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Review of Quantitative Trait Loci Identified in the Chicken

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Abstract

Methods for mapping QTL are actively used in the chicken to identify chromosomal regions contributing to variation in traits related to growth, disease resistance, egg production, behavior, and metabolic parameters. However, higher-resolution mapping and better knowledge of the genetic architecture underlying QTL are needed for successful application of this information into breeding programs. Therefore, this paper summarizes and integrates original, primary QTL studies in the chicken to identify basic information on the genetic architecture of quantitative traits in chickens. The results of this review show several instances of consensus of QTL locations for similar traits from independent studies. Furthermore, the consensus of QTL location for different traits and evidence for QTL with parent-of-origin effect, transgressive alleles, epistatic QTL, and QTL × sex interaction in chicken are presented and discussed. This information can be helpful in identifying genes or mutations underlying the QTL and in the application of genomic information in marker-assisted breeding programs.

Keywords

chicken, quantitative trait loci, genetic architecture, high-resolution mapping, marker-assisted breeding

Disciplines

Agriculture | Animal Sciences | Genetics and Genomics | Poultry or Avian Science

Comments

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Review of Quantitative Trait Loci Identified in the Chicken

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ABSTRACT Methods for mapping QTL are actively used in the chicken to identify chromosomal regions contributing to variation in traits related to growth, disease resistance, egg production, behavior, and metabolic parameters. However, higher-resolution mapping and better knowledge of the genetic architecture underlying QTL are needed for successful application of this information into breeding programs. Therefore, this paper summarizes and integrates original, primary QTL studies in the chicken to identify basic information on the genetic architecture of quantitative traits in chickens. The results of this review show several instances of consensus of QTL locations for similar traits from independent studies. Furthermore, the consensus of QTL location for different traits and evidence for QTL with parent-of-origin effect, transgressive alleles, epistatic QTL, and QTL × sex interaction in chicken are presented and discussed. This information can be helpful in identifying genes or mutations underlying the QTL and in the application of genomic information in marker-assisted breeding programs.

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INTRODUCTION

In the past 10 yr, QTL mapping studies in the chicken have identified chromosomal regions that contribute to variation in economically important traits. The ultimate goal of these studies is generally to identify genetic markers that are close to the QTL [linkage disequilibrium (**LD**) markers] or the gene underlying the QTL (direct marker) and to use this information in marker-assisted breeding programs (Dekkers, 2004). This goal is difficult to achieve because of polygenic inheritance, epistasis, incomplete penetrance, variable expressivity, and pleiotropy of QTL (Lander and Schork, 1994; Glazier et al., 2002) but can be furthered by compiling results across studies. The objective of this review, therefore, was to identify consensus information on the genetic architecture of complex quantitative traits in chickens by summarizing and integrating results from primary QTL studies.

A similar review conducted by Hocking (2005) summarized chicken QTL results published through the end of 2004. There has been rapid progress in QTL studies, with 17 new papers reporting 370 QTL in chickens since the Hocking (2005) review. Hocking (2005) proposed use of much more stringent significance thresholds for inclusion of QTL than that used in the current paper, which is appropriate when evaluating single studies. Because the purpose of the present review was to discern biological patterns from independent studies, less stringent thresholds were used for reporting. Furthermore, the reports analyzed for the preparation of this review were used to establish the Chicken QTLdb (http://www.animalgenome.org/QTLdb/chicken.html), which allows for easy search and comparison of QTL results from different studies and complements other major public QTL databases for the chicken: ChickCmap (http://www.animalsciences.nl/Cmap) and ChickVD (http://chicken.genomics.org.cn; Wang et al., 2005).

Results of the present review will be useful for directing future genetic and genomic studies. It is particularly timely in that we are now at a transition point in analysis methods for quantitative traits in the chicken because of the recent availability of genomic sequence information (Soller et al., 2006).

MATERIALS AND METHODS

Results from the chicken QTL mapping studies published in refereed journals were summarized. Papers were identified through PubMed (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=PubMed) at the end of May 2006, using the key words "QTL," "quantitative trait loci," "chicken," and "poultry." Four additional papers reporting chicken QTL results that were cited in the papers identified via the PubMed search and 2 papers accepted for publication were also included. Of the 50 reviewed papers, 21 focused on growth and body composition, 13 on disease resistance, 8 on egg production, 5 on behavior, and 3 on metabolic parameters. Studies reporting QTL for egg production and metabolic parameters also reported QTL for BW and composition. In some instances,

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Table 1. Hierarchical classification of phenotypic traits

Trait category	Trait abbreviation[1]	Trait
Growth	GR	Growth-related traits
	AF	Abdominal fat weight adjusted to BW or CW or as a percentage of BW
	BCo	Color of the breast
	BM	Breast muscle weight adjusted to BW or CW or as a percentage of BW
	Bursa	Bursa weight adjusted to BW
	BW	BW
	CS	Conformation score
	CW	Carcass weight adjusted to BW or as a percentage of BW
	DS	Drumstick weight adjusted to CW or as a percentage of BW
	DSM	Drumstick muscle weight adjusted to CW
	Feath	Feathering
	FI	Feed intake
	FNTH	Weight of the front half of the carcass as percentage of BW
	G/F	Feed efficiency or gain/feed consumed
	Gizz	Gizzard weight adjusted to CW
	Heart	Heart weight adjusted to CW or as percentage of BW
	IF	Percentage of i.m. fat
	Intes	Intestine length adjusted to CW
	Liver	Liver weight adjusted to CW or as percentage of BW
	Lung	Lung weight adjusted to BW
	MCo	Meat color
	SF	Skin fat weight adjusted to BW or CW
	Shank	Shank weight adjusted to BW
	Spleen	Spleen weight adjusted to BW or CW or as percentage of BW
	Thigh	Thigh weight adjusted to CW
	TM	Thigh muscle weight adjusted to CW
	TPL	Transport loss
	Wing	Wing weight adjusted to CW
	WM	White meat weight percentage of BW
Disease resistance	DR	Disease resistance related traits
	ABR-x	Antibody response to x
	CECUM-y	Cecal bacterial burden after challenging with y
	CLOAC-y	Cloacal swabs after challenging with y
	MD	Marek's disease-related traits
	MORT-TOT	Total mortality
	SPLEEN-y	Spleen bacterial burden after challenging with y
	Tm ABR-x	Time to achieve maximum ABR-x
	OS	Oocyst shedding
Egg	Egg	Egg production and egg quality-related traits
	AFE	Age at first egg
	ASM	Age at sexual maturity
	AH	Albumin height
	AW	Albumen weight
	BMC	Bone mineral content
	BMD	Bone mineral density
	BWfin	BW at the end of the test period
	EN	Number of eggs
	ENT	The sum of the egg laid by a bird
	EPR	Egg production ratio, the number of eggs/number of producing days
	ES shape	Eggshell shape or the ratio of the width of the egg at its largest point to the egg's length at its longest point
	ESC	Eggshell color
	ESP	Eggshell percentage
	ESS	Eggshell strength
	EST	Eggshell thickness
	ESW	Eggshell weight
	EW	Egg weight
	HA	Humerus area
	HBMC	Humerus BMC adjusted to BW and cumulative egg production
	HBMD	Humerus BMD adjusted to BW and cumulative egg production
	HL	Humerus length
	HU	Haugh units
	HW	Humerus width
	LLE	Long length of egg
	Lss	Lightness; luminance or lightness component
	Rss	Redness; chromatic component from green to red
	SGRAV	Egg-specific gravity
	SLE	Short length of egg
	TA	Tibia area
	TBCC	Total blood cell count adjusted to CW
	TBF	Tibia breaking force adjusted to BW
	TBMC	Tibia BMC adjusted to BW and cumulative egg production
	TBMD	Tibia BMD adjusted to BW and cumulative egg production

