ÇUKUROVA UNIVERSITY
INSTITUTE OF NATURAL AND APPLIED SCIENCES

PhD DISSERTATION

Faheem Shehzad BALOCH

QTL MAPPING OF SOME IMPORTANT AGRONOMIC CHARACTERISTICS IN BREAD WHEAT (*Triticum aestivum* L.)

DEPARTMENT OF FIELD CROPS

ADANA, 2012

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We certify that the thesis titled above was reviewed and approved for the award of degree of the Doctor of Philosophy (PhD) by the board of jury on 22/06/2012.

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ABSTRACT

Ph.D THESIS

QTL MAPPING OF SOME IMPORTANT AGRONOMIC CHARACTERISTICS IN WHEAT (*Triticum aestivum* L.)

Faheem Shehzad BALOCH

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This research was conducted to genetically dissect the quantitative trait loci (QTLs) for spike related and other agronomic traits, and to understand their mode of inheritance in bread wheat during the years of 2009-2011.

A genetic linkage map was constructed based on recombinant inbred lines (RILs) derived from cross between the Turkish cultivar Gerek and Moroccon cultivