0.0939	0.6627	24		0.0555	0.7966	24	
Six	522	Xmv24	Xmv-24		0.3691	0.0759	24		-0.0238	0.9121	24		0.0541	0.8017	24	
Six	523	D6Mit15	M148		0.3805	0.0979	20		0.2332	0.3223	20		-0.1012	0.6713	20	
Six	524	D6Mit14	M190		0.3805	0.0979	20		0.2332	0.3223	20		-0.1012	0.6713	20	
Six	525	D6Byu2			0.2609	0.2181	24		0.2108	0.3229	24		-0.1324	0.5375	24	
Six	526	D6Ncvs48	D605		0.4164	0.043	24	X	-0.034	0.8747	24		0.0179	0.9338	24	
Six	527	P40-rs1	P40-1		0.4315	0.0353	24	X	-0.1108	0.6063	24		0.028	0.8965	24	
Six	528	Iapls3-18			0.4315	0.0353	24	X	-0.1108	0.6063	24		0.028	0.8965	24	
Six	529	Xmmv54	Xp-13, Xmmv-54		0.4315	0.0353	24	X	-0.1108	0.6063	24		0.028	0.8965	24	
Six	530	Xmmv7	Xp-13, Xmmv-7		0.4315	0.0353	24	X	-0.1108	0.6063	24		0.028	0.8965	24	
Seven	531	D7Mis1	Msv		-0.1043	0.6277	24		-0.1622	0.449	24		0.158	0.461	24	
Seven	532	Mr66-2			0.0675	0.7596	23		-0.5583	0.0056	23	X	0.4149	0.049	23	X
Seven	533	Iapls3-4;1-11			0.0456	0.8325	24		-0.5637	0.0041	24	X	0.4248	0.0385	24	X
Seven	534	D7Ncvs52	D440		-0.1981	0.4023	20		-0.2695	0.2505	20		-0.0751	0.753	20	
Seven	535	D7Ncvs53	D625		-0.1759	0.4222	23		-0.2764	0.2018	23		0.2188	0.3159	23	
Seven	536	D7Mc2	KB6, D7McC2		-0.1802	0.4107	23		-0.2594	0.232	23		0.2179	0.3179	23	
Seven	537	D7Ncvs3	B312		-0.1594	0.4675	23		-0.2978	0.1676	23		0.2622	0.2267	23	
Seven	538	D7Ncvs37	D310		-0.1594	0.4675	23		-0.2978	0.1676	23		0.2622	0.2267	23	
Seven	539	D7Ncvs38	D204		-0.1655	0.4983	19		-0.3028	0.2077	19		0.2189	0.3678	19	
Seven	540	D7Ncvs48	D593		-0.2528	0.2334	24		-0.0579	0.7883	24		0.3068	0.1448	24	
Seven	541	Pmv18	Xmmv-35, Env-35		-0.3499	0.1017	23		-0.165	0.4517	23		0.3224	0.1335	23	
Seven	542	Cyp2a5	Coh, P450IIA		-0.2789	0.2088	22		-0.1669	0.4578	22		0.2861	0.1968	22	
Seven	543	Cyb2b9,10	P450IIB		-0.3029	0.16	23		-0.1465	0.5048	23		0.2964	0.1696	23	
Seven	544	Cyb2e1			-0.2939	0.1633	24		-0.2236	0.2935	24		0.2618	0.2166	24	
Seven	545	Pmv-15			-0.3209	0.1262	24		0.0458	0.8317	24		0.1455	0.4974	24	
Seven	546	Gpi-1			-0.3209	0.1262	24		0.0458	0.8317	24		0.1455	0.4974	24	
Seven	547	Lyb-8			-0.316	0.1325	24		0.1544	0.4712	24		0.1664	0.4371	24	
Seven	548	Mag			-0.3103	0.1496	23		0.0436	0.8434	23		0.1159	0.5985	23	
Seven	549	Atp4a			-0.3209	0.1262	24		0.0458	0.8317	24		0.1455	0.4974	24	
Seven	550	Abpa			-0.3209	0.1262	24		0.0458	0.8317	24		0.1455	0.4974	24	
Seven	551	D7Ncvs6	B54		-0.2054	0.3718	21		0.0834	0.7193	21		0.2051	0.3726	21	
Seven	552	Cebp			-0.3209	0.1262	24		0.0458	0.8317	24		0.1455	0.4974	24	
Seven	553	D7Rp2e			-0.3017	0.1618	23		0.0986	0.6544	23		0.1087	0.6215	23	
Seven	554	Odc-rs6	BRS-6		-0.2814	0.1828	24		-0.1549	0.4698	24		0.1383	0.5193	24	
Seven	555	D7Ncvs39	D134		-0.5311	0.0343	16	X	-0.2361	0.3786	16		-0.0767	0.7778	16	
Seven	556	Xmv30	Xmv-30		-0.3256	0.1205	24		-0.1417	0.5089	24		0.1723	0.4207	24	
Seven	557	Ngfg	D7Nds5		-0.3256	0.1205	24		-0.1417	0.5089	24		0.1723	0.4207	24	
Seven	558	D7Ncvs55	B433		-0.401	0.0991	18		-0.0008	0.9974	18		0.0349	0.8908	18	
Seven	559	Tam1	Tam-1		-0.3256	0.1205	24		-0.1417	0.5089	24		0.1723	0.4207	24	
Seven	560	P198-7			-0.2535	0.232	24		-0.1191	0.5795	24		0.2123	0.3193	24	
Seven	561	Cycb9			-0.1937	0.3645	24		0.0115	0.9576	24		0.1855	0.3854	24	
Seven	562	D7Ncvs56	B425		-0.3103	0.2101	18		-0.0818	0.7468	18		0.2876	0.2471	18	
Seven	563	Mtv1			-0.3442	0.0996	24		-0.0531	0.8054	24		0.2446	0.2493	24	
Seven	564	Gabrb-3			-0.3442	0.0996	24		-0.0531	0.8054	24		0.2446	0.2493	24	
Seven	565	Xmv-33			-0.3442	0.0996	24		-0.0531	0.8054	24		0.2446	0.2493	24	
Seven	566	D7Hms1			-0.3442	0.0996	24		-0.0531	0.8054	24		0.2446	0.2493	24	
Seven	567	D7H15S12	DN10;D7Nic1		-0.3442	0.0996	24		-0.0531	0.8054	24		0.2446	0.2493	24	

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N	PCTWRED	P2ALPHA	P2N	AVGBASE1	P3ALPHA	P3N	
Seven	568	p	28RN		-0.3442	0.0996	24	-0.0531	0.8054	24	0.2446	0.2493	24	
Seven	569	Hpmv-1			-0.3442	0.0996	24	-0.0531	0.8054	24	0.2446	0.2493	24	
Seven	570	D7Nds2	T28		-0.2221	0.297	24	-0.0891	0.679	24	0.0155	0.9427	24	
Seven	571	D7Nds3			-0.2221	0.297	24	-0.0891	0.679	24	0.0155	0.9427	24	
Seven	572	D7Ncvs57	B534		-0.0777	0.731	22	-0.1607	0.4748	22	-0.0256	0.91	22	
Seven	573	D7Ncvs40	D160		-0.1637	0.4556	23	-0.036	0.8705	23	-0.0579	0.7931	23	
Seven	574	D7Ncvs58	D445		-0.1369	0.5435	22	-0.0246	0.9136	22	0.075	0.7403	22	
Seven	575	D7Ncvs59	B514		-0.1893	0.3988	22	0.018	0.9365	22	-0.1089	0.6294	22	
Seven	576	D7Ncvs49	D667		-0.2233	0.3178	22	0.0649	0.7742	22	-0.1225	0.5869	22	
Seven	577	D7Mit7	L12		0.156	0.5112	20	-0.03	0.9002	20	-0.1947	0.4107	20	
Seven	578	Ras14			0.0454	0.8333	24	0.1499	0.4846	24	-0.1483	0.4893	24	
Seven	579	Lfol			-0.1785	0.4267	22	0.1396	0.5356	22	-0.2017	0.368	22	
Seven	580	D7Ncvs41	B61		-0.1124	0.6097	23	0.1678	0.4442	23	-0.2766	0.2014	23	
Seven	581	D7Ncvs42	D62		-0.1124	0.6097	23	0.1678	0.4442	23	-0.2766	0.2014	23	
Seven	582	D7Ncvs43	D28		-0.1124	0.6097	23	0.1678	0.4442	23	-0.2766	0.2014	23	
Seven	583	D7Ncvs44	D53		-0.1124	0.6097	23	0.1678	0.4442	23	-0.2766	0.2014	23	
Seven	584	Mod2r	Mod-2		-0.0877	0.6838	24	0.157	0.4637	24	-0.2572	0.225	24	
Seven	585	Hma			-0.3181	0.2678	14	0.0349	0.9058	14	-0.2921	0.311	14	
Seven	586	D7Ncvs45	B326		-0.4183	0.0948	17	0.1372	0.5995	17	-0.0681	0.795	17	
Seven	587	Odc-rs7			-0.1255	0.5591	24	0.278	0.1884	24	-0.2736	0.1958	24	
Seven	588	D7Ncvs46	B229		-0.3006	0.2255	18	0.1959	0.4358	18	-0.402	0.0981	18	
Seven	589	D7Ncvs50	D424		-0.0777	0.752	19	0.185	0.4482	19	-0.2315	0.3403	19	
Seven	590	D7Ncvs63	B423		-0.1086	0.658	19	0.134	0.5843	19	-0.2594	0.2835	19	
Seven	591	Rt-6			-0.137	0.5232	24	0.1734	0.4176	24	-0.2867	0.1743	24	
Seven	592	Lapls1-9			-0.137	0.5232	24	0.1734	0.4176	24	-0.2867	0.1743	24	
Seven	593	D7Mc1	KB4, D7McC1		-0.137	0.5232	24	0.1734	0.4176	24	-0.2867	0.1743	24	
Seven	594	Cycb-3			-0.1132	0.607	23	0.1722	0.4322	23	-0.3167	0.141	23	
Seven	595	Hbb			-0.137	0.5232	24	0.1734	0.4176	24	-0.2867	0.1743	24	
Seven	596	D7Byu1			-0.137	0.5232	24	0.1734	0.4176	24	-0.2867	0.1743	24	
Seven	597	D7Mit17	M91		-0.0921	0.744	15	0.226	0.418	15	-0.2755	0.3204	15	
Seven	598	Fis1	Fis-1		-0.0138	0.9491	24	0.0953	0.6578	24	0.1817	0.3954	24	
Seven	599	Fgf3	D7Nds4, Int-2		-0.0138	0.9491	24	0.0953	0.6578	24	0.1817	0.3954	24	
Seven	600	D7Ncvs47	B16		-0.0616	0.7855	22	0.4066	0.0604	22	0.1539	0.4941	22	
Seven	601	D7Ncvs64	D654		-0.0028	0.9901	23	0.0582	0.7921	23	0.1378	0.5306	23	
Seven	602	D7Ncvs65	B465		0.0472	0.8435	20	0.3567	0.1226	20	-0.0103	0.9657	20	
Seven	603	D7Ncvs51	D466		-0.0316	0.8891	22	0.1161	0.6068	22	0.2118	0.3441	22	
Seven	604	Xmmv76	Xp-9, Xmmv-76		0.0046	0.9831	24	0.0307	0.8868	24	0.0997	0.6431	24	
Seven	605	D7Mit12	M23		-0.1977	0.4034	20	0.3069	0.1882	20	-0.0804	0.7361	20	
Eight	606	D8Ncvs31	B321		-0.031	0.8884	23	0.2564	0.2377	23	-0.5119	0.0125	23	X
Eight	607	Defcr			0.0041	0.9847	24	0.2345	0.27	24	-0.4698	0.0205	24	X
Eight	608	Defcr-rs1			-0.0858	0.6903	24	0.0098	0.9639	24	-0.4559	0.0252	24	X
Eight	609	D8Mc1	KB2, D8McC1		-0.0858	0.6903	24	0.0098	0.9639	24	-0.4559	0.0252	24	X
Eight	610	D8Byu1			-0.0858	0.6903	24	0.0098	0.9639	24	-0.4559	0.0252	24	X
Eight	611	Ras16			-0.0858	0.6903	24	0.0098	0.9639	24	-0.4559	0.0252	24	X
Eight	612	D8Byu2			-0.1845	0.3881	24	0.1898	0.3744	24	-0.2993	0.1554	24	
Eight	613	D8Blr2			-0.088	0.6825	24	0.2723	0.1981	24	-0.3441	0.0997	24	
Eight	614	D8Ncvs32	D202		-0.1066	0.6284	23	0.1953	0.3719	23	-0.281	0.1941	23	
Eight	615	D8Ncvs56	B619		-0.0844	0.716	21	0.18	0.435	21	-0.2418	0.2909	21	
Eight	616	D8Mit4	M71		0.0219	0.9271	20	0.2131	0.3671	20	-0.1961	0.4074	20	
Eight	617	Ras15-2			-0.082	0.7033	24	0.1841	0.3892	24	-0.2615	0.2171	24	
Eight	618	D8Ncvs57	D630		-0.0563	0.7937	24	0.0847	0.6938	24	-0.2964	0.1596	24	
Eight	619	D8Ncvs58	B468		-0.0929	0.6658	24	0.1696	0.4281	24	-0.2593	0.2212	24	
Eight	620	D8Mit8	M257		-0.0372	0.8631	24	-0.2113	0.3216	24	-0.124	0.5637	24	
Eight	621	Cph1	Cph-1		-0.0938	0.663	24	-0.3584	0.0854	24	-0.0264	0.9025	24	
Eight	622	D8Ncvs33	D203		0.0376	0.8682	22	-0.1671	0.4574	22	0.0354	0.8759	22	
Eight	623	D8Ncvs34	D73		0.0118	0.9573	23	-0.185	0.398	23	0.0262	0.9056	23	
Eight	624	D8Ncvs35	B217		0.0118	0.9573	23	-0.185	0.398	23	0.0262	0.9056	23	
Eight	625	D8Ncvs36	B338		0.0118	0.9573	23	-0.185	0.398	23	0.0262	0.9056	23	
Eight	626	D8Ncvs5	B257		0.019	0.9314	23	-0.1666	0.4474	23	0.0178	0.9359	23	
Eight	627	D8Ncvs37	D259		0.019	0.9314	23	-0.1666	0.4474	23	0.0178	0.9359	23	
Eight	628	D8Ncvs38	D111		0.019	0.9314	23	-0.1666	0.4474	23	0.0178	0.9359	23	
Eight	629	Aprt-ps			0.0095	0.9657	23	-0.199	0.3626	23	0.0657	0.7657	23	
Eight	630	Lpl			0.0446	0.8361	24	-0.1722	0.4212	24	0.0341	0.8744	24	
Eight	631	DNcvs49	D567		-0.0382	0.8696	21	-0.3305	0.1434	21	0.1175	0.6119	21	
Eight	632	D7Ncvs59	B566		0.0365	0.872	22	-0.2112	0.3454	22	0.0482	0.8313	22	
Eight	633	D8Ncvs60	D511		0.0514	0.8249	21	-0.1647	0.4756	21	-0.0461	0.8427	21	
Eight	634	D8Ncvs61	B510		-0.063	0.7806	22	-0.181	0.4203	22	0.0044	0.9845	22	
Eight	635	D8Ncvs62	B411		-0.0515	0.8156	23	-0.154	0.483	23	0.0483	0.8268	23	
Eight	636	D8Ncvs50	D412		0.0897	0.699	21	-0.067	0.7729	21	0.3207	0.1564	21	
Eight	637	D8Mit9	A62		0.129	0.5987	19	0.316	0.1876	19	-0.0947	0.6997	19	
Eight	638	D8Ncvs63	B505		-0.0166	0.9432	21	-0.2764	0.2252	21	0.1465	0.5262	21	
Eight	639	D8Ncvs51	D611		0.0298	0.8899	24	-0.3352	0.1093	24	0.1172	0.5856	24	
Eight	640	D8Ncvs39	D127		0.1324	0.547	23	-0.2242	0.3038	23	0.012	0.9567	23	
Eight	641	D8Ncvs40	B342		0.1324	0.547	23	-0.2242	0.3038	23	0.012	0.9567	23	
Eight	642	D8Ncvs6	B290		0.1324	0.547	23	-0.2242	0.3038	23	0.012	0.9567	23	
Eight	643	D8Bir1			0.1546	0.4707	24	-0.2284	0.2831	24	0.0284	0.895	24	
Eight	644	Ucp			0.1546	0.4707	24	-0.2284	0.2831	24	0.0284	0.895	24	
Eight	645	D8Ncvs64	B544		0.0793	0.7126	24	-0.2537	0.2316	24	-0.0072	0.9734	24	
Eight	646	D8Byu3			0.2054	0.3357	24	-0.1859	0.3844	24	0.0464	0.8296	24	
Eight	647	D8Ncvs41	D11		0.1324	0.547	23	-0.2242	0.3038	23	0.012	0.9567	23	
Eight	648	D8Ncvs42	B12		0.1324	0.547	23	-0.2242	0.3038	23	0.012	0.9567	23	