Continued

Table 1 (Continued). Hierarchical classification of phenotypic traits

Trait category	Trait abbreviation[1]	Trait
	TL	Tibia length
	TME	Tibia modulus of elasticity
	TSN	Tibia strain
	TSS	Tibia stress
	TW	Tibia width
	Yss	Yellowness; chromatic component from blue to yellow
	YW	Yolk weight
Behavior	Behav	Behavior-related traits
	CFL	Contrafreeloading
	Fear	Fear-related traits
	FP	Feather pecking
	RFP	Receiving feather pecking
	Social	Social tendency
Metabolic	Meta	Metabolic parameter-related traits
	BWas	BW under ascites conditions
	CH	Plasma concentration of cholesterol
	CORT	Corticosterone response after a manual restraint test
	Creat	Creatinine kinase concentration adjusted to BW
	FHS	Fluid in the heart sac
	GL	Plasma concentration of glucose
	HCT	Hematocrit value
	IGF-I	Plasma concentration of insulin-like growth factor
	INS	Plasma concentration of insulin
	Liver A.	Liver abnormalities
	MCV	Mean blood cell volume adjusted to CW
	PCV	Packed cell volume adjusted to CW
	RV	Right ventricular weight as percentage of BW
	RV:TV	Ratio of right ventricular weight as a percentage to total ventricular weight
	TBCC	Total blood cell count adjusted to CW
	TG	Plasma concentration of triglycerides
	Trop	Troponin T concentration
	TV	Total ventricular weight as percentage of BW

[1]x = noninfectious antigens: *Brucella abortus* (BA), *Escherichia coli* vaccine (*E. coli* v), keyhole limpet hemocyanin (KLH), lipopolysaccharide (LPS), lipoteichoic acid (LTA), *Mycobacterium butyricum* (MB), Newcastle disease virus vaccine (NDV v), sheep red blood cells (SRBC), *Salmonella enteritidis* vaccine (SEv); and y = live pathogens: *S. enteritidis* (SE); *Salmonella typhimurium* (ST).

multiple papers reported different aspects of the analysis of the same population.

The evaluated phenotypic traits were classified into 5 major trait categories (Table 1): "growth" for traits related to BW, body composition, and feed intake; "egg" for traits related to egg production, egg quality and skeleton; "disease resistance" (**DR**) for traits related to DR, "metabolic" for traits related to metabolic parameters, and "behavior" for traits related to behavior. This working version of trait ontology to discuss these general trait categories may differ slightly from that found in some databases, because there is no standard trait ontology for poultry. For studies that evaluated QTL for carcass, organ, or tissue weights, only those QTL identified using adjustments for BW or carcass weight were summarized, as those are likely the most biologically relevant. From studies that reported QTL for both BW and weight gain, only QTL for BW were included.

In addition to QTL that were significant at a 5% genome-wise and experiment-wise level, QTL with suggestive linkage evidence at the 20% genome-wise level, the 5% chromosome-wise level, and the 1% single-point level were also included. The inclusion of suggestive QTL was done to help discern supportive evidence of QTL location among independent studies.

The QTL locations reported in the original publications were converted to consensus map (**Cmap**) locations based on marker positions in the current version of the chicken consensus linkage map (Schmid et al., 2005; http://www.animalsciences.nl/Cmap). For single-point analyses, the Cmap positions of markers with significant associations were presented as the Cmap QTL location. For QTL detected by multipoint QTL analyses, the QTL were positioned on the Cmap in the same marker intervals and at equal distances from the closest marker, as in the original publication. If the information provided by the original publication was not sufficient to calculate QTL distance from at least 1 of the flanking markers, the average Cmap location of the QTL flanking markers was given as the Cmap QTL location. Less than 1% of the data was rejected because of unresolved discrepancies regarding the locations of flanking markers between the original paper and the consensus map.

RESULTS AND DISCUSSION

Population Designs Used in Chicken QTL Studies

Population designs used in chicken QTL studies are summarized in Table 2 and are designated as F_2, backcross

Table 2. Population structures and lines used in chicken QTL studies

Cross[1]	Line description[2]	Population structure[3]	Reference
BD × BD	Divergently selected (S_5) on ABR to *Escherichia coli*	F_2 and BC	Yonash et al. (2001)
BD × BD	Divergently selected (S_{11}) on ABR to *E. coli*	F_2 and BC	Yunis et al. (2002)
BD × BD	Genetically different outcrossed BD lines originating from WR breed	F_2-F_3	van Kaam et al. (1998, 1999a,b) Rabie et al. (2005) Hamoen et al. (2001) Jennen et al. (2004)
		AIL-F_9	Jennen et al. (2005)
BS × BD	Selected (S_{20}) for production traits, substantial differences in resistance to coccidiosis and MD	F_2	Zhu et al. (2003)
BS × BD	Commercial broiler lines that differed in WM percentage	F_2	McElroy et al. (2006)
BS × F &WL	Outbred broiler sires × highly inbred F (M15.2) and highly inbred WL (G-B2)	F_2	Zhou et al. (2006a,b)
BS × F & WL	Outbred broiler sires × highly inbred F (15.2) and WL MHC congenic G-B1 and G-B2 lines	F_1	Kaiser and Lamont (2002)
BS × F & WL	Outbred broiler sires × highly inbred F (M15.2) and highly inbred WL (Ghs6)	F_1	Deeb and Lamont (2003)
BS × F, WL & SP	Outbred broiler sires × highly inbred F (M15.2), WL MHC congenic (G-B1 and G-B2), and SP (21.1) lines	F_1	Kaiser et al. (2002)
BSD × WL	Commercial strain of broilers × WL primary breeding stock line	F_2	Schreiweis et al. (2005, 2006)
BS × WL	BS selected for high growth rate and breast muscle yields × WL derived from commercial pure line	F_2	Sewalem et al. (2002) Ikeobi et al. (2002, 2004) Navarro et al. (2005)
BS × WL	Broiler × layer	F_2	Nones et al. (2006)
CR × WL	Cornish meat-type strain 21 selected for high growth rate and low abdominal fat weight from control strain 20 × WL	F_2	Hansen et al. (2005)
FL × LL	Divergently selected (S_7) on abdominal fat weight at 9 wk	F_2	Lagarrigue et al. (2006)
		F_2 and AB (BC_2)	Abasht et al. (2006)
IW × IW	Divergently selected for high and low ABR to SRBC	F_2	Siwek et al.[4] (2003a,b, 2004, 2006)
RIR × WL	RIR segregating to the slow feathering gene on *GgaZ* × WL selected on feed efficiency, egg mass production, and egg quality	F_2	Tuiskula-Haavisto et al. (2002, 2004)
RJF × WL	RJF male from a relatively closed European zoo population, originally obtained from Thailand × WL (SLU13)	F_2	Kerje et al. (2003) Schutz et al. (2002, 2004)
S × WL	S, a Japanese native line with unimproved BW gain × WL	F_2	Tatsuda et al. (2000)
S × WR	S, a Japanese native line with unimproved BW gain × WR breed with excellent BW gain	F_2	Tatsuda and Fujinaka (2001a,b)
RIR × GIP	RIR × GIP, a native Polish breed	F_2	Wardecka et al. (2002)
RIR × WL	RIR × WL selected (S_{14}) for reduction in nondestructive deformation of eggs	F_2	Sasaki et al. (2004)
WL × F	Highly inbred WL (G-B1) × highly inbred F (M15.2 and M5.1) lines	F_2	Zhou et al. (2003)
WL × WL	Commercial layer lines, fixed for B blood group (B2 and B15)	BC	McElroy et al. (2005)
WL × WL	*Salmonella*-resistant and susceptible lines (6_1 and N) from IAH	F_2	Tilquin et al. (2005)
WL × WL	*Salmonella*-resistant and susceptible lines (6_1 and15I) from IAH	BC	Mariani et al. (2001)
WL × WL	MD-resistant (6_3) and susceptible (7_2) lines	F_2	Yonash et al. (1999)
WL × WL	Commercial chicken WL laying lines differing for behavioral traits (FP and open-field behaviors)	F_2	Buitenhuis et al. (2003a,b, 2004)
WR × WR	Divergently selected (S_{41}) for high or low BW at 8 wk	F_2	Jacobsson et al. (2005) Park et al. (2006)

[1]BS = broiler breeder sire line; BD = broiler breeder dam line; BSD = broiler breeder F_1 progeny, F = Fayoumi; WL = White Leghorn; IW = ISA Warren; S = Satsumadori; SP = Spanish WR = White Plymouth Rock; RJF = Red Jungle Fowl; CR = Cornish Rock, LL = Lean line, FL = Fat line; GlP = Green-legged Partrigenous.