SAG QTL w/ 1994 marker set

analysis run 12-16-94 by SRM

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N		PCTWRED	P2ALPHA	P2N		AVGBASE1	P3ALPHA	P3N	
Eight	649	D8Ncvs65	D623		0.1312	0.5606	22		-0.2057	0.3584	22		0.0195	0.9312	22	
Eight	650	D8Nds1			0.1836	0.4016	23		0.0261	0.9058	23		0.0229	0.9173	23	
Eight	651	Es1	Es-N, Es-1		0.2321	0.2751	24		0.1392	0.5164	24		0.1303	0.5439	24	
Eight	652	D8Ncvs66	B568		0.089	0.7171	19		0.3727	0.1161	19		-0.0097	0.9686	19	
Eight	653	D8Ncvs67	D489		0.0996	0.6676	21		0.1512	0.5129	21		-0.0526	0.821	21	
Eight	654	D8Ncvs52	D448		0.2745	0.205	23		0.1178	0.5926	23		-0.0174	0.9374	23	
Eight	655	D8Ncvs53	D428		0.2745	0.205	23		0.1178	0.5926	23		-0.0174	0.9374	23	
Eight	656	D8Ncvs68	B419		0.3083	0.1627	22		0.159	0.4798	22		0.0046	0.9838	22	
Eight	657	D8Ncvs43	D112		0.2135	0.34	22		0.191	0.3945	22		0.007	0.9754	22	
Eight	658	D8Ncvs44	B230		0.2794	0.22	21		0.2456	0.2833	21		-0.0289	0.901	21	
Eight	659	D8Ncvs8	B114		0.2054	0.3471	23		0.1776	0.4176	23		0.0111	0.9601	23	
Eight	660	D8Ncvs45	D351		0.2054	0.3471	23		0.1776	0.4176	23		0.0111	0.9601	23	
Eight	661	D8Byu4			0.2241	0.2926	24		0.1654	0.4398	24		0.0262	0.9032	24	
Eight	662	D8Byu5			0.2241	0.2926	24		0.1654	0.4398	24		0.0262	0.9032	24	
Eight	663	Gnb-rs1			0.4446	0.0382	22	X	0.1939	0.3874	22		0.0741	0.7431	22	
Eight	664	D8Mit11	A105		0.048	0.8406	20		0.3122	0.1803	20		-0.1124	0.637	20	
Eight	665	Acadm			0.0979	0.6489	24		0.159	0.4581	24		-0.0417	0.8465	24	
Eight	666	D8Ncvs54	D469		0.3487	0.1213	21		0.1503	0.5156	21		-0.2757	0.2263	21	
Eight	667	Zfp4	Zfp-4, Fnp-2		0.2985	0.1566	24		0.1953	0.3604	24		-0.0723	0.7372	24	
Eight	668	D8Ncvs46	B174		0.2771	0.2006	23		0.2156	0.3231	23		-0.0978	0.657	23	
Eight	669	D8Byu6			0.3137	0.1355	24		0.1847	0.3876	24		0.0264	0.9026	24	
Eight	670	D8Ncvs70	B546		0.6997	0.0053	14	X	0.0344	0.9072	14		-0.1143	0.6971	14	
Eight	671	D8Ncvs69	B475		0.6595	0.0008	22	X	0.1389	0.5375	22		-0.1067	0.6366	22	
Eight	672	D8Ncvs55	D444		0.4123	0.0633	21		0.1539	0.5054	21		0.1948	0.3974	21	
Eight	673	Mpmv21	Mpmv-21		0.094	0.6623	24		-0.0832	0.6991	24		0.1076	0.6167	24	
Eight	674	Xmmv29	Xmmv-29, Env-29		0.1572	0.4847	22		-0.12	0.5949	22		-0.0041	0.9854	22	
Eight	675	Emv2	Bv-1, Emv-2		-0.0381	0.8599	24		-0.0152	0.9438	24		0.1145	0.5943	24	
Eight	676	D8Ncvs47	B45		-0.0791	0.7197	23		-0.002	0.9928	23		0.0953	0.6653	23	
Eight	677	D8Ncvs48	D46		-0.0791	0.7197	23		-0.002	0.9928	23		0.0953	0.6653	23	
Nine	678	D9Ncvs34	B323		-0.097	0.6598	23		0.1299	0.5547	23		0.0406	0.8542	23	
Nine	679	D9Ncvs35	D325		-0.061	0.7929	21		-0.1045	0.6523	21		0.1526	0.5091	21	
Nine	680	Ets1	Ets-1		-0.055	0.7987	24		0.1107	0.6065	24		0.0623	0.7723	24	
Nine	681	Fli1	Fli-1		-0.055	0.7987	24		0.1107	0.6065	24		0.0623	0.7723	24	
Nine	682	Lap1	Lap-1		-0.051	0.8217	22		0.0372	0.8695	22		0.041	0.8561	22	
Nine	683	Xmmv2	Env-2, Xmmv-2		0.0524	0.8079	24		0.1426	0.5062	24		0.1446	0.5001	24	
Nine	684	Xmv16	Xmv-16		0.0903	0.6746	24		0.0609	0.7776	24		0.1048	0.626	24	
Nine	685	Upk2			0.0903	0.6746	24		0.0609	0.7776	24		0.1048	0.626	24	
Nine	686	Cd3d	D9Mit23, T3d		0.0903	0.6746	24		0.0609	0.7776	24		0.1048	0.626	24	
Nine	687	Cd3e	T3e		0.0903	0.6746	24		0.0609	0.7776	24		0.1048	0.626	24	
Nine	688	Apoa1	Apoa-1, Alp-1,		0.1397	0.515	24		0.0407	0.8504	24		0.1352	0.5287	24	
Nine	689	Ncam	D9Mit22		0.1068	0.6194	24		0.0928	0.6662	24		0.2755	0.1926	24	
Nine	690	Drd2	D2dr		0.1429	0.5054	24		0.012	0.9557	24		0.2366	0.2657	24	
Nine	691	D9Mit4	M151		0.3109	0.1821	20		-0.3077	0.1869	20		0.3112	0.1817	20	
Nine	692	D9Bir1			0.1429	0.5054	24		0.012	0.9557	24		0.2366	0.2657	24	
Nine	693	D9Byu1			0.3069	0.1446	24		-0.2398	0.259	24		0.3956	0.0557	24	
Nine	694	D9Mit21	Cypla2. D15		0.0591	0.8045	20		-0.5095	0.0217	20	X	0.3792	0.0992	20	
Nine	695	D9Ncvs49	D403		0.053	0.8149	22		-0.2029	0.365	22		0.4963	0.0188	22	X
Nine	696	D9Ncvs51	B404		0.0503	0.8196	23		-0.2189	0.3155	23		0.4179	0.0472	23	X
Nine	697	Xmv15	Xmv-15		0.0586	0.7858	24		-0.2623	0.2156	24		0.3869	0.0618	24	
Nine	698	Cal1h			0.0874	0.699	22		-0.2452	0.2714	22		0.4587	0.0318	22	X
Nine	699	D9Ncvs36	D201		0.0604	0.7842	23		-0.2984	0.1667	23		-0.017	0.9388	23	
Nine	700	d	Emv-3/Dbv		0.1703	0.4264	24		-0.2166	0.3094	24		0.0436	0.8399	24	
Nine	701	Saac			-0.2161	0.4783	13		-0.0396	0.8978	13		-0.115	0.7082	13	
Nine	702	D9Mit8	M211		0.1703	0.4264	24		-0.2166	0.3094	24		0.0436	0.8399	24	
Nine	703	Ctl2	Cdt, Ctl-2		-0.0106	0.9638	21		-0.2402	0.2942	21		-0.2099	0.3611	21	
Nine	704	D9Ncvs47	D488		0.1062	0.6379	22		-0.1895	0.3982	22		0.2407	0.2805	22	
Nine	705	D9Ncvs52	B487		0.1541	0.4935	22		-0.197	0.3795	22		0.2291	0.3051	22	
Nine	706	Gsta			0.2453	0.248	24		-0.1914	0.3704	24		0.079	0.7136	24	
Nine	707	D9Nds2	T30		0.2453	0.248	24		-0.1914	0.3704	24		0.079	0.7136	24	
Nine	708	Gst2-2	Gst-2.2		0.2453	0.248	24		-0.1914	0.3704	24		0.079	0.7136	24	
Nine	709	D9Byu2			0.2453	0.248	24		-0.1914	0.3704	24		0.079	0.7136	24	
Nine	710	D9Ncvs37	B232		0.1188	0.5893	23		-0.2013	0.357	23		0.0214	0.9229	23	
Nine	711	D9Ncvs38	D233		0.1188	0.5893	23		-0.2013	0.357	23		0.0214	0.9229	23	
Nine	712	D9Ncvs39	B80		0.1976	0.378	22		-0.2176	0.3306	22		0.072	0.7501	22	
Nine	713	D9Ncvs40	D81		0.1952	0.3965	21		-0.2821	0.2154	21		0.0648	0.7803	21	
Nine	714	D9Ncvs53	B401		0.0819	0.7037	24		-0.1375	0.5218	24		0.1087	0.6132	24	
Nine	715	Mod1	Mod-1		0.1788	0.4032	24		-0.1415	0.5095	24		0.1588	0.4586	24	
Nine	716	D9Mit11	L60		0.1788	0.4032	24		-0.1415	0.5095	24		0.1588	0.4586	24	
Nine	717	D9Rti1			0.168	0.4665	21		-0.24	0.2948	21		0.1393	0.547	21	
Nine	718	Pqm3	Pqm-3		0.1788	0.4032	24		-0.1415	0.5095	24		0.1588	0.4586	24	
Nine	719	D9Ncvs54	B653		0.1878	0.3794	24		-0.1726	0.42	24		0.2674	0.2065	24	
Nine	720	D9Byu3			0.1282	0.5506	24		-0.1297	0.5458	24		0.1933	0.3655	24	
Nine	721	D9Ncvs41	D14		0.2249	0.3022	23		-0.1185	0.5904	23		0.0734	0.7394	23	
Nine	722	D9Ncvs55	B467		0.2811	0.2438	19		-0.0569	0.8172	19		-0.0181	0.9415	19	
Nine	723	D9Mit12	M73		0.2963	0.1597	24		-0.1373	0.5223	24		0.0505	0.8147	24	
Nine	724	Rbp1	Crbp		0.3059	0.2492	16		-0.2543	0.3419	16		0.2852	0.2843	16	
Nine	725	Rbb2	Crbb-2		0.308	0.1528	23		-0.1676	0.4446	23		0.0177	0.9361	23	
Nine	726	D9Ncvs48	D594		0.308	0.1631	22		-0.1982	0.3766	22		0.1495	0.5066	22	
Nine	727	D9Ncvs42	B231		0.204	0.3626	22		-0.1508	0.5029	22		0.1658	0.4608	22	
Nine	728	Ltw3	Ltw-3		0.4424	0.0446	21	X	-0.2724	0.2322	21		0.249	0.2764	21	
Nine	729	D9Ncvs17	B308		0.2746	0.2162	22		-0.0171	0.9399	22		0.183	0.415	22	

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CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N	PCTWRED	P2ALPHA	P2N	AVGBASE1	P3ALPHA	P3N
Nine	730	D9Ncvs43	D309		0.3649	0.0869	23	-0.2094	0.3377	23	0.2809	0.1941	23
Nine	731	D9Byu4			0.3089	0.1419	24	-0.1866	0.3827	24	0.2439	0.2508	24
Nine	732	Ras12-2			0.3755	0.0706	24	-0.2133	0.317	24	0.2882	0.172	24
Nine	733	Gnat1	Gnat-1		0.331	0.1141	24	-0.2956	0.1608	24	0.2171	0.3083	24
Nine	734	Fv2	Fv-2		0.558	0.007	22 X	0.0297	0.8957	22	0.3111	0.1588	22
Nine	735	D9Mit15	M160		0.3575	0.1218	20	0.3024	0.195	20	0.4811	0.0318	20 X
Nine	736	D9Ncvs44	D343		0.0618	0.7795	23	0.0353	0.8728	23	0.2928	0.1752	23
Nine	737	D9Ncvs56	D471		0.0791	0.7134	24	0.0283	0.8957	24	0.3005	0.1537	24
Nine	738	D9Mit20	L64		0.2128	0.3677	20	0.041	0.8636	20	0.3808	0.0976	20
Nine	739	Bgls	Bgl-s		0.1409	0.5316	22	0.1125	0.6181	22	0.3166	0.1512	22
Nine	740	Xmmv31	Xmmv-31, Env-31		-0.0207	0.9234	24	-0.0539	0.8026	24	0.1734	0.4179	24
Nine	741	D9Ncvs45	B196		0.0157	0.9507	18	0.034	0.8935	18	-0.0873	0.7304	18
Nine	742	D9Mit18	M10		-0.045	0.8507	20	-0.3304	0.1548	20	0.3138	0.1779	20
Nine	743	D9Mit19	M157		-0.045	0.8507	20	-0.3304	0.1548	20	0.3138	0.1779	20
Nine	744	P40-3			-0.1224	0.569	24	-0.079	0.7136	24	0.156	0.4668	24
Nine	745	D9Ncvs50	D599		0.0616	0.7749	24	0.0221	0.9183	24	0.0241	0.911	24
Nine	746	D9Ncvs57	B600		0.0616	0.7749	24	0.0221	0.9183	24	0.0241	0.911	24
Nine	747	Cck			0.0098	0.9637	24	0.0321	0.8815	24	0.0607	0.7783	24
Nine	748	D9Byu6			0.0235	0.9134	24	-0.0459	0.8313	24	0.0698	0.746	24
Nine	749	D9Ncvs46	D9		0.109	0.629	22	-0.2137	0.3396	22	0.1824	0.4164	22
Nine	750	D9Byu5			0.1682	0.4321	24	-0.2159	0.3109	24	0.2115	0.3211	24
Nine	751	Hm2	Hm-2		0.1682	0.4321	24	-0.2159	0.3109	24	0.2115	0.3211	24
Nine	752	Tel9q			0.1241	0.5634	24	-0.1159	0.5898	24	0.158	0.4608	24
Ten	753	D10Byu18	D0Byu18		0.1858	0.3846	24	-0.2595	0.2208	24	0.3939	0.0569	24
Ten	754	Hpmv5	Hpmv-5		0.1506	0.4825	24	-0.2098	0.3252	24	0.2955	0.161	24
Ten	755	Tapls3-21			-0.1709	0.4247	24	-0.1879	0.3793	24	0.2487	0.2413	24
Ten	756	D10Ncvs34	D655		-0.0331	0.8807	23	-0.1886	0.3887	23	0.1442	0.5116	23
Ten	757	D10Ncvs28	D83		0.1852	0.3975	23	-0.2261	0.2995	23	0.3231	0.1326	23
Ten	758	D10Byu1			0.0124	0.9543	24	-0.1586	0.4592	24	0.3031	0.15	24
Ten	759	Hmg1-rs2			-0.0898	0.6985	21	-0.1247	0.5902	21	0.4102	0.0648	21
Ten	760	D10Bir2			-0.0569	0.7917	24	-0.1196	0.5779	24	0.2962	0.1599	24
Ten	761	Xmmv15	Xmmv-15		-0.0745	0.7419	22	-0.3642	0.0956	22	0.2776	0.2111	22
Ten	762	D10Ncvs36	B676		0.0617	0.7747	24	-0.2127	0.3184	24	0.1535	0.4739	24
Ten	763	D10Mit3	A114		0.0171	0.9429	20	-0.1669	0.4818	20	0.0095	0.9684	20
Ten	764	D10Ncvs35	D549		-0.2184	0.3052	24	0.1148	0.5933	24	-0.0495	0.8183	24
Ten	765	D10Mit15	Sqr3. D30		0.1489	0.5309	20	-0.0466	0.8452	20	0.22	0.3514	20
Ten	766	Fps15	Fps1-5		-0.0105	0.9612	24	0.202	0.3439	24	0.1795	0.4014	24
Ten	767	D10Mit11	A88		0.132	0.5791	20	-0.0483	0.8398	20	0.2485	0.2907	20
Ten	768	D10Mit10	M7		0.132	0.5791	20	-0.0483	0.8398	20	0.2485	0.2907	20
Ten	769	Hal			-0.0248	0.9105	23	0.2427	0.2644	23	0.1998	0.3607	23
Ten	770	D10Bir1			-0.1933	0.3656	24	0.2592	0.2213	24	-0.0484	0.8224	24
Ten	771	Xmv31	Xmv-31		0.0677	0.7534	24	0.3086	0.1423	24	0.0432	0.8411	24
Ten	772	Kcnc2			0.1629	0.4468	24	0.3072	0.1443	24	0.0918	0.6697	24
Ten	773	Ms15-8			0.129	0.5574	23	0.34	0.1124	23	0.0896	0.6842	23
Ten	774	D10Mit14	A124		0.3349	0.1489	20	0.093	0.6965	20	0.0123	0.9591	20
Ten	775	Mdm1	Mdm-1		0.0938	0.7113	18	0.238	0.3415	18	-0.025	0.9217	18
Ten	776	D10Ncvs29	B238		0.1197	0.5957	22	0.0621	0.7836	22	-0.0489	0.829	22
Ten	777	D10Ncvs30	B24		-0.0821	0.7095	23	-0.1168	0.5956	23	-0.0428	0.8464	23
Ten	778	D10Ncvs31	D29		-0.0821	0.7095	23	-0.1168	0.5956	23	-0.0428	0.8464	23
Ten	779	D10Ncvs32	B49		-0.0821	0.7095	23	-0.1168	0.5956	23	-0.0428	0.8464	23
Ten	780	D10Ncvs33	D50		-0.1208	0.5922	22	-0.0996	0.6593	22	-0.0471	0.8351	22
Ten	781	Ms6-3			-0.1419	0.5184	23	-0.089	0.6863	23	-0.0622	0.7779	23
Eleven	782	D11Ncvs74	B452		0.2544	0.2415	23	0.286	0.1858	23	-0.3277	0.127	23
Eleven	783	D11Ncvs43	D168		0.3481	0.1326	20	0.3139	0.1778	20	-0.2753	0.2401	20
Eleven	784	Pmv22	Pmv-22		0.2996	0.155	24	0.2538	0.2314	24	-0.2736	0.1958	24
Eleven	785	D11Mit2	L14		0.5352	0.0182	19 X	0.2335	0.336	19	-0.265	0.2729	19
Eleven	786	D11Byu1			0.3657	0.0788	24	0.2116	0.3209	24	-0.2628	0.2148	24
Eleven	787	D11Ncvs73	D565		0.304	0.1585	23	0.2128	0.3297	23	-0.3377	0.1151	23
Eleven	788	D11Ncvs42	D124		0.305	0.1571	23	0.2088	0.339	23	-0.3009	0.1629	23
Eleven	789	Gins			0.1629	0.4468	24	0.2484	0.2419	24	-0.1859	0.3845	24
Eleven	790	D11Byu3			0.1228	0.5675	24	0.3225	0.1243	24	-0.2588	0.222	24
Eleven	791	P40-rs5	P40-5		0.1111	0.6052	24	0.2232	0.2944	24	-0.2778	0.1888	24
Eleven	792	D11Ncvs69	D451		0.1403	0.523	23	0.1171	0.5947	23	-0.3772	0.076	23
Eleven	793	U2afbp-rs	D461, D11Ncvs75		-0.0766	0.7219	24	0.247	0.2445	24	-0.1646	0.4421	24
Eleven	794	D11Byu2			0.1111	0.6052	24	0.2232	0.2944	24	-0.2778	0.1888	24
Eleven	795	D11Byu4			0.1111	0.6052	24	0.2232	0.2944	24	-0.2778	0.1888	24
Eleven	796	Hba			0.1454	0.5186	22	0.2835	0.2011	22	-0.2982	0.1776	22
Eleven	797	D11Mit51			0.1235	0.5654	24	-0.0655	0.7609	24	0.0471	0.8271	24
Eleven	798	D11Mit20			-0.003	0.9892	23	0.0064	0.977	23	0.1694	0.4397	23
Eleven	799	D11Ncvs70	D583		-0.221	0.2995	24	-0.384	0.0639	24	0.1639	0.4441	24
Eleven	800	D11Ncvs44	D344		-0.1866	0.3939	23	-0.2928	0.1752	23	0.1979	0.3654	23
Eleven	801	D11Ncvs45	D264		-0.2036	0.3514	23	-0.3986	0.0596	23	0.1815	0.4073	23
Eleven	802	D11Ncvs46	D155		-0.0169	0.9437	20	-0.3017	0.1961	20	0.2538	0.2802	20
Eleven	803	D11Ncvs47	B156		-0.0105	0.9641	21	-0.2794	0.2199	21	0.2464	0.2816	21
Eleven	804	D11Ncvs48	B180		-0.1022	0.6425	23	-0.399	0.0593	23	0.231	0.2888	23
Eleven	805	Hist3			-0.1029	0.6402	23	-0.3949	0.0622	23	0.1978	0.3657	23
Eleven	806	Il5	D11Nds9, Il-5		-0.1412	0.5106	24	-0.317	0.1312	24	0.2926	0.1654	24
Eleven	807	D11H4S10	HD4P, D4S10h		-0.1412	0.5106	24	-0.317	0.1312	24	0.2926	0.1654	24
Eleven	808	D11Ncvs76	B597		-0.0544	0.8054	23	-0.4049	0.0553	23	0.115	0.6013	23
Eleven	809	D11Mit23			-0.1412	0.5106	24	-0.317	0.1312	24	0.2926	0.1654	24
Eleven	810	Sparc			-0.1553	0.4687	24	-0.2414	0.2557	24	0.2835	0.1794	24