[2]Line descriptions are based on the information provided in the papers reporting QTL results. S_x = selection was carried out for x generation in founder line; ABR = antibody response; FP = feather pecking; MD = Marek's disease; SRBC = sheep red blood cells; WM = white meat; IAH = Institute for Animal Health.

[3]AB (BC_2) = advanced backcross population (BC_2 generation); AIL-F_9 = advanced intercross line F_9 generation; BC = backcross population; F_x = F_x population; F_2-F_3 = a third generation (G_3) was produced by intercrossing the F_2 individuals.

[4]An F_2 population from a WL × WL cross used in Buitenhuis et al. (2003a,b, 2004) was also used in Siwek et al. (2003a, 2006).

(**BC**), and F_1 designs. In these designs, the first generation is produced by crossing 2 divergent populations. A second generation (**G_2**) is produced by backcrossing to 1 of the parental lines (BC design) or by intercrossing the first generation individuals (F_2 design). In the F_1 design used by Kaiser et al. (2002), Kaiser and Lamont (2002), and Deeb and Lamont (2003), males from an outbred line were crossed to an inbred line to produce F_1 half-sib families whose genotypes and phenotypes were used for QTL mapping, as in a half-sib design (Soller et al., 2006). In F_2 and BC designs, phenotypic information of G_2 is used for QTL mapping. In an F_2-F_3 design, a third generation is produced by intercrossing the G_2 individuals, and mean phenotypes of the third generation progeny of G_2 birds are used for analysis of QTL segregation in the G_2. Among QTL mapping designs, the F_2 design is the most frequently used in chicken QTL studies.

The aforementioned designs have the advantage of detecting QTL with a limited number of markers across the genome because of the extensive LD that is generated; however, the resolution of QTL location that is obtained using these designs is generally low (Soller et al., 2006). High-resolution mapping of QTL location can be obtained using an advanced BC (**AB**) strategy, in which the BC animals carrying recombinant chromosomes are identified and progeny tested. Such an approach was used by Abasht et al. (2006) to refine a fatness QTL region on GGA5 (Figure 1). The advanced intercross line (**AIL**) approach proposed by Darvasi and Soller (1995) can also be used to improve resolution of QTL location (Jennen et al., 2005). To obtain AIL, the F_2 generation is further intercrossed for several generations.

Evidence for QTL

QTL Distribution Across Phenotypic Traits and Across the Genome. Table 3 summarizes the distribution of identified QTL across the 5 major trait categories. About 700 QTL were reported in the reviewed studies. There were more QTL for growth than the other traits, possibly because more studies investigated traits from this category. About 31% of the QTL were significant at the 5% genome-wise level. The percentage of genome-wise significant QTL was less for DR (6%), because most studies of DR traits used single-point analyses.

The Cmap location of QTL was visualized (Figure 1) with MapChart 2.0 (Voorrips, 2002). There was no obvious pattern for QTL distribution across the genome. However, the distal end of some of the macrochromosomes tended to have a lower QTL density, which is unexpected, because a higher gene density in subtelomeric regions of the macrochromosomes has been reported (Hillier et al., 2004). Relatively poor coverage with genetic markers could have reduced the power of detecting QTL in these regions.

Consensus of QTL Location for Similar Traits from Independent Studies Provides Strong Evidence for QTL Presence. Despite differences in experimental conditions and populations used for QTL mapping, independent studies found QTL for similar traits in similar locations in several instances (Figure 1). Considering that the confidence interval of most QTL location estimates covers over 20 cM (Soller et al., 2006) and sometimes the complete chromosome (Schreiweis et al., 2005), QTL for similar traits that are reported as separate in Figure 1 may actually represent 1 QTL. For example, QTL for antibody response to different antigens were detected in the same chromosomal regions in independent populations in GGA3 (25 to 85 cM), GGA5 (65 to 90 and 198 cM), GGA6 (55 to 85 cM), GGA8 (45 to 80 cM), GGAZ (65 to 115 cM), and GGA18 (0 to 40 cM; Figure 1). These results provide strong evidence of QTL for antibody response in these regions. It is not possible at the level of resolution of the current studies to differentiate between a single broad-function QTL in each region that controls antibody response to multiple antigens and multiple closely spaced QTL that exert separate influence on response to the different antigens. Future fine-mapping studies with higher marker saturation in populations that allow greater resolution (AIL, AB, and LD mapping in outbred lines) and tests of candidate genes in these QTL regions may help to resolve these questions.

Some QTL were reported only in 1 study and were not detected in other studies that analyzed the same trait in different populations. Population type and size, genetic background, segregation of specific QTL in specific populations, and differences in trait definition or measurement may have led to inconsistencies in QTL results among experiments. Furthermore, type I and II errors in QTL detection may also contribute to lack of agreement in QTL results.

Consensus of QTL Location for Different Traits Explains Genetic Basis of Correlation Among Traits. Quantitative trait loci affecting different traits were mapped to similar chromosomal regions (Figure 1). Such results represent evidence for the basis of genetic correlations among traits and for correlated response to selection, if they are indeed controlled by the same pleiotropic QTL or by closely linked QTL that are in LD. Higher resolution analysis is required to distinguish LD from pleiotropy.

In the instance of closely linked QTL, the results of high-resolution mapping would help to avoid selecting for an undesirable QTL allele for 1 trait while selecting for the desirable allele for the other trait in MAS. However, constraints will exist if an undesirable correlation is caused by pleiotropy. In this case, selection for the desirable QTL allele effect for 1 trait would cause an undesirable (antagonistic) effect in the other traits, and the best approach would be to select for the allele that has the most beneficial net effect across traits.

Complicating Factors for Application of QTL Results

Parent-of-Origin Effect. Tests to detect QTL with parent-of-origin effects have been conducted in only some of the summarized studies (Ikeobi et al., 2002; Sewalem et al., 2002; Buitenhuis et al., 2003a,b; Siwek et al., 2003b;