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CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N		PCTWRED	P2ALPHA	P2N		AVGBASE1	P3ALPHA	P3N
Eleven	811	Rcvrn	RD23		-0.1316	0.5494	23		-0.0967	0.6607	23		0.0174	0.9371	23
Eleven	812	D11Mit4	A124		-0.0713	0.7652	20		-0.2164	0.3594	20		0.0607	0.7993	20
Eleven	813	D11Ncvs71	D454		-0.1062	0.6849	17		0.0354	0.8926	17		0.0748	0.7755	17
Eleven	814	D11Ncvs77	B453		-0.1075	0.6812	17		0.0436	0.8681	17		-0.0329	0.9001	17
Eleven	815	D11Ncvs49	B159		-0.2181	0.3173	23		-0.0193	0.9303	23		0.03	0.8918	23
Eleven	816	D11Ncvs50	B194		-0.2181	0.3173	23		-0.0193	0.9303	23		0.03	0.8918	23
Eleven	817	D11Ncvs51	B313		-0.1953	0.3837	22		-0.0014	0.9952	22		0.0052	0.9816	22
Eleven	818	D11Ncvs52	D314		-0.2181	0.3173	23		-0.0193	0.9303	23		0.03	0.8918	23
Eleven	819	D11Mit29			-0.2172	0.3194	23		0.0108	0.961	23		-0.0162	0.9415	23
Eleven	820	Glut4	D11Mit15, Glut-		-0.2407	0.2573	24		-0.0076	0.972	24		0.0092	0.966	24
Eleven	821	D11Mcl	KA2, D11McC1		-0.2407	0.2573	24		-0.0076	0.972	24		0.0092	0.966	24
Eleven	822	D11Mit15	Glut-4. D5		-0.1789	0.4505	20		-0.2322	0.3245	20		0.0043	0.9858	20
Eleven	823	Zfp3	Zfp-3, Fnp-1		-0.2407	0.2573	24		-0.0076	0.972	24		0.0092	0.966	24
Eleven	824	Asqr1	Asqr-1		-0.219	0.3154	23		-0.0144	0.9481	23		-0.0135	0.9512	23
Eleven	825	Asqr2	Asqr-2		-0.219	0.3154	23		-0.0144	0.9481	23		-0.0135	0.9512	23
Eleven	826	Trp53			-0.2407	0.2573	24		-0.0076	0.972	24		0.0092	0.966	24
Eleven	827	Atp1b2	Atpb-2, Amog		-0.2407	0.2573	24		-0.0076	0.972	24		0.0092	0.966	24
Eleven	828	Acrb			-0.1896	0.3863	23		-0.1477	0.5011	23		0.0825	0.7083	23
Eleven	829	D11Nds1			-0.1706	0.4364	23		-0.0099	0.9644	23		0.0201	0.9275	23
Eleven	830	Mpmv2	Mpmv-2		-0.176	0.4106	24		-0.1033	0.6309	24		0.0519	0.8095	24
Eleven	831	Brp8	Brp-8		-0.1482	0.5572	18		-0.0136	0.9574	18		0.1776	0.4807	18
Eleven	832	Iabls2-11			-0.176	0.4106	24		-0.1033	0.6309	24		0.0519	0.8095	24
Eleven	833	D11Ncvs78	B684		-0.4498	0.0533	19		0.0552	0.8225	19		0.0424	0.8631	19
Eleven	834	D11Bir1			-0.4402	0.0313	24	X	-0.0819	0.7036	24		0.1404	0.5128	24
Eleven	835	Xmv42	Xmv-42		-0.4402	0.0313	24	X	-0.0819	0.7036	24		0.1404	0.5128	24
Eleven	836	Mpmv4	Xmmv-3, Env-3,		-0.4402	0.0313	24	X	-0.0819	0.7036	24		0.1404	0.5128	24
Eleven	837	Tca3	Tca-3		-0.4402	0.0313	24	X	-0.0819	0.7036	24		0.1404	0.5128	24
Eleven	838	Mip1a			-0.4402	0.0313	24	X	-0.0819	0.7036	24		0.1404	0.5128	24
Eleven	839	Mip1b			-0.4402	0.0313	24	X	-0.0819	0.7036	24		0.1404	0.5128	24
Eleven	840	D11Mit38	Po		-0.4402	0.0313	24	X	-0.0819	0.7036	24		0.1404	0.5128	24
Eleven	841	Mpo			-0.3709	0.0893	22		-0.2569	0.2485	22		0.2668	0.23	22
Eleven	842	D11Ncvs53	D195		-0.4768	0.0214	23	X	-0.0747	0.7347	23		0.1278	0.5612	23
Eleven	843	D11Ncvs54	B258		-0.4768	0.0214	23	X	-0.0747	0.7347	23		0.1278	0.5612	23
Eleven	844	D11Ncvs55	D256		-0.4227	0.0563	21		-0.2411	0.2924	21		0.2445	0.2854	21
Eleven	845	D11Ncvs56	B277		-0.4768	0.0214	23	X	-0.0747	0.7347	23		0.1278	0.5612	23
Eleven	846	D11Ncvs80	D622		-0.5532	0.0062	23	X	-0.0266	0.9041	23		0.1407	0.5219	23
Eleven	847	D11Ncvs81	B520		-0.2662	0.2087	24		0.0138	0.9491	24		0.0157	0.9421	24
Eleven	848	D11Ncvs57	D276		-0.4768	0.0214	23	X	-0.0747	0.7347	23		0.1278	0.5612	23
Eleven	849	D11Ncvs58	D249		-0.3377	0.1243	22		0.1161	0.607	22		0.2872	0.195	22
Eleven	850	D11Ncvs59	B248		-0.3181	0.139	23		0.1702	0.4376	23		0.2889	0.1812	23
Eleven	851	D11Ncvs79	D407		-0.2539	0.3812	14		0.036	0.9028	14		0.0712	0.8089	14
Eleven	852	D11Ncvs82	B406		-0.0441	0.8919	12		0.3556	0.2566	12		-0.1721	0.5929	12
Eleven	853	D11Ncvs72	D645		-0.1141	0.632	20		0.2456	0.2966	20		-0.0078	0.9739	20
Eleven	854	Hoxb	Hox-2		0.0138	0.9489	24		0.162	0.4495	24		0.059	0.7841	24
Eleven	855	D11Ncvs83	D615		-0.0267	0.9014	24		-0.0712	0.7411	24		0.0266	0.9017	24
Eleven	856	D11Mit14	AntP91A. D2		0.0398	0.8675	20		-0.4274	0.0601	20		0.2129	0.3675	20
Eleven	857	P91A			-0.0763	0.7232	24		-0.0615	0.7753	24		0.0635	0.7683	24
Eleven	858	Krt1	Krt-1		-0.1464	0.5051	23		-0.0359	0.8707	23		0.0793	0.719	23
Eleven	859	Mpmv8	Mpmv-8		0.0525	0.8074	24		-0.0254	0.9064	24		-0.1073	0.6178	24
Eleven	860	Gfap	D11Nds7		0.2116	0.3208	24		-0.2731	0.1966	24		0.0435	0.84	24
Eleven	861	D11Nds2			0.2116	0.3208	24		-0.2731	0.1966	24		0.0435	0.84	24
Eleven	862	Empb3			0.1548	0.4702	24		-0.1861	0.384	24		0.1163	0.5885	24
Eleven	863	Myla			0.3419	0.102	24		-0.1837	0.3902	24		0.0866	0.6876	24
Eleven	864	D11Ncvs84	D518		0.3779	0.0829	22		0.2399	0.2822	22		0.1313	0.5603	22
Eleven	865	D11Ncvs62	D166		0.0745	0.7355	23		0.2948	0.1721	23		0.2521	0.2459	23
Eleven	866	D11Ncvs63	B167		0.0153	0.9461	22		0.4978	0.0184	22	X	0.2117	0.3443	22
Eleven	867	D11Ncvs61	D130		-0.0111	0.9607	22		0.0091	0.968	22		0.2687	0.2267	22
Eleven	868	D11Ncvs60	D19		0.1609	0.498	20		0.1408	0.5539	20		0.3662	0.1123	20
Eleven	869	D11Jknle			0.1351	0.5701	20		0.1699	0.474	20		0.3708	0.1075	20
Eleven	870	Es3	Es-3		0.1049	0.6256	24		0.2715	0.1994	24		0.2636	0.2133	24
Eleven	871	Tk1	Tk-1		0.1049	0.6256	24		0.2715	0.1994	24		0.2636	0.2133	24
Eleven	872	D11Mit48			0.1049	0.6256	24		0.2715	0.1994	24		0.2636	0.2133	24
Eleven	873	Gaa			0.1049	0.6256	24		0.2715	0.1994	24		0.2636	0.2133	24
Eleven	874	D11Ncvs64	D37		0.1134	0.6438	19		0.2438	0.3146	19		0.2457	0.3106	19
Eleven	875	D11Ncvs65	B38		0.1175	0.6024	22		0.2971	0.1794	22		0.277	0.212	22
Eleven	876	D11Ncvs66	B221		0.2312	0.3132	21		0.4179	0.0594	21		0.1057	0.6483	21
Eleven	877	D11Ncvs68	D183		-0.2366	0.289	22		0.1612	0.4735	22		0.172	0.4442	22
Eleven	878	D11Ncvs67	B182		-0.028	0.8993	23		0.1752	0.424	23		0.21	0.3362	23
Twelve	879	D12Mit1	M50		0.2967	0.2039	20		0.3568	0.1225	20		0.2309	0.3274	20
Twelve	880	Tpo			0.0358	0.8742	22		0.2004	0.3712	22		0.1667	0.4583	22
Twelve	881	Nmyc1	Nmyc-1, Nmyc		-0.0676	0.7538	24		0.1714	0.4232	24		0.1998	0.3492	24
Twelve	882	D12Nyu10			-0.0549	0.7987	24		0.2736	0.1957	24		0.2165	0.3095	24
Twelve	883	D12Nyu7			-0.0803	0.7158	23		0.285	0.1875	23		0.277	0.2007	23
Twelve	884	D12Byu1			-0.0549	0.7987	24		0.2736	0.1957	24		0.2165	0.3095	24
Twelve	885	D12Ncvs28	D172		-0.0936	0.671	23		0.3681	0.084	23		0.1982	0.3645	23
Twelve	886	D12Ncvs29	D96		-0.0936	0.671	23		0.3681	0.084	23		0.1982	0.3645	23
Twelve	887	D12Ncvs30	D97		0.0231	0.9188	22		0.398	0.0666	22		0.2425	0.2768	22
Twelve	888	D12Ncvs31	B94		-0.099	0.661	22		0.3892	0.0734	22		0.1576	0.4835	22
Twelve	889	D12Ncvs32	D93		-0.0936	0.671	23		0.3681	0.084	23		0.1982	0.3645	23
Twelve	890	D12Ncvs2	B87		-0.0936	0.671	23		0.3681	0.084	23		0.1982	0.3645	23
Twelve	891	D12Ncvs33	D88		-0.0936	0.671	23		0.3681	0.084	23		0.1982	0.3645	23