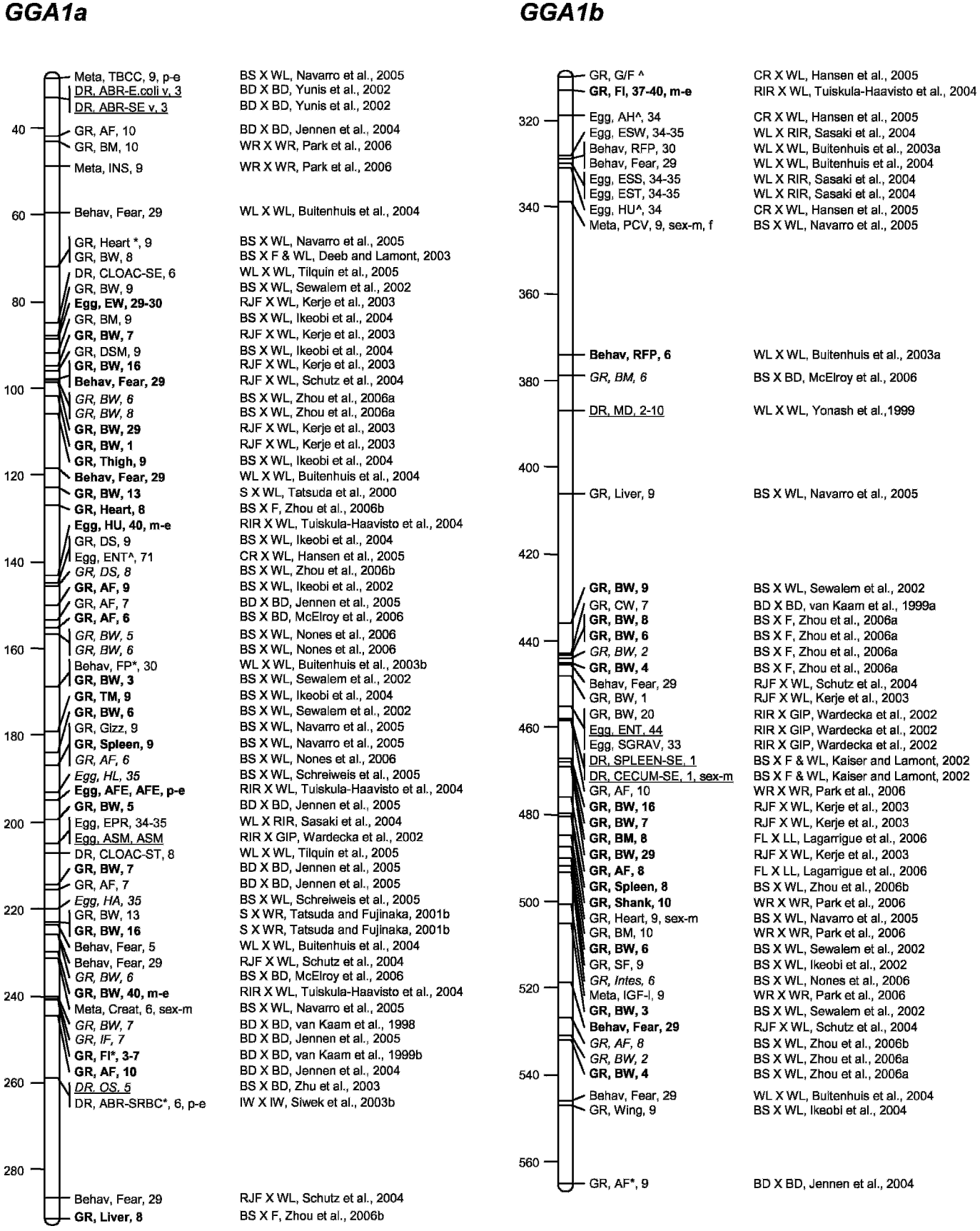
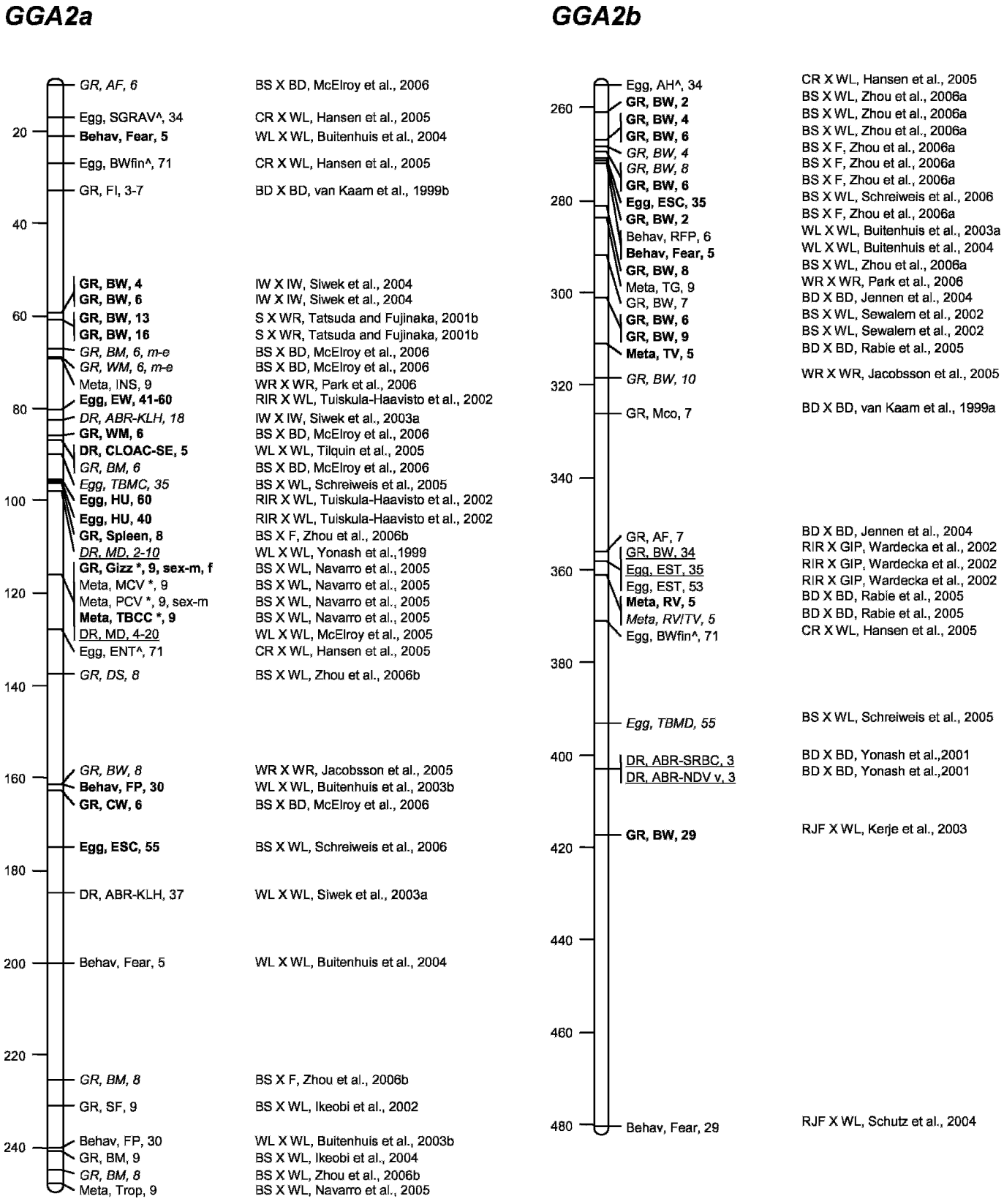


Figure 1 continued.



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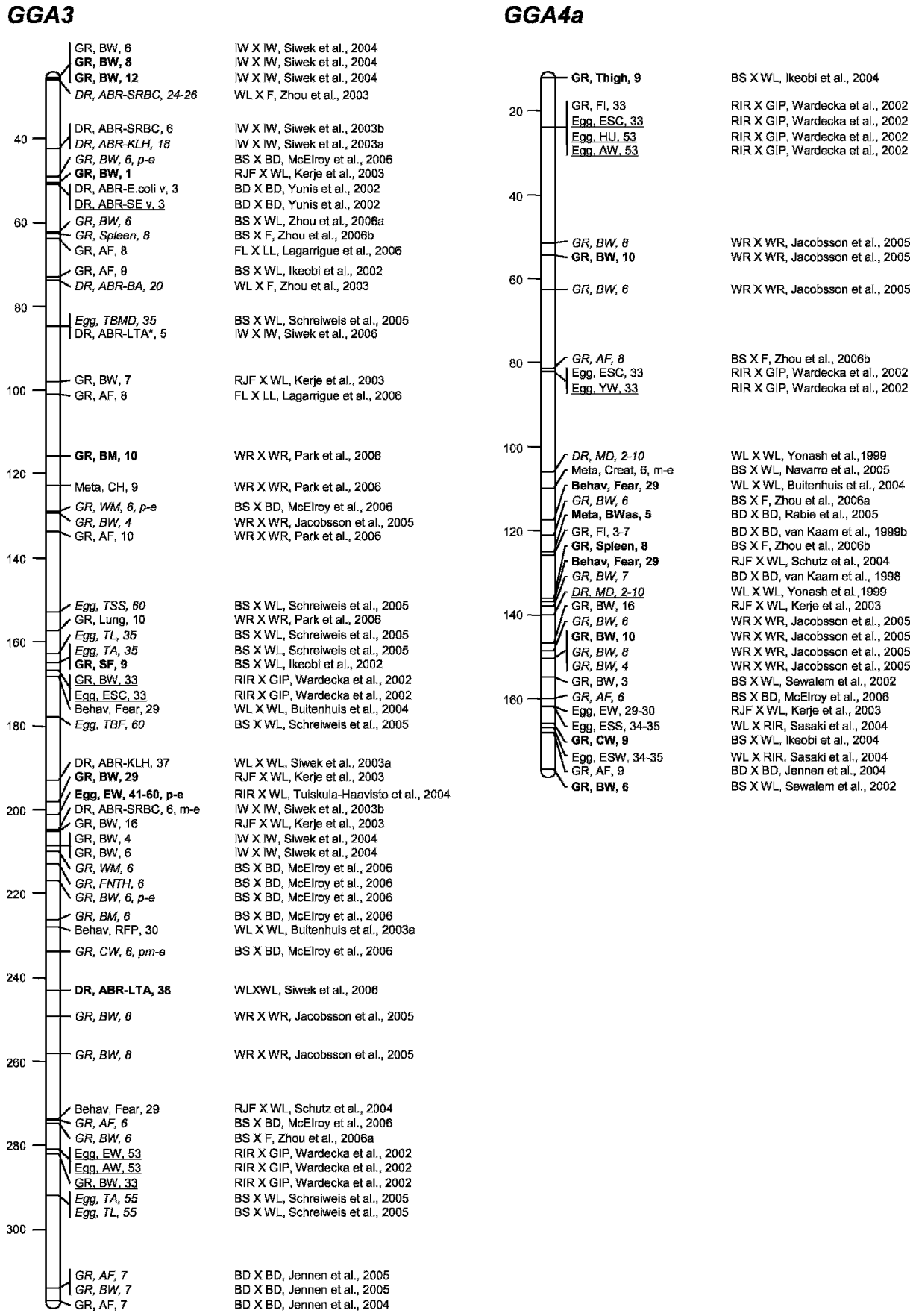