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N		PCTWRED	P2ALPHA	P2N		AVGBASE1	P3ALPHA	P3N
Twelve	892	D12Ncvs1	B76		-0.0936	0.671	23		0.3681	0.084	23		0.1982	0.3645	23
Twelve	893	D12Ncvs34	B47		-0.0936	0.671	23		0.3681	0.084	23		0.1982	0.3645	23
Twelve	894	D12Ncvs35	B356		-0.0936	0.671	23		0.3681	0.084	23		0.1982	0.3645	23
Twelve	895	D12Ncvs36	D286		-0.0936	0.671	23		0.3681	0.084	23		0.1982	0.3645	23
Twelve	896	D12Ncvs3	B289		-0.0936	0.671	23		0.3681	0.084	23		0.1982	0.3645	23
Twelve	897	D12Ncvs37	D352		-0.023	0.919	22		0.3263	0.1383	22		0.294	0.1842	22
Twelve	898	Odc	D12Ndsl1		-0.0693	0.7475	24		0.354	0.0896	24		0.2087	0.3278	24
Twelve	899	Rrm2			-0.0693	0.7475	24		0.354	0.0896	24		0.2087	0.3278	24
Twelve	900	D12Ncvs38	D98		-0.1688	0.4413	23		0.3532	0.0983	23		0.0315	0.8865	23
Twelve	901	D12Ncvs39	D311		-0.1577	0.4832	22		0.3304	0.1331	22		0.0135	0.9525	22
Twelve	902	D12Nyu2			0.0457	0.8359	23		0.5008	0.0149	23	X	0.0778	0.7241	23
Twelve	903	Ahr	Ah		-0.0974	0.6506	24		0.2138	0.3157	24		-0.0811	0.7063	24
Twelve	904	Ahr	Ah		0.0916	0.6703	24		0.1474	0.4919	24		-0.0585	0.7861	24
Twelve	905	D12Mit2	M27		0.1418	0.5086	24		0.1583	0.4601	24		-0.0527	0.8067	24
Twelve	906	D12Ncvs44	D602		0.1418	0.5086	24		0.1583	0.4601	24		-0.0527	0.8067	24
Twelve	907	D12Ncvs46	B603		0.1563	0.4657	24		0.2626	0.215	24		-0.0371	0.8633	24
Twelve	908	D12Ncvs47	B635		0.3517	0.092	24		0.3342	0.1105	24		-0.1668	0.4359	24
Twelve	909	Eif4e	eIF-4E		0.3762	0.07	24		0.3911	0.0588	24		-0.1901	0.3735	24
Twelve	910	D12Ncvs41	B269		0.5071	0.0135	23	X	0.119	0.5885	23		-0.0668	0.7619	23
Twelve	911	D12Ncvs42	D5		0.5071	0.0135	23	X	0.119	0.5885	23		-0.0668	0.7619	23
Twelve	912	D12Ncvs40	D267		0.5732	0.0203	16	X	0.0004	0.9989	16		0.0201	0.941	16
Twelve	913	Iapls2-10			0.5446	0.0059	24	X	0.1463	0.4953	24		-0.0389	0.8568	24
Twelve	914	D12Nyu1			0.5967	0.0027	23	X	0.1471	0.503	23		-0.1009	0.6468	23
Twelve	915	Pmv3	Pmv-3		0.5446	0.0059	24	X	0.1463	0.4953	24		-0.0389	0.8568	24
Twelve	916	Rnula2	U1-9,10		0.5446	0.0059	24	X	0.1463	0.4953	24		-0.0389	0.8568	24
Twelve	917	D12Mit3	L41		0.257	0.2741	20		0.4326	0.0567	20		0.0729	0.76	20
Twelve	918	D12Mit5	L58		0.0185	0.9317	24		0.4756	0.0188	24	X	-0.0464	0.8297	24
Twelve	919	D12Ncvs48	B523		-0.0032	0.9885	23		0.2308	0.2894	23		-0.0533	0.809	23
Twelve	920	Mtv9	Mtv-9		0.0219	0.9192	24		0.1978	0.3541	24		-0.0076	0.9719	24
Twelve	921	Chga			0.061	0.7823	23		0.3553	0.0962	23		-0.0543	0.8057	23
Twelve	922	D12Mit7	M62		-0.1042	0.628	24		0.2635	0.2135	24		-0.131	0.5418	24
Twelve	923	Spi1	Aat, Spi-1, Pre		-0.1042	0.628	24		0.2635	0.2135	24		-0.131	0.5418	24
Twelve	924	Aat	pC1.2		-0.0855	0.6981	23		0.2658	0.2203	23		-0.1824	0.4047	23
Twelve	925	D12Ncvs45	D581		-0.0608	0.7827	23		0.1283	0.5596	23		-0.2242	0.3038	23
Twelve	926	Spi2	Contr, Spi-2		-0.0779	0.7173	24		0.3685	0.0765	24		-0.213	0.3176	24
Twelve	927	D12Ncvs49	B664		-0.0291	0.8926	24		0.3569	0.0869	24		-0.2483	0.242	24
Twelve	928	Cbg			-0.0904	0.6745	24		0.4052	0.0495	24	X	-0.0273	0.8991	24
Twelve	929	Mpmv24	Xmmv-34,Env-34,		-0.3194	0.1282	24		0.3629	0.0813	24		-0.3011	0.1527	24
Twelve	930	D12Ncvs43	D355		-0.2604	0.2419	22		0.2776	0.211	22		-0.1819	0.4178	22
Twelve	931	D12H14S17			-0.3448	0.0989	24		0.2579	0.2236	24		-0.2188	0.3043	24
Twelve	932	Ckb	Ck-3		-0.3448	0.0989	24		0.2579	0.2236	24		-0.2188	0.3043	24
Twelve	933	Crip			-0.325	0.1302	23		0.2305	0.2901	23		-0.3516	0.1	23
Twelve	934	D12Ndsl2	T1		-0.2647	0.2594	20		0.2693	0.2508	20		-0.2234	0.3437	20
Twelve	935	D12Mit8	Igh-C. D7		-0.2647	0.2594	20		0.2693	0.2508	20		-0.2234	0.3437	20
Twelve	936	Igh-C	Igh-C		-0.327	0.1189	24		0.256	0.2273	24		-0.2933	0.1643	24
Twelve	937	Igh-1			-0.327	0.1189	24		0.256	0.2273	24		-0.2933	0.1643	24
Twelve	938	Bcga			-0.0524	0.8214	21		0.1318	0.5689	21		-0.308	0.1744	21
Twelve	939	Igh-Ox			-0.3233	0.1529	21		-0.0933	0.6875	21		-0.1346	0.5609	21
Twelve	940	Igh-Sa2			-0.2779	0.1886	24		0.3002	0.1541	24		-0.2765	0.191	24
Twelve	941	Igh-Sa4			-0.3655	0.0791	24		0.0816	0.7045	24		-0.2701	0.2018	24
Twelve	942	D12Byu2			-0.417	0.0427	24	X	0.0386	0.8578	24		-0.2888	0.171	24
Twelve	943	D12Nyu13			-0.417	0.0427	24	X	0.0386	0.8578	24		-0.2888	0.171	24
Twelve	944	D12N1	DAbI-12		-0.417	0.0427	24	X	0.0386	0.8578	24		-0.2888	0.171	24
Twelve	945	Igh-V			-0.417	0.0427	24	X	0.0386	0.8578	24		-0.2888	0.171	24
Twelve	946	Igh-V3609			-0.417	0.0427	24	X	0.0386	0.8578	24		-0.2888	0.171	24
Twelve	947	Igh-Vdx			-0.4249	0.0433	23	X	0.0237	0.9147	23		-0.2451	0.2596	23
Twelve	948	Igh-Nbp			-0.4061	0.0607	22		0.1013	0.6537	22		-0.3199	0.1467	22
Twelve	949	Igh-Gte			-0.4061	0.0607	22		0.1013	0.6537	22		-0.3199	0.1467	22
Twelve	950	Odc-rs8			-0.417	0.0427	24	X	0.0386	0.8578	24		-0.2888	0.171	24
Twelve	951	Igh-Np			-0.4061	0.0607	22		0.1013	0.6537	22		-0.3199	0.1467	22
Twelve	952	Igh-Bgl			-0.3639	0.151	17		0.0206	0.9373	17		-0.2074	0.4244	17
Twelve	953	Igh-Sfc			-0.3813	0.0881	21		0.097	0.6757	21		-0.2333	0.3087	21
Twelve	954	Xmmv25	Env-25, Xmmv-25		-0.3886	0.0669	23		-0.2193	0.3148	23		0.1028	0.6406	23
Thirteen	955	Tcrg			0.3048	0.1573	23		-0.2496	0.2508	23		0.0139	0.9496	23
Thirteen	956	D13Mit3	M79		0.3238	0.1638	20		0.3468	0.1341	20		-0.084	0.7247	20
Thirteen	957	Hist1			0.3403	0.112	23		-0.0137	0.9506	23		0.0381	0.8629	23
Thirteen	958	D13Kyo1	Hh1tts		0.2632	0.225	23		0.0934	0.6715	23		0.0061	0.9778	23
Thirteen	959	D13Byu1			-0.0629	0.7703	24		0.1223	0.5691	24		-0.1284	0.5498	24
Thirteen	960	Il9	D24, D13Mit13,I		0.0081	0.9713	22		0.2949	0.1827	22		0.0623	0.7829	22
Thirteen	961	Ms6-1			-0.0515	0.8153	23		0.2992	0.1655	23		-0.0375	0.865	23
Thirteen	962	Iapls1-13			-0.0918	0.6695	24		0.3128	0.1366	24		-0.0413	0.8482	24
Thirteen	963	D13Ncvs44	B417		-0.3945	0.0768	21		0.3762	0.0928	21		-0.0094	0.9677	21
Thirteen	964	Tpmt			-0.5235	0.0087	24	X	0.3079	0.1432	24		-0.0121	0.9551	24
Thirteen	965	D13Ncvs45	B515		-0.3398	0.1043	24		0.1392	0.5166	24		0.0003	0.999	24
Thirteen	966	Pmv41	Pmv-41		-0.2165	0.3096	24		0.255	0.2292	24		-0.0934	0.6643	24
Thirteen	967	Hmg1-rs3			-0.1994	0.3736	22		0.2196	0.326	22		-0.1793	0.4248	22
Thirteen	968	Hmg1-rs5			-0.2981	0.2152	19		0.2758	0.253	19		0.0238	0.9231	19
Thirteen	969	D13Ncvs33	B8		-0.2018	0.3558	23		0.2513	0.2474	23		-0.0827	0.7076	23
Thirteen	970	D13Ncvs34	D23		-0.2018	0.3558	23		0.2513	0.2474	23		-0.0827	0.7076	23
Thirteen	971	Iapls1-1			-0.0526	0.8073	24		-0.004	0.9852	24		0.068	0.7523	24
Thirteen	972	D13Ncvs35	B211		-0.3041	0.1688	22		0.3377	0.1243	22		-0.0881	0.6967	22

SAG QTL w/ 1994 marker set

analysis run 12-16-94 by SRM

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N		PCTWRED	P2ALPHA	P2N		AVGBASE1	P3ALPHA	P3N
Thirteen	973	Srd5a1	Srd5a-1		-0.3033	0.1496	24		0.3651	0.0794	24		-0.1276	0.5522	24
Thirteen	974	D13Byu2			-0.3225	0.1539	21		0.3725	0.0964	21		0.0365	0.8751	21
Thirteen	975	D13Byu3			-0.2353	0.2684	24		0.3995	0.0531	24		0.0471	0.827	24
Thirteen	976	Xmv13	Xmv-13		-0.1426	0.5061	24		0.1102	0.6083	24		0.0324	0.8805	24
Thirteen	977	D13Mit11	A91		-0.2179	0.356	20		0.0917	0.7005	20		0.0011	0.9965	20
Thirteen	978	D13Ncvs46	B530		-0.2059	0.3579	22		0.0081	0.9713	22		0.1066	0.6368	22
Thirteen	979	D13Mit9	M147		-0.3293	0.1345	22		0.2374	0.2874	22		-0.0137	0.9518	22
Thirteen	980	Rasa			-0.2598	0.2313	23		0.0416	0.8504	23		0.0724	0.7428	23
Thirteen	981	P198-13			-0.2407	0.2571	24		0.0387	0.8577	24		0.0392	0.8556	24
Thirteen	982	As1	As-1		-0.2393	0.2835	22		0.0711	0.7532	22		0.09	0.6905	22
Thirteen	983	Lth1	Lth-1		-0.2393	0.2835	22		0.0711	0.7532	22		0.09	0.6905	22
Thirteen	984	D13Byu4			-0.3058	0.1462	24		0.0784	0.7159	24		0.03	0.8893	24
Thirteen	985	D13Mc1	D13McC1		-0.2407	0.2571	24		0.0387	0.8577	24		0.0392	0.8556	24
Thirteen	986	D13McC2	KB3, D13McC2		-0.2407	0.2571	24		0.0387	0.8577	24		0.0392	0.8556	24
Thirteen	987	D13Bir1			-0.1702	0.4267	24		-0.0404	0.8515	24		-0.0867	0.6872	24
Thirteen	988	D13Ncvs9	B348		-0.2168	0.3205	23		-0.037	0.8671	23		-0.04	0.8562	23
Thirteen	989	D13Ncvs36	D350		-0.2168	0.3205	23		-0.037	0.8671	23		-0.04	0.8562	23
Thirteen	990	D13Ncvs37	D188		-0.2168	0.3205	23		-0.037	0.8671	23		-0.04	0.8562	23
Thirteen	991	Iapls2-6			-0.0788	0.7145	24		-0.147	0.4931	24		0.0675	0.7538	24
Thirteen	992	Pmv9	Pmv-9		-0.0784	0.7221	23		-0.1145	0.603	23		0.0931	0.6728	23
Thirteen	993	Iapls3-14			-0.0716	0.7394	24		-0.1335	0.5341	24		0.0626	0.7715	24
Thirteen	994	D13Ncvs39	B213		0.0332	0.8805	23		-0.2472	0.2554	23		-0.0824	0.7085	23
Thirteen	995	D13Ncvs42	D591		0.0277	0.9024	22		-0.1483	0.5102	22		-0.0755	0.7386	22
Thirteen	996	D13Ncvs47	B592		0.0021	0.9923	24		-0.1564	0.4655	24		0.0207	0.9234	24
Thirteen	997	D13Ncvs48	B522		-0.1319	0.5487	23		-0.1565	0.4757	23		0.0361	0.8701	23
Thirteen	998	Iapls1-8			-0.1548	0.4702	24		0.0879	0.683	24		-0.1295	0.5463	24
Thirteen	999	Hmg1-rs4			-0.174	0.4272	23		-0.1658	0.4495	23		-0.1239	0.5732	23
Thirteen	1000	D13Ncvs40	D296		-0.132	0.5481	23		-0.3204	0.136	23		0.2188	0.3159	23
Thirteen	1001	D13Ncvs41	D92		-0.132	0.5481	23		-0.3204	0.136	23		0.2188	0.3159	23
Thirteen	1002	D13Ncvs43	D638		-0.102	0.6354	24		-0.3225	0.1243	24		0.23	0.2795	24
Thirteen	1003	D13Byu5	DOByu10		-0.102	0.6354	24		-0.3225	0.1243	24		0.23	0.2795	24
Thirteen	1004	Tel13q	TB-3, TD-2		-0.0342	0.8738	24		-0.2823	0.1814	24		0.174	0.4163	24
Thirteen	1005	Tel8-31			-0.1177	0.584	24		-0.2932	0.1644	24		-0.1068	0.6194	24
Thirteen		D13Mit139			-0.3612	0.1177	20		0.1482	0.533	20		0.1369	0.5648	20
Fourteen	1006	D14Byu1			-0.1542	0.472	24		-0.3382	0.106	24		0.0122	0.955	24
Fourteen	1007	Odc-rs9			-0.0517	0.8105	24		-0.3439	0.0999	24		0.0499	0.8168	24
Fourteen	1008	D14Ncvs41	B576		0.1041	0.735	13		-0.0956	0.756	13		0.3809	0.1991	13
Fourteen	1009	D14Ncvs43	B538		-0.091	0.6871	22		-0.3249	0.1401	22		-0.028	0.9014	22
Fourteen	1010	Rarb			-0.0517	0.8105	24		-0.3439	0.0999	24		0.0499	0.8168	24
Fourteen	1011	D14Ncvs42	D575		0.0682	0.8167	14		-0.1807	0.5364	14		-0.2277	0.4337	14
Fourteen	1012	P40-rs6	P40-6		-0.0517	0.8105	24		-0.3439	0.0999	24		0.0499	0.8168	24
Fourteen	1013	D14Mit1	A103		-0.1165	0.6247	20		-0.2576	0.2729	20		-0.1033	0.6646	20
Fourteen	1014	D14Ncvs44	B648		0.1544	0.4713	24		-0.335	0.1095	24		-0.0102	0.9623	24
Fourteen	1015	Plau	D14Nds1		-0.0652	0.7622	24		-0.4381	0.0322	24	X	0.0299	0.8899	24
Fourteen	1016	D14Mit2	A24		-0.1444	0.5436	20		-0.3592	0.1198	20		-0.1357	0.5685	20
Fourteen	1017	D14Nds1	Plau. T10		-0.1444	0.5436	20		-0.3592	0.1198	20		-0.1357	0.5685	20
Fourteen	1018	D14Ncvs34	D176		-0.4118	0.1272	15		-0.132	0.639	15		0.2503	0.3683	15
Fourteen	1019	D14Pas1			-0.3428	0.1093	23		-0.3027	0.1603	23		0.2692	0.2142	23
Fourteen	1020	Mtv11	Mtv-11, Mtv-7a		-0.3056	0.1464	24		-0.2502	0.2384	24		0.3199	0.1276	24
Fourteen	1021	D14Mit6	A119		-0.2337	0.3214	20		-0.4871	0.0294	20	X	0.4155	0.0685	20
Fourteen	1022	Ms15-7			-0.2591	0.2326	23		-0.2832	0.1903	23		0.2564	0.2377	23
Fourteen	1023	D14Mit5	M214		-0.2122	0.3691	20		-0.49	0.0283	20	X	0.3234	0.1643	20
Fourteen	1024	D14Ncvs45	B436		-0.41	0.052	23		-0.1562	0.4768	23		0.1372	0.5324	23
Fourteen	1025	D14Ncvs35	D192		-0.4799	0.0205	23	X	-0.1989	0.3629	23		0.245	0.2598	23
Fourteen	1026	D14Ncvs36	B191		-0.4799	0.0205	23	X	-0.1989	0.3629	23		0.245	0.2598	23
Fourteen	1027	Ms6-5			-0.3143	0.1441	23		-0.2478	0.2543	23		0.3192	0.1376	23
Fourteen	1028	D14Ncvs37	B198		-0.3766	0.084	22		-0.1739	0.4389	22		0.3146	0.1539	22
Fourteen	1029	D14Ncvs38	B193		-0.3279	0.1468	21		-0.2068	0.3684	21		0.3188	0.159	21
Fourteen	1030	D14Ncvs39	B235		-0.3762	0.0769	23		-0.2162	0.3218	23		0.2988	0.1661	23
Fourteen	1031	Ang			-0.4351	0.0336	24	X	-0.2188	0.3043	24		0.2607	0.2186	24
Fourteen	1032	Glud			-0.32	0.1574	21		-0.1808	0.4328	21		0.142	0.5391	21
Fourteen	1033	Myhc-a			-0.4351	0.0336	24	X	-0.2188	0.3043	24		0.2607	0.2186	24
Fourteen	1034	Xmv-19			-0.4351	0.0336	24	X	-0.2188	0.3043	24		0.2607	0.2186	24
Fourteen	1035	D14Byu2			-0.4559	0.0378	21	X	-0.1428	0.5368	21		0.1438	0.534	21
Fourteen	1036	D14Byu3			-0.4351	0.0336	24	X	-0.2188	0.3043	24		0.2607	0.2186	24
Fourteen	1037	D14Byu4			-0.4351	0.0336	24	X	-0.2188	0.3043	24		0.2607	0.2186	24
Fourteen	1038	Rpl32-rs1			-0.4351	0.0336	24	X	-0.2188	0.3043	24		0.2607	0.2186	24
Fourteen	1039	Tcra			-0.4351	0.0336	24	X	-0.2188	0.3043	24		0.2607	0.2186	24
Fourteen	1040	Nrl			-0.4351	0.0336	24	X	-0.2188	0.3043	24		0.2607	0.2186	24
Fourteen	1041	Np			-0.4351	0.0336	24	X	-0.2188	0.3043	24		0.2607	0.2186	24
Fourteen	1042	Np2			-0.4351	0.0336	24	X	-0.2188	0.3043	24		0.2607	0.2186	24
Fourteen	1043	Rib1	Rib-1		-0.5454	0.0071	23	X	-0.1606	0.4642	23		0.2171	0.3196	23
Fourteen	1044	D14Ncvs40	D438		-0.2917	0.1666	24		-0.1481	0.4899	24		0.3347	0.1099	24
Fourteen	1045	Hpg			-0.2917	0.1666	24		-0.1481	0.4899	24		0.3347	0.1099	24
Fourteen	1046	D14Byu5			-0.1935	0.4006	21		-0.2185	0.3413	21		0.157	0.4967	21
Fourteen	1047	Rb1	Rb-1		-0.1852	0.4093	22		-0.1982	0.3765	22		0.1102	0.6253	22
Fourteen	1048	For5	For-5		-0.1856	0.4206	21		-0.2195	0.3391	21		0.1825	0.4285	21
Fourteen	1049	Es10	Es-10		-0.1965	0.3574	24		-0.1316	0.5398	24		0.132	0.5388	24
Fourteen	1050	Iapls3-15			-0.1267	0.5551	24		-0.1706	0.4254	24		0.1394	0.5158	24
Fourteen	1051	P198-14			-0.1079	0.6603	19		-0.5699	0.0108	19	X	0.3969	0.0925	19
Fourteen	1052	D14Mit7	L27												