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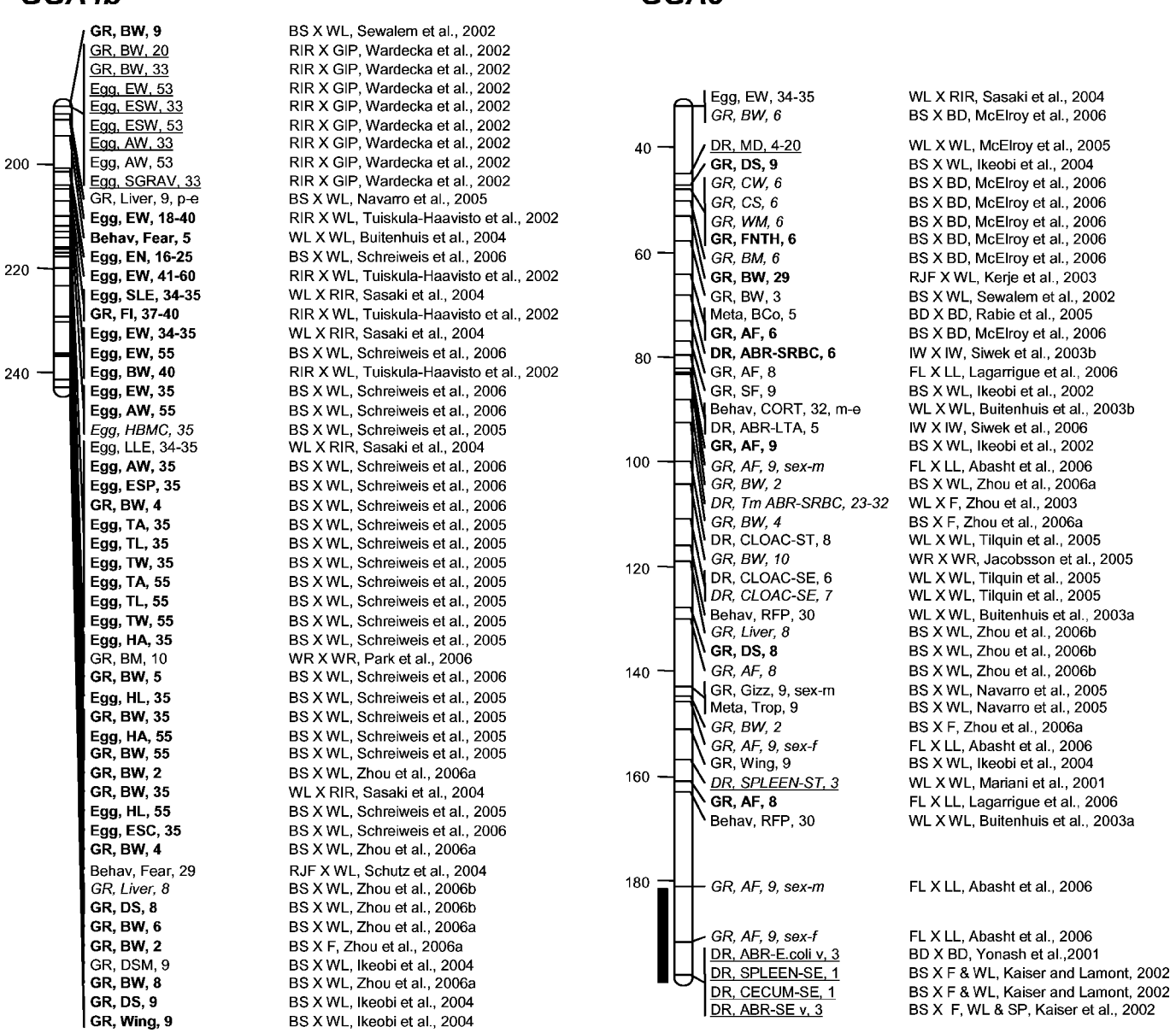

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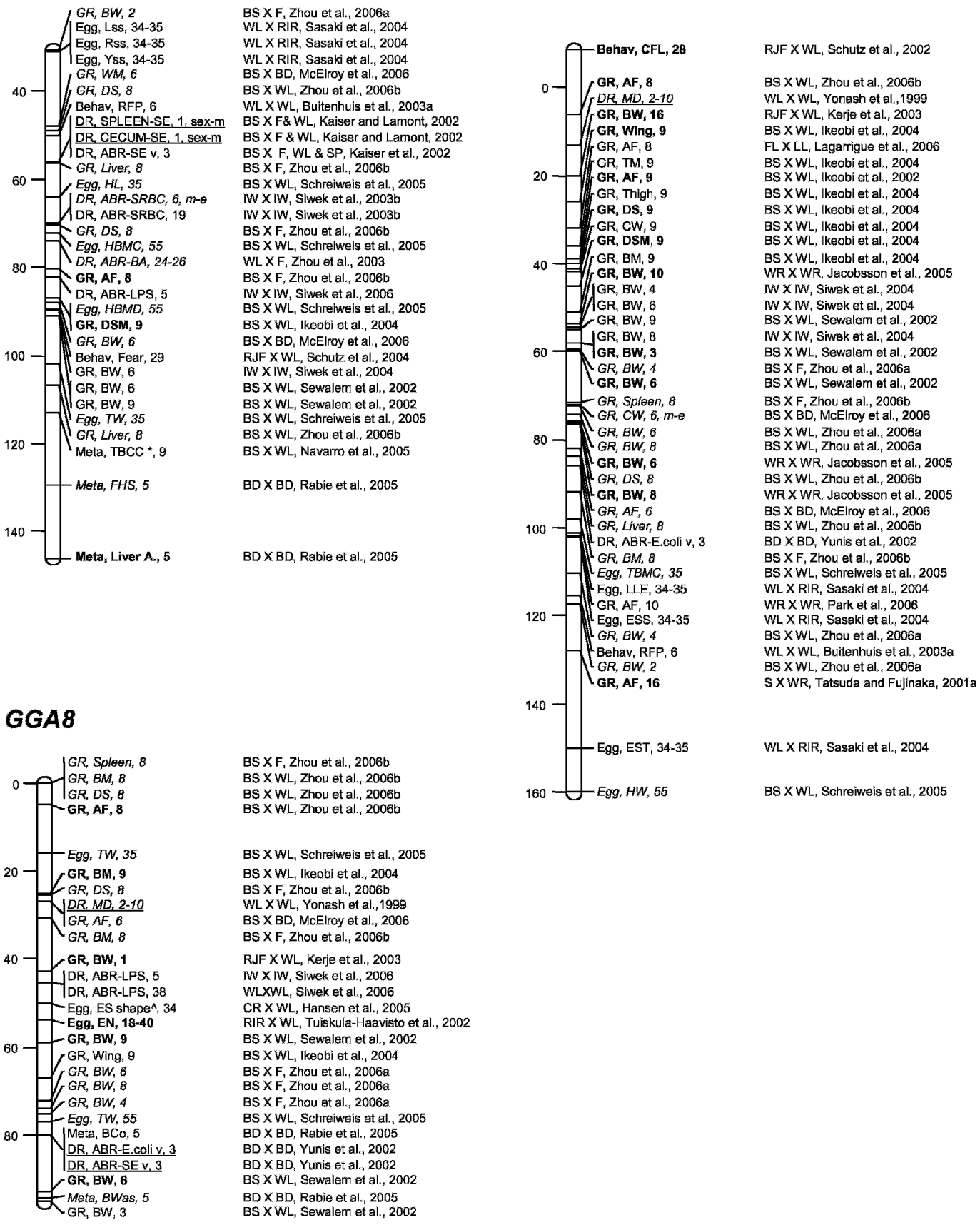

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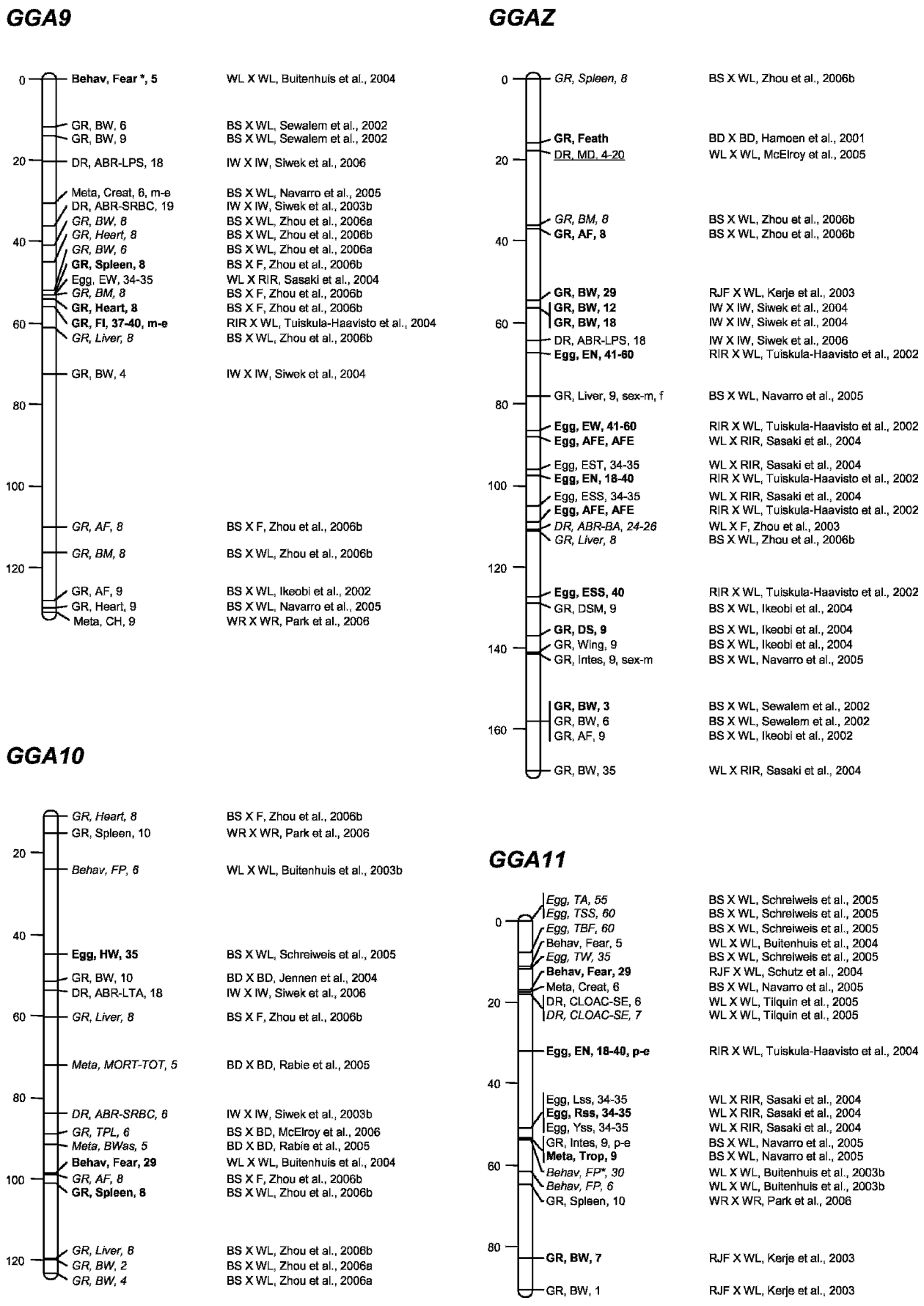

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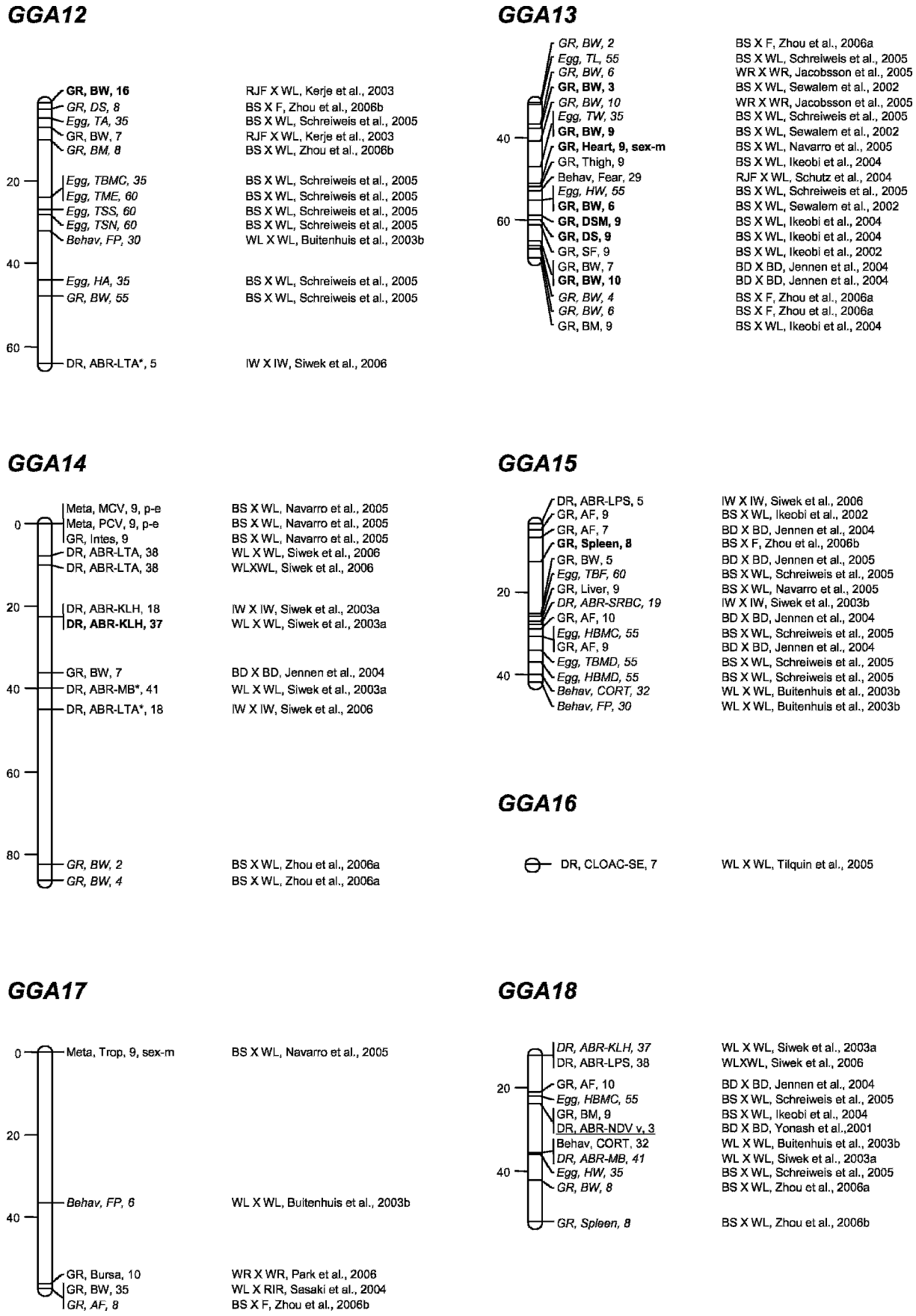