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N		PCTWRED	P2ALPHA	P2N		AVGBASE1	P3ALPHA	P3N
Fourteen	1053	D14Ncvs48	B610		0.0178	0.9356	23		-0.1475	0.5019	23		-0.1381	0.5298	23
Fourteen	1054	D14Ncvs49	D609		0.0178	0.9356	23		-0.1475	0.5019	23		-0.1381	0.5298	23
Fourteen	1055	D14Mc1	D14McC1		-0.2538	0.2544	22		-0.266	0.2314	22		0.3096	0.1609	22
Fourteen	1056	D14Ncvs47	B473		-0.1018	0.6438	23		-0.1251	0.5694	23		0.212	0.3314	23
Fourteen	1057	D14Ncvs46	D474		-0.1088	0.6127	24		-0.0917	0.67	24		0.1937	0.3645	24
Fifteen	1058	D15Nds2	T18		0.2364	0.3157	20		0.2728	0.2446	20		-0.0175	0.9417	20
Fifteen	1059	P198			0.1526	0.4765	24		0.3542	0.0895	24		-0.1489	0.4873	24
Fifteen	1060	D1Nds3			0.1526	0.4765	24		0.3542	0.0895	24		-0.1489	0.4873	24
Fifteen	1061	D15Mit12	M34		0.327	0.1593	20		0.3371	0.1461	20		0.0228	0.9241	20
Fifteen	1062	D15Mit13	A36		0.2335	0.2722	24		0.3859	0.0625	24		-0.1149	0.5929	24
Fifteen	1063	D15Bir1			0.1867	0.3824	24		0.3506	0.093	24		-0.1368	0.5237	24
Fifteen	1064	D15Mit8	A79		0.2674	0.2544	20		0.3032	0.1938	20		-0.0039	0.987	20
Fifteen	1065	D15Mit7	M30		0.2674	0.2544	20		0.3032	0.1938	20		-0.0039	0.987	20
Fifteen	1066	D15Mit6	A59		0.176	0.4711	19		0.2293	0.345	19		0.0771	0.7538	19
Fifteen	1067	D15Mit5	L1		0.2674	0.2544	20		0.3032	0.1938	20		-0.0039	0.987	20
Fifteen	1068	D15Tu7			0.1383	0.5608	20		0.4298	0.0586	20		-0.1916	0.4183	20
Fifteen	1069	D15Tu8	D17Tu8		0.1447	0.5315	21		0.4124	0.0632	21		-0.1355	0.5582	21
Fifteen	1070	D15Ncvs27	B598		0.0721	0.7436	23		0.4017	0.0574	23		-0.1571	0.474	23
Fifteen	1071	D15Ncvs25	D595		0.0448	0.8392	23		0.4238	0.0439	23	X	-0.1665	0.4477	23
Fifteen	1072	D15Ncvs28	B582		0.0766	0.7219	24		0.4272	0.0373	24	X	-0.1475	0.4915	24
Fifteen	1073	Iapls2-1			-0.1515	0.4798	24		0.4905	0.015	24	X	-0.0175	0.9354	24
Fifteen	1074	D15Ncvs17	D214		0.0574	0.7946	23		0.4345	0.0383	23	X	-0.0944	0.6685	23
Fifteen	1075	D15Ncvs18	B79		0.0574	0.7946	23		0.4345	0.0383	23	X	-0.0944	0.6685	23
Fifteen	1076	D15Ncvs19	B359		-0.0449	0.8468	21		0.3467	0.1236	21		-0.0571	0.8059	21
Fifteen	1077	D15Mit3	L78		0.5322	0.0157	20	X	0.326	0.1606	20		-0.2799	0.2319	20
Fifteen	1078	D15Ncvs26	D531		0.5384	0.008	23	X	0.1085	0.6221	23		-0.3339	0.1195	23
Fifteen	1079	Pdgfb	S1s		0.1677	0.4443	23		0.2952	0.1714	23		-0.2294	0.2924	23
Fifteen	1080	Cyb2d9			0.0083	0.9724	20		0.3643	0.1144	20		-0.266	0.257	20
Fifteen	1081	D15Mit2	L10		0.2451	0.2976	20		0.2435	0.3008	20		-0.0958	0.6878	20
Fifteen	1082	Atf4	Atf-4		0.1046	0.6519	21		0.2843	0.2116	21		-0.2246	0.3277	21
Fifteen	1083	D15Mit1	L29		0.2049	0.3369	24		0.3446	0.0992	24		-0.3843	0.0637	24
Fifteen	1084	D15Ncvs20	D293		0.2351	0.2802	23		0.3426	0.1095	23		-0.3771	0.0761	23
Fifteen	1085	D15Mit15	Hoxc		0.359	0.12	20		0.5656	0.0093	20	X	-0.1774	0.4544	20
Fifteen	1086	Hoxc	Hox-3		0.3601	0.0839	24		0.3124	0.1373	24		-0.0903	0.6749	24
Fifteen	1087	mk=Nfe2			0.4835	0.0194	23	X	0.2309	0.2892	23		-0.1811	0.4083	23
Fifteen	1088	Spt2	55k, Spt-2		0.4157	0.0543	22		0.2041	0.3623	22		-0.2087	0.3514	22
Fifteen	1089	Pmv42	Pmv-42		0.3305	0.1147	24		0.6261	0.0011	24	X	-0.1873	0.3807	24
Fifteen	1090	Iapls3-20			0.2875	0.1731	24		0.3658	0.0787	24		-0.1323	0.5377	24
Fifteen	1091	D15Ncvs21	B335		0.3647	0.1139	20		0.4304	0.0582	20		-0.127	0.5937	20
Fifteen	1092	D15Ncvs29	B631		0.1571	0.485	22		0.491	0.0203	22	X	-0.226	0.3119	22
Fifteen	1093	D15Ncvs22	D185		0.2202	0.3248	22		0.4846	0.0223	22	X	-0.0918	0.6846	22
Fifteen	1094	D15Ncvs23	B107		0.1931	0.3774	23		0.5092	0.0131	23	X	-0.0625	0.777	23
Fifteen	1095	D15Ncvs24	D147		0.1931	0.3774	23		0.5092	0.0131	23	X	-0.0625	0.777	23
Sixteen	1096	D16Ncvs27	D504		0.0839	0.7178	21		0.0025	0.9914	21		-0.2423	0.2899	21
Sixteen	1097	Prm1	Prm-1		0.0387	0.8577	24		-0.0274	0.8988	24		-0.1531	0.4751	24
Sixteen	1098	Comt			-0.0221	0.9183	24		-0.0521	0.8091	24		-0.0128	0.9525	24
Sixteen	1099	Iapls3-6;1-12			-0.0221	0.9183	24		-0.0521	0.8091	24		-0.0128	0.9525	24
Sixteen	1100	D16Mit3	M127		-0.0596	0.7974	21		0.1695	0.4626	21		0.0641	0.7826	21
Sixteen	1101	Hmg1-rs7			0.0258	0.9092	22		0.2316	0.2998	22		0.0494	0.8272	22
Sixteen	1102	D16Mit4	M203		0.2656	0.2577	20		0.1791	0.45	20		0.1242	0.6019	20
Sixteen	1103	D16Bir1			0.1625	0.448	24		0.1857	0.3851	24		-0.0485	0.822	24
Sixteen	1104	Iapls1-6			0.0315	0.8838	24		0.2991	0.1557	24		-0.0446	0.836	24
Sixteen	1105	D16Ncvs24	D123		0.0055	0.98	23		0.3157	0.1422	23		-0.0629	0.7754	23
Sixteen	1106	D16Ncvs6	B136		-0.0027	0.9906	22		0.3603	0.0995	22		-0.0207	0.9271	22
Sixteen	1107	D16Nds2			0.2027	0.5067	13		0.279	0.3559	13		0.1882	0.538	13
Sixteen	1108	Mtv6	Mtv-6		0.0315	0.8838	24		0.2991	0.1557	24		-0.0446	0.836	24
Sixteen	1109	Mls3	Mls-3		-0.0179	0.9371	22		0.3111	0.1588	22		-0.0837	0.7111	22
Sixteen	1110	Xmmv13	Xmmv-13		-0.0217	0.9219	23		0.3131	0.1457	23		-0.0743	0.7362	23
Sixteen	1111	Xmmv21	Emv-21, Xmmv-21		-0.0217	0.9219	23		0.3131	0.1457	23		-0.0743	0.7362	23
Sixteen	1112	Ly7	Ly-7		-0.0377	0.8612	24		0.3397	0.1044	24		-0.0527	0.8067	24
Sixteen	1113	D16Ncvs25	B349		0.0786	0.7216	23		0.3791	0.0745	23		-0.0157	0.9434	23
Sixteen	1114	D16Ncvs26	D347		0.0786	0.7216	23		0.3791	0.0745	23		-0.0157	0.9434	23
Sixteen	1115	D16Ncvs28	B463		0.029	0.8954	23		0.2683	0.2158	23		-0.1336	0.5434	23
Sixteen	1116	Iapls2-15			-0.0019	0.9931	23		0.3554	0.0961	23		0.0139	0.9499	23
Sixteen	1117	Pmv35	Pmv-35		-0.0814	0.7053	24		0.0879	0.683	24		-0.0286	0.8946	24
Sixteen	1118	D16Nds1			-0.0355	0.8724	23		0.0308	0.8891	23		-0.0467	0.8324	23
Sixteen	1119	D16Ros2	Ar-6		-0.0355	0.8724	23		0.0308	0.8891	23		-0.0467	0.8324	23
Sixteen	1120	D16Byu1			-0.1015	0.6371	24		0.1558	0.4671	24		0.0563	0.7938	24
Sixteen	1121	D16Byu2			-0.1745	0.4494	21		0.3737	0.0951	21		-0.1337	0.5635	21
Sixteen	1122	D16Mit5	A38		0.0592	0.8098	19		0.3406	0.1535	19		0.0493	0.841	19
Sixteen	1123	D16Mc1	D16McC1		-0.0098	0.9637	24		0.3736	0.0721	24		0.0498	0.8172	24
Sixteen	1124	D16H21S16	D21S16h		-0.1932	0.3657	24		0.2531	0.2328	24		0.3099	0.1405	24
Sixteen	1125	Pmv14	Pmv-14		-0.1932	0.3657	24		0.2531	0.2328	24		0.3099	0.1405	24
Sixteen	1126	App			-0.1625	0.4481	24		0.1509	0.4814	24		0.015	0.9445	24
Sixteen	1127	D16Mit6	L7		0.1278	0.5913	20		0.1343	0.5724	20		-0.2764	0.2381	20
Sixteen	1128	Sod1	Sod-1		0.0193	0.9288	24		0.2374	0.2639	24		-0.2883	0.1719	24
Sixteen	1129	Pmv16	Pmv-16		-0.1127	0.6002	24		0.1318	0.5392	24		-0.2024	0.3429	24
Seventeen	1130	D17Leh119			-0.1095	0.6106	24		-0.2181	0.306	24		0.1016	0.6365	24
Seventeen	1131	D17Leh66e			-0.078	0.7302	22		-0.1796	0.4239	22		0.0954	0.6727	22
Seventeen	1132	D17Byu1			-0.0057	0.9791	24		-0.2244	0.2919	24		-0.1297	0.5458	24
Seventeen	1133	D17Rp17e	Rp17		0.0625	0.7716	24		-0.1807	0.3982	24		-0.1852	0.3862	24

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N	PCTWRED	P2ALPHA	P2N	AVGBASE1	P3ALPHA	P3N
Seventeen	1134	D17Tu50			0.0236	0.9193	21	-0.1573	0.496	21	-0.2408	0.2931	21
Seventeen	1135	Plg			0.0625	0.7716	24	-0.1807	0.3982	24	-0.1852	0.3862	24
Seventeen	1136	Tcp1	Tcp-1, Tp63, p6		-0.0288	0.8963	23	-0.1986	0.3636	23	-0.2179	0.318	23
Seventeen	1137	D17Leh66d			0.0625	0.7716	24	-0.1807	0.3982	24	-0.1852	0.3862	24
Seventeen	1138	D17Pas1			0.1371	0.5329	23	-0.1436	0.5132	23	-0.2199	0.3134	23
Seventeen	1139	D17Pri1			0.1124	0.6012	24	-0.1369	0.5236	24	-0.1668	0.4359	24
Seventeen	1140	D17Leh111			0.1124	0.6012	24	-0.1369	0.5236	24	-0.1668	0.4359	24
Seventeen	1141	D17Leh94			0.1124	0.6012	24	-0.1369	0.5236	24	-0.1668	0.4359	24
Seventeen	1142	D17Tu30			0.0703	0.7621	21	-0.1142	0.6219	21	-0.2211	0.3355	21
Seventeen	1143	D17Ncvs41	B443		0.092	0.6689	24	0.0457	0.8321	24	-0.1112	0.605	24
Seventeen	1144	D17Tu36			0.1824	0.4549	19	-0.105	0.6688	19	-0.0657	0.7893	19
Seventeen	1145	D17Ncvs22	D128		0.196	0.382	22	-0.0464	0.8375	22	-0.1608	0.4746	22
Seventeen	1146	D17Ncvs23	B129		0.1427	0.5159	23	-0.1497	0.4953	23	-0.1539	0.4831	23
Seventeen	1147	D17Ncvs24	D69		0.11	0.6259	22	-0.1786	0.4265	22	-0.1259	0.5766	22
Seventeen	1148	D17Ncvs25	B72		-0.0033	0.9894	19	0.0655	0.79	19	-0.2682	0.2669	19
Seventeen	1149	D17Ncvs40	B682		0.1124	0.6012	24	-0.1369	0.5236	24	-0.1668	0.4359	24
Seventeen	1150	D17Ncvs39	D681		0.0082	0.9736	19	-0.2286	0.3466	19	-0.415	0.0773	19
Seventeen	1151	D17Leh12			0.1532	0.4749	24	-0.1442	0.5014	24	-0.2511	0.2366	24
Seventeen	1152	Hmg1-rs6			0.1588	0.4692	23	-0.1608	0.4636	23	-0.2129	0.3294	23
Seventeen	1153	Hba-ps4			0.1532	0.4749	24	-0.1442	0.5014	24	-0.2511	0.2366	24
Seventeen	1154	D17Lon2			0.1902	0.4089	21	-0.029	0.9008	21	-0.2866	0.2078	21
Seventeen	1155	D17Lon3	sazD		0.1902	0.4089	21	-0.029	0.9008	21	-0.2866	0.2078	21
Seventeen	1156	D17Ncvs26	D31		-0.073	0.7468	22	-0.2219	0.321	22	-0.2006	0.3707	22
Seventeen	1157	D17Ncvs27	B32		-0.073	0.7468	22	-0.2219	0.321	22	-0.2006	0.3707	22
Seventeen	1158	Glo1	Glo-1		-0.003	0.989	24	-0.2327	0.274	24	-0.2094	0.3261	24
Seventeen	1159	D17Leh26			-0.003	0.989	24	-0.2327	0.274	24	-0.2094	0.3261	24
Seventeen	1160	D17H21S56	D21S56h		-0.003	0.989	24	-0.2327	0.274	24	-0.2094	0.3261	24
Seventeen	1161	Pim1	D17Mit23, Pim-1		-0.003	0.989	24	-0.2327	0.274	24	-0.2094	0.3261	24
Seventeen	1162	Cryal	Crya-1, Acry-1		-0.2441	0.2617	23	-0.2828	0.1911	23	-0.0973	0.6588	23
Seventeen	1163	D17Ncvs28	B4		-0.0703	0.762	21	-0.0172	0.941	21	-0.2022	0.3795	21
Seventeen	1164	D17Ncvs29	D3		-0.0204	0.9281	22	0.1204	0.5935	22	-0.2857	0.1975	22
Seventeen	1165	D17Ncvs30	D135		-0.1139	0.6231	21	-0.0128	0.9561	21	-0.3526	0.1169	21
Seventeen	1166	D17Ncvs31	B315		-0.0374	0.8653	23	0.0347	0.8753	23	-0.2066	0.3442	23
Seventeen	1167	D17Ncvs32	D320		-0.0374	0.8653	23	0.0347	0.8753	23	-0.2066	0.3442	23
Seventeen	1168	Coll1a2	Coll1a-2		-0.0552	0.7978	24	0.0409	0.8495	24	-0.2156	0.3117	24
Seventeen	1169	H2Tlev1	H-2Tlev1		-0.0552	0.7978	24	0.0409	0.8495	24	-0.2156	0.3117	24
Seventeen	1170	H2S	H-2S		-0.0552	0.7978	24	0.0409	0.8495	24	-0.2156	0.3117	24
Seventeen	1171	Grr	grc		0.0514	0.825	21	0.0961	0.6787	21	-0.2119	0.3564	21
Seventeen	1172	D17Leh89			-0.0689	0.7547	23	0.0729	0.7408	23	-0.2036	0.3513	23
Seventeen	1173	D17Leh525			-0.0689	0.7547	23	0.0729	0.7408	23	-0.2036	0.3513	23
Seventeen	1174	Hsp70	Hsp68		-0.0552	0.7978	24	0.0409	0.8495	24	-0.2156	0.3117	24
Seventeen	1175	D17Nds2	T9		-0.1074	0.6523	20	0.244	0.2998	20	-0.2893	0.216	20
Seventeen	1176	D17Mit24	Thy-19. D12		-0.1074	0.6523	20	0.244	0.2998	20	-0.2893	0.216	20
Seventeen	1177	D17Mit22	MHCeb2. D16		-0.1074	0.6523	20	0.244	0.2998	20	-0.2893	0.216	20
Seventeen	1178	D17Mit21	MHCab2. D21		-0.1074	0.6523	20	0.244	0.2998	20	-0.2893	0.216	20
Seventeen	1179	D17Mit16	A25		-0.1074	0.6523	20	0.244	0.2998	20	-0.2893	0.216	20
Seventeen	1180	D17Mit13	L57		-0.1074	0.6523	20	0.244	0.2998	20	-0.2893	0.216	20
Seventeen	1181	D17Tu49			0.0514	0.825	21	0.0961	0.6787	21	-0.2119	0.3564	21
Seventeen	1182	D17Ncvs33	B6		-0.2934	0.2373	18	0.2049	0.4148	18	-0.2448	0.3275	18
Seventeen	1183	D17Ncvs38	D441		-0.0281	0.8986	23	0.0506	0.8188	23	-0.2055	0.3469	23
Seventeen	1184	D17Ncvs37	D689		-0.0552	0.7978	24	0.0409	0.8495	24	-0.2156	0.3117	24
Seventeen	1185	Gln3-4			0.0884	0.6811	24	-0.0123	0.9545	24	-0.2188	0.3043	24
Seventeen	1186	Gln3-7			0.0884	0.6811	24	-0.0123	0.9545	24	-0.2188	0.3043	24
Seventeen	1187	D17Leh173			0.0884	0.6811	24	-0.0123	0.9545	24	-0.2188	0.3043	24
Seventeen	1188	Msl5-3			0.1423	0.5172	23	-0.0406	0.8542	23	-0.2217	0.3093	23
Seventeen	1189	D17Mit11	M145		0.0547	0.8188	20	0.1881	0.4271	20	-0.2997	0.1992	20
Seventeen	1190	D17Tu16			0.1636	0.4785	21	0.0043	0.9852	21	-0.1503	0.5156	21
Seventeen	1191	Tpx1	Tpx-1		0.1636	0.4785	21	0.0043	0.9852	21	-0.1503	0.5156	21
Seventeen	1192	D17Leh116			0.0742	0.7303	24	0.1018	0.6358	24	-0.2166	0.3093	24
Seventeen	1193	D17Leh154			0.0742	0.7303	24	0.1018	0.6358	24	-0.2166	0.3093	24
Seventeen	1194	D17Leh23			0.0742	0.7303	24	0.1018	0.6358	24	-0.2166	0.3093	24
Seventeen	1195	Iapls1-3			0.0742	0.7303	24	0.1018	0.6358	24	-0.2166	0.3093	24
Seventeen	1196	Tcte1	Tcte-1, D17Sil		0.0742	0.7303	24	0.1018	0.6358	24	-0.2166	0.3093	24
Seventeen	1197	D17Ncvs7	B89		-0.0154	0.9445	23	0.1033	0.6389	23	0.0329	0.8815	23
Seventeen	1198	D17Ncvs34	D90		-0.0154	0.9445	23	0.1033	0.6389	23	0.0329	0.8815	23
Seventeen	1199	Upg1	Upg-1		-0.0421	0.8451	24	0.0881	0.6823	24	0.0271	0.8999	24
Seventeen	1200	D17Byu2			-0.0336	0.8763	24	0.1088	0.6129	24	0.0206	0.9238	24
Seventeen	1201	D17Mit10	L36		-0.0824	0.7298	20	0.3407	0.1416	20	-0.0095	0.9682	20
Seventeen	1202	D17Mit6	M254		-0.0824	0.7298	20	0.3407	0.1416	20	-0.0095	0.9682	20
Seventeen	1203	D17Mit7	L4; A23		-0.0591	0.8044	20	0.2159	0.3605	20	-0.0219	0.927	20
Seventeen	1204	Ckb-rs2	Ck-2		-0.0134	0.9505	24	0.0212	0.9218	24	0.0102	0.9623	24
Seventeen	1205	Trp53-ps			-0.0134	0.9505	24	0.0212	0.9218	24	0.0102	0.9623	24
Seventeen	1206	Hprt-ps1	Rp-10, Hprt-2		0.1847	0.3876	24	0.1666	0.4364	24	-0.1267	0.5551	24
Seventeen	1207	D17Mit3	L28		0.1074	0.6618	19	0.2657	0.2716	19	-0.3143	0.1901	19
Seventeen	1208	D17Nds41	DONds41		-0.1149	0.5928	24	0.3259	0.1201	24	-0.4231	0.0394	24 X
Seventeen	1209	D17Ncvs35	D291		-0.2829	0.2139	21	0.3683	0.1004	21	-0.0557	0.8105	21
Seventeen	1210	D17Ncvs36	B35		-0.187	0.393	23	0.3811	0.0728	23	-0.0473	0.8303	23
Eighteen	1211	D18Mit15	L87		-0.524	0.0177	20 X	-0.0606	0.7997	20	0.0369	0.8773	20
Eighteen	1212	D18Mit14	L13		-0.2892	0.2161	20	-0.1393	0.5582	20	0.0422	0.8598	20
Eighteen	1213	D18Mit17	Gr1-1. D118		-0.4127	0.0706	20	-0.1698	0.4743	20	0.1789	0.4503	20
Eighteen	1214	Iapls3-7			-0.4931	0.0143	24 X	0.058	0.7878	24	-0.084	0.6963	24