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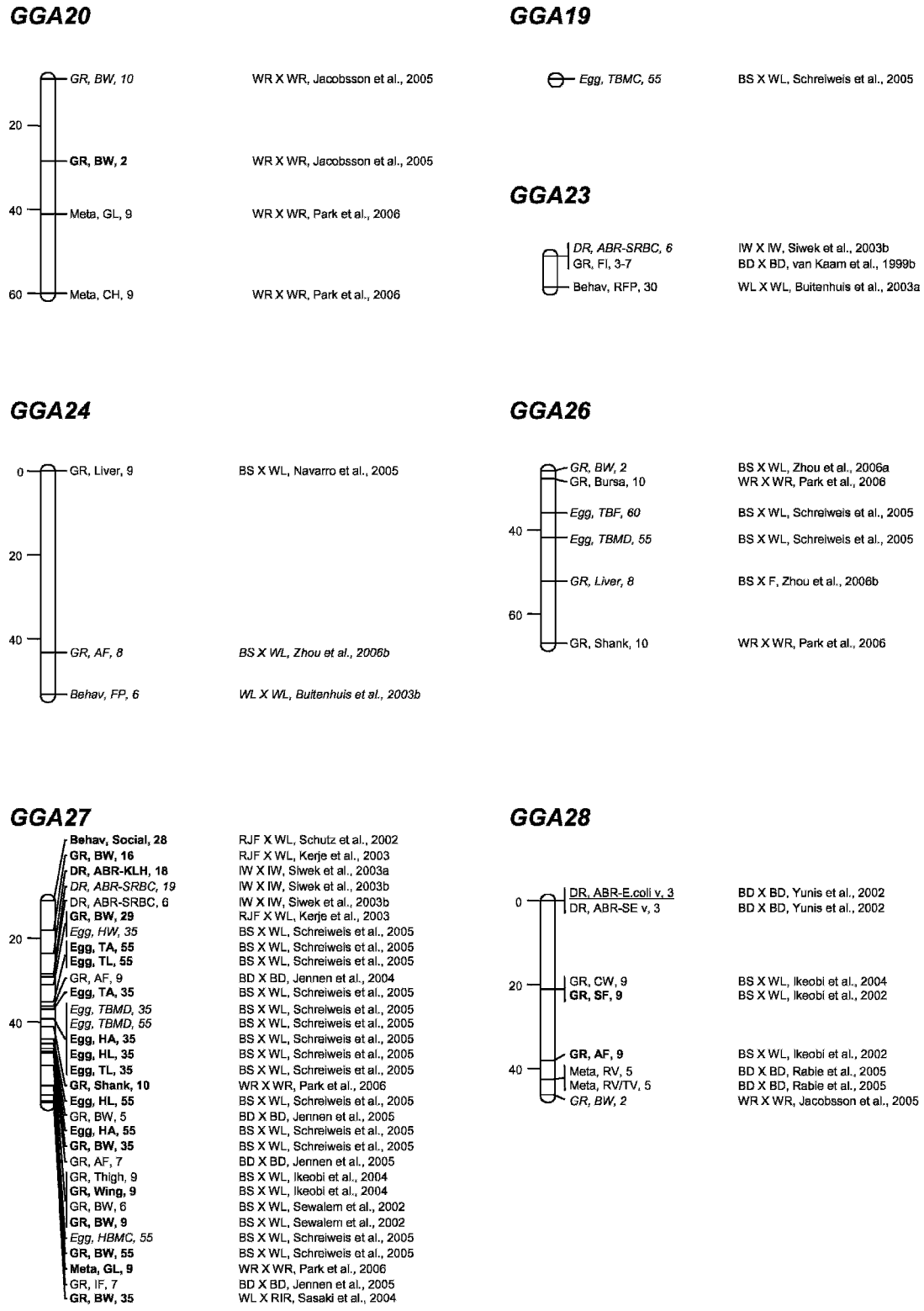

Figure 1. Chicken QTL detected on different chromosomes. QTL locations are presented based on Chicken Cmap distances (http://www.animalsciences.nl/Cmap) of markers used for QTL detection. For each QTL are listed, from left to right: trait class, trait, age (wk) at phenotypic measurement, QTL interaction (if there was any), and type of cross. The abbreviations for traits, trait class, and cross are described in Tables 1 and 2, respectively. Quantitative trait loci interactions include QTL × sex interaction (sex-m, sex-f, and sex-m,f for significant and suggestive QTL effect in male, female, and both sexes, respectively) and QTL with parent of origin effect (p-e, m-e, and pm-e for paternal, maternal, and different level of paternal and maternal expression, respectively). Genome- and experiment-wise significant ($P < 0.05$) QTL are presented in bold. Suggestive QTL: genome-wise ($P < 0.2$), chromosome-wise ($P < 0.05$), and single-point ($P < 0.01$) QTL are presented in roman, italic, and underlined letters, respectively. Quantitative trait loci that were suggestive in both chromosome-wise ($P < 0.05$) and single-point ($P < 0.01$) analyses are presented as both italic and underlined. The sign ^ represents QTL detected based on Markov chain Monte Carlo methods (Hansen et al., 2005), and statistic tests for these QTL are not comparable with the significant and suggestive QTL described above. The sign * indicates QTL for which 1 of the flanking markers presented in the original publication was not found in chicken Cmap. In these cases, the position of the other flanking marker was given as the QTL location. The thick line close to GGA5 is the fine-mapped QTL region for female-specific fatness QTL (Abasht et al., 2005).

Table 3. Distribution of identified QTL across trait categories

	Significance level[2]							
	Multipoint analyses							
Trait category[1]	GW (5%)	GW (20%)	CW	Single point analyses	MCMC	Total	GW significance (%)	PO[3]
GR	137	109	128	8	1	383	36	12
DR	5	30	23	25	—	83	6	3
Egg	49	20	46	19	9	143	34	4
Behav	15	27	8	—	—	50	30	1
Meta	7	26	5	—	—	38	18	5
Total	213	212	210	52	10	697	31	25

[1]GR = traits related to BW, body composition, and feed intake; Egg = traits related to egg production and egg quality; DR = traits related to disease resistance; Meta = traits related to metabolic parameters; Behav = traits related to behavior.

[2]Number of significant QTL at a 5% genome-wise (GW) level, with suggestive linkage evidence at the 20% GW level, at the 5% chromosome-wise (CW) level, at the 1% single-point level, and by test statistics based on Markov chain Monte Carlo (MCMC) methods.

[3]PO = number of QTL with parent-of-origin effect.

Buitenhuis et al., 2004; Ikeobi et al., 2004; Tuiskula-Haavisto et al., 2004; Navarro et al., 2005; Abasht et al., 2006; McElroy et al., 2006; Nones et al., 2006). The number of QTL reported with parent-of-origin effect is, therefore, likely to be underestimated (Table 3). There was agreement among multiple independent studies for QTL with parent-of-origin effect on GGA1, GGA3, GGA9, and GGA11 (Figure 1). The regions on GGA1 and GGA3, for which 3 independent studies reported parent-of-origin QTL, correspond to where chicken orthologs of mammalian imprinted genes were in silico mapped by Dunzinger et al. (2005). Both paternally and maternally expressed QTL were detected in these regions (Figure 1). In mammals, imprinted genes are also mostly seen in pairs or clusters, and most imprinted domains contain both maternally and paternally expressed genes (Vu and Hoffman, 2000; Reik and Walter, 2001; Dunzinger et al., 2005). Most chicken orthologs of mammalian imprinted genes showed synteny conservation between mammals and birds and have been mapped to distinct chromosomal regions that exhibit asynchronous DNA replication (Dunzinger et al., 2005). However, the imprinting center and many of the local regulatory elements identified in mammals have not been identified in analysis of the chicken ortholog to the imprinted mammalian *Ascl2-Igf2-H19* region (Yokomine et al., 2005). These findings collectively suggest that parent-of-origin-specific QTL that have been detected in chicken may result from genomic imprinting but may involve different mechanisms or genes than in mammals.