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N	PCTWRED	P2ALPHA	P2N	AVGBASE1	P3ALPHA	P3N
Eighteen	1215	D18Ncvs22	B548		-0.4963	0.0188	22 X	-0.1364	0.5449	22	0.121	0.5918	22
Eighteen	1216	D18Ncvs3	B346		-0.5822	0.0036	23 X	-0.0424	0.8478	23	0.1108	0.6147	23
Eighteen	1217	D18Ncvs17	D345		-0.5822	0.0036	23 X	-0.0424	0.8478	23	0.1108	0.6147	23
Eighteen	1218	D18Mit10	A100		-0.506	0.0228	20 X	-0.4135	0.0699	20	0.2914	0.2125	20
Eighteen	1219	D18Ncvs5		39	-0.6043	0.0102	17 X	-0.1652	0.5264	17	0.0798	0.7608	17
Eighteen	1220	D18Byu22	DOByu22		-0.4771	0.0248	22 X	-0.0076	0.9731	22	0.0588	0.7949	22
Eighteen	1221	D18Mit9	M209		-0.0624	0.7937	20	-0.0707	0.7672	20	0.0966	0.6855	20
Eighteen	1222	Iapls3-12			-0.0061	0.9773	24	-0.2164	0.3097	24	0.2138	0.3158	24
Eighteen	1223	D18Ncvs18	B173		-0.2314	0.2881	23	-0.0365	0.8688	23	0.0225	0.9189	23
Eighteen	1224	D18Ncvs19	D174		-0.2314	0.2881	23	-0.0365	0.8688	23	0.0225	0.9189	23
Eighteen	1225	D18Byu1			-0.1952	0.3608	24	-0.0457	0.8321	24	0.04	0.8527	24
Eighteen	1226	Iapls1-5			-0.2081	0.3292	24	0.0309	0.8861	24	0.0299	0.8899	24
Eighteen	1227	D18Mit8	L24		-0.1215	0.6097	20	-0.0655	0.7837	20	0.1763	0.4571	20
Eighteen	1228	Xmv29	Xmv-29		-0.2032	0.3409	24	0.0033	0.9877	24	0.1327	0.5366	24
Eighteen	1229	D18Ncvs23	D621		-0.2806	0.2307	20	0.0794	0.7395	20	0.0655	0.7839	20
Eighteen	1230	D18Ncvs24	B620		-0.1719	0.4562	21	0.0595	0.7977	21	-0.0497	0.8305	21
Eighteen	1231	D18Ncvs20	D184		-0.198	0.3652	23	0.048	0.8277	23	-0.1234	0.575	23
Eighteen	1232	D18Ncvs21	B288		-0.198	0.3652	23	0.048	0.8277	23	-0.1234	0.575	23
Eighteen	1233	D18Mit7	M108		-0.0947	0.6911	20	0.0344	0.8854	20	-0.1308	0.5825	20
Eighteen	1234	D18Mit4	M51		-0.3932	0.0863	20	-0.159	0.5032	20	-0.0973	0.6833	20
Nineteen	1235	Mr66-3			0.3504	0.1012	23	0.2932	0.1745	23	0.0256	0.9078	23
Nineteen	1236	D19Ncvs16	B281		0.3936	0.0632	23	0.3793	0.0742	23	-0.021	0.9242	23
Nineteen	1237	D19Ncvs17	D282		0.3936	0.0632	23	0.3793	0.0742	23	-0.021	0.9242	23
Nineteen	1238	D19Ncvs30	B431		0.2793	0.1967	23	0.4235	0.044	23 X	-0.085	0.6999	23
Nineteen	1239	D19Ncvs26	D430		0.4613	0.0353	21 X	0.3981	0.0739	21	0.0103	0.9648	21
Nineteen	1240	D19Ncvs18	B158		0.508	0.0374	17 X	0.0452	0.8631	17	-0.1068	0.6832	17
Nineteen	1241	D19Ncvs19	D157		0.508	0.0374	17 X	0.0452	0.8631	17	-0.1068	0.6832	17
Nineteen	1242	Emk			0.593	0.0023	24 X	-0.0208	0.9231	24	0.0941	0.6618	24
Nineteen	1243	D19Rp19e			0.593	0.0023	24 X	-0.0208	0.9231	24	0.0941	0.6618	24
Nineteen	1244	Ly10	Ly-10, Ly-m10		0.5663	0.0049	23 X	-0.2261	0.2995	23	0.1027	0.641	23
Nineteen	1245	D19Byu1			0.593	0.0023	24 X	-0.0208	0.9231	24	0.0941	0.6618	24
Nineteen	1246	D19Byu2			0.5716	0.0035	24 X	0.0082	0.9697	24	-0.0141	0.948	24
Nineteen	1247	Cd5	Ly-1		0.4212	0.0404	24 X	0.2672	0.2068	24	-0.1733	0.4179	24
Nineteen	1248	D19Mit22			0.4212	0.0404	24 X	0.2672	0.2068	24	-0.1733	0.4179	24
Nineteen	1249	D19Ncvs1	B292		0.1568	0.4972	21	0.1854	0.421	21	-0.2423	0.29	21
Nineteen	1250	Osbp			0.2216	0.2981	24	0.2306	0.2783	24	-0.2259	0.2885	24
Nineteen	1251	Cntf			0.2216	0.2981	24	0.2306	0.2783	24	-0.2259	0.2885	24
Nineteen	1252	Pomc2	Pomc-2		0.1205	0.5748	24	0.3284	0.1171	24	-0.0982	0.648	24
Nineteen	1253	D19Ncvs27	D519		-0.0901	0.6826	23	0.2536	0.243	23	-0.0217	0.9215	23
Nineteen	1254	Gsl5	GL-Y, Gsl-5		-0.085	0.6931	24	0.277	0.1901	24	-0.0162	0.9401	24
Nineteen	1255	D19Byu3			-0.085	0.6931	24	0.277	0.1901	24	-0.0162	0.9401	24
Nineteen	1256	Ea4	Ea-4		-0.1638	0.4664	22	0.2657	0.232	22	-0.0431	0.8489	22
Nineteen	1257	D19Nyu1			0.0839	0.6968	24	0.0083	0.9694	24	0.1433	0.5041	24
Nineteen	1258	D19Mit16			0.0839	0.6968	24	0.0083	0.9694	24	0.1433	0.5041	24
Nineteen	1259	D19Ncvs20	B358		0.2282	0.307	22	0.1002	0.6572	22	0.0571	0.8008	22
Nineteen	1260	D19Ncvs31	B628		0.2472	0.2554	23	-0.0522	0.813	23	0.101	0.6466	23
Nineteen	1261	Lpc1	Lipo-I, Lpc-1		0.0911	0.672	24	0.0259	0.9045	24	0.1325	0.5372	24
Nineteen	1262	D19J1			0.0723	0.7429	23	-0.1088	0.6212	23	0.2168	0.3204	23
Nineteen	1263	Iapls3-8			0.2168	0.309	24	-0.1733	0.4182	24	0.1694	0.4288	24
Nineteen	1264	Rbb4	Rbp-4		0.4308	0.0356	24 X	-0.5041	0.012	24 X	0.2425	0.2535	24
Nineteen	1265	D19Ncvs33	B455		0.0179	0.9354	23	-0.477	0.0214	23 X	-0.1434	0.5139	23
Nineteen	1266	D19Ncvs32	D685		-0.1551	0.4798	23	-0.4272	0.042	23 X	0.1041	0.6366	23
Nineteen	1267	Cyp2c			0.1687	0.4308	24	-0.461	0.0234	24 X	0.1001	0.6417	24
Nineteen	1268	D19Mit19			0.1687	0.4308	24	-0.461	0.0234	24 X	0.1001	0.6417	24
Nineteen	1269	D19Byu4			0.1687	0.4308	24	-0.461	0.0234	24 X	0.1001	0.6417	24
Nineteen	1270	D19Ncvs25	B91		-0.0757	0.7315	23	-0.0518	0.8146	23	0.0764	0.7291	23
Nineteen	1271	D19Ncvs24	D278		0.2293	0.2925	23	-0.1574	0.4732	23	0.1726	0.4309	23
Nineteen	1272	D19Ncvs29	D472		0.2318	0.2993	22	-0.42	0.0516	22	-0.0171	0.9399	22
Nineteen	1273	D19Ncvs28	D402		0.2283	0.2832	24	-0.4894	0.0152	24 X	0.0642	0.7658	24
Nineteen	1274	D19Mit1	A17		0.1306	0.543	24	-0.4706	0.0203	24 X	0.1955	0.3599	24
Nineteen	1275	D19Ncvs21	D51		0.226	0.3119	22	-0.0937	0.6782	22	0.0398	0.8605	22
Nineteen	1276	D19Ncvs22	B262		0.0279	0.902	22	-0.2152	0.3362	22	-0.0193	0.9321	22
Nineteen	1277	D19Ncvs23	D261		0.1382	0.5295	23	-0.3411	0.1111	23	0.0918	0.6771	23
Nineteen	1278	P40-rs7	P40-7		-0.0416	0.8471	24	-0.3601	0.0839	24	0.1709	0.4246	24
Nineteen	1279	D19Mit6			0.0478	0.8246	24	-0.1381	0.52	24	0.1637	0.4447	24
Nineteen	1280	D19Mc1	D19McC1		0.1605	0.4538	24	-0.1329	0.5359	24	0.0562	0.7942	24
Nineteen	1281	Xmv18	Xmv-18		0.1605	0.4538	24	-0.1329	0.5359	24	0.0562	0.7942	24
X-tent	1282	DXMit1	L43		-0.0134	0.9552	20	-0.2305	0.3282	20	-0.1366	0.5657	20
X-tent	1283	Rsvp			-0.0019	0.9934	21	-0.1198	0.6049	21	-0.1254	0.588	21
X-tent	1284	Cf8	Cf-8		-0.0019	0.9934	21	-0.1198	0.6049	21	-0.1254	0.588	21
X-tent	1285	Oat-rs1			-0.0971	0.6518	24	-0.0792	0.7128	24	-0.0564	0.7934	24
X-tent	1286	Iapls3-11			-0.0255	0.9057	24	-0.1274	0.553	24	-0.0094	0.9651	24
X-tent	1287	DXNcvs10	B555		-0.0639	0.7667	24	-0.2153	0.3124	24	-0.2603	0.2193	24
X-tent	1288	DXNds3			-0.0132	0.9522	23	-0.2037	0.3512	23	-0.1439	0.5125	23
X-tent	1289	Iapls2-13			-0.0951	0.6586	24	-0.2567	0.2259	24	-0.1553	0.4688	24
X-tent	1290	DXWas17			-0.0585	0.7858	24	-0.121	0.5733	24	-0.2395	0.2597	24
X-tent	1291	DXNcvs9	D270		0.1358	0.5467	22	0.2104	0.3472	22	-0.1292	0.5667	22
X-tent	1292	DXNcvs4	B285		0.2752	0.2037	23	0.2537	0.2428	23	-0.3053	0.1567	23
X-tent	1293	TelXq			0.1703	0.4264	24	0.2664	0.2083	24	-0.2759	0.1919	24
X-firm	1294	DXWas17			-0.0585	0.7858	24	-0.121	0.5733	24	-0.2395	0.2597	24
X-firm	1295	DXNds3			-0.0132	0.9522	23	-0.2037	0.3512	23	-0.1439	0.5125	23

SAG QTL w/ 1994 marker set

analysis run 12-16-94 by SRM

CHROMOSOME	NO.	LOCUS	ALIAS	CM	PCTRED	P1ALPHA	P1N	PCTWRED	P2ALPHA	P2N	AVGBASE1	P3ALPHA	P3N
X-firm	1297	Rsvp			-0.0019	0.9934	21	-0.1198	0.6049	21	-0.1254	0.588	21
X-firm	1298	Cf-8			-0.0019	0.9934	21	-0.1198	0.6049	21	-0.1254	0.588	21
X-firm	1299	DXMit1	L43		-0.0134	0.9552	20	-0.2305	0.3282	20	-0.1366	0.5657	20