There are conflicting results on allelic expression analyses of some chicken orthologs of mammalian imprinted genes. Monoallelic expression of both paternal and maternal alleles of *IGF2* was reported by Koski et al. (2000). However, biallelic expression of *IGF2* and of several other chicken orthologs of mammalian imprinted genes (*IGF2R*, *ASCL2*, and *INS*) were reported by O'Neill et al. (2000), Nolan et al. (2001) and Yokomine et al. (2005). Such studies have, however, been limited to a few chicken orthologs of mammalian imprinted genes. In addition, genes that are subject to imprinting may differ between mammals and birds. Large-scale evaluation of allelic gene expression by simultaneous analysis of high-throughput single nucleotide polymorphisms (**SNP**) and microarrays would help to answer this biologically important question.

Hidden Genetic Variation. In chickens, as in other species, QTL mapping studies enable empirical detection of transgressive (cryptic) genetic variation by identifying transgressive alleles (Frankel, 1995). Transgressive QTL alleles show trait effects that are in the opposite direction to what would be expected based on the mean phenotypic difference among the breeds that are crossed. Examples of transgressive QTL alleles detected in the chicken include a low-fat allele from a high-fat line by Abasht et al. (2006) and Zhou et al. (2006b), a disease-resistance allele from susceptible lines by McElroy et al. (2006), and a low-egg weight allele from a high-egg weight line by Tuiskula-Haavisto et al. (2002). Transgressive alleles may exist in a population because of no or limited selection for the trait, drift, pleiotropic effects of the QTL allele on other traits that are under selection, or close linkage and LD with QTL that are under selection.

Another possible mechanism for the appearance of transgressive alleles is based on a shift in the allele effect in the mapping population as a result of the change in the matrix of genetic interactions (Gibson and Dworkin, 2004). This change can occur when crossing populations with different genetic backgrounds or when transferring a QTL allele to another genetic background (QTL introgression). In these cases, the transgressive effect is caused by epistasis in synergetic or antagonistic ways (Gibson and Dworkin, 2004; Carlborg et al., 2006). For example, dramatic improvement in red fruit color was observed in a nearly isogenic line produced by transferring the transgressive QTL allele from the wild tomato (in which fruits remain green even when ripe) to a cultivated tomato (Tanksley and McCouch, 1997)—an example of a transgressive effect produced by a combination of alleles at different loci (epistasis) from the 2 types of tomato.

Results from chicken QTL studies clearly show that beneficial alleles can be found in lines with generally

undesirable characteristics. However, it would be difficult to fine map such QTL or to use them in selection without understanding whether the transgressive alleles represent true single locus effects or appeared because of epistasis. Furthermore, possible negative pleiotropic effects of transgressive alleles should be evaluated before using them in a selection program.

Epistatic QTL. The QTL results summarized in this review were detected using nonepistatic models that do not account for interactions among QTL. These models have been successful in detecting many QTL (Figure 1). However, Carlborg and Haley (2004) showed that additional QTL can be detected by simultaneous mapping of QTL using an epistatic model. Total phenotypic variance was better explained by considering individual and epistatic QTL effects (Carlborg and Haley, 2004). Carlborg et al. (2003, 2004, 2006) reanalyzed chicken populations that were initially analyzed using traditional QTL methods with epistatic models for growth. Results showed important epistatic interactions for early growth rate and enabled identification of epistatic patterns and networks among QTL. Some statistically detected epistatic QTL did, however, not have an epistatic pattern that was biologically meaningful (Carlborg and Haley, 2004).

Epistatic QTL mapping could help to better understand the genetic architecture of quantitative traits, which is so important in dissecting the underlying quantitative trait genes and for implementating QTL results in selection programs. However, it is difficult to detect epistatic interactions among closely linked QTL based on an analysis of F_2 populations because of limited mapping resolution. Therefore, breakdown of LD among epistatic QTL as a result of recombination in a high-resolution QTL mapping program can lead to a change in QTL effect, appearance of new QTL, or disappearance of the targeted QTL.

QTL by Sex Interaction. Several QTL that show interactions with sex have been identified in both autosomal (GGA1, GGA2, GGA5, GGA6, GGA13, and GGA17) and sex (GGAZ) chromosomes (Figure 1). A QTL by sex interaction could result if the QTL affects only 1 sex (sex-specific effect), affects both sexes but at different levels (sex-biased effect), or affects both sexes but in opposite directions (sex-antagonistic effect; Anholt and Mackay, 2004). More generally, a QTL by sex interaction can be considered as a genotype by environment interaction, considering sex as an organismal environment for gene expression (Abasht et al., 2006).

Not all chicken QTL studies that included both sexes have evaluated evidence for QTL by sex interactions, and some did not report the specific traits for which the sex interaction was detected. The number of QTL reported with sex interaction (~20) is, therefore, likely to be underestimated. Furthermore, in some studies, the QTL by sex interaction was tested only for locations that were significant in the initial analysis using models without sex interaction (Ikeobi et al., 2002; Sewalem et al., 2002; Ikeobi et al., 2004; Nones et al., 2006), which does not detect QTL with sex-antagonistic effects and has less power to detect QTL with sex-specific and sex-biased effects. Conducting a full genome scan with a QTL by sex interaction model or conducting the analysis separately for each sex could help to detect these kinds of interactions. However, the larger number of tests conducted could also lead to an increase in false positive results. Further experiments are needed to confirm QTL by sex interactions detected in an initial genome scan before application in selection. Using an AB generation (BC_2), Abasht et al. (2006) confirmed a sex interaction for fatness QTL that was identified in an F_2 population.

CONCLUSIONS AND FUTURE PROSPECTS

This review clearly demonstrates that chicken QTL studies have been successful in identifying QTL underlying variation in economically important traits. In combination, the results of the primary QTL studies enabled identification of basic information on the genetic architecture underling complex traits in the chicken. This information can be helpful in identifying genes or mutations underlying the QTL and in the application of genomic information in marker-assisted breeding programs.

To date, most of the chicken QTL analyses have been carried out using experimental crosses, which limits direct application of the QTL results in commercial lines. Revolutionary opportunities have now opened for analysis of quantitative traits in the chicken because of the availability of sequence information (Hillier et al., 2004) and a large number of SNP (Wong et al., 2004). The major changes that are occurring in quantitative trait analysis in the chicken include changes in genotyping strategies (SNP markers instead of microsatellite or RFLP markers) and statistical analysis methods (LD mapping; Soller et al., 2006). These new approaches allow the use of commercial breeding populations for QTL mapping, which enables direct application of QTL results in commercial breeding programs (de Koning et al., 2003, 2004; Soller et al., 2006).

About 700 curated QTL from the reports analyzed for this review paper have been used to establish the Chicken QTLdb (http://www.animalgenome.org/QTLdb/chicken.html) as a new member of the Animal QTLdb, which is expanded from the Pig QTLdb described in Hu et al. (2005). Similar to the Pig QTLdb, the Chicken QTLdb integrates available chicken QTL data in the public domain by a chicken consensus linkage map (Schmid et al., 2005), which facilitates the use of the QTL information in future studies. The Chicken QTLdb also introduces a chicken trait classification and ontology to describe traits. A notable feature of the Chicken QTLdb is that it allows publishers and authors to enter their own data directly into the database, and thus the database will be continually updated. The chicken QTLdb allows for easy search and comparison of QTL results from different studies. This facilitates a narrowing of possible chromosomal regions from overlapping QTL results of different studies, which will speed positional searches for underlying genes (Hu et al., 2005). Because the Chicken QTLdb is part of the Animal QTLdb, QTL comparisons among comparable

traits can be conducted across species, which may facilitate additional narrowing of QTL-containing chromosomal regions and will help locate underlying genes and previously undiscovered QTL with inferences from different species.

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NOTE IN PROOF

Several errata have recently been published for papers cited in this review. The original citations and their respective errata are as follows: Buitenhuis et al., 2003a, erratum in Poult. Sci. 2006. 85:1117; Buitenhuis et al., 2003b, erratum in Poult. Sci. 2006. 85:1115–1116; Buitenhuis et al., 2004, erratum in Behav. Genet. 2006. Online First; Siwek et al., 2003a, erratum in Poult. Sci. 2006. 85:1118–1119; Siwek et al., 2004, erratum in Poult. Sci. 2006. 85:1120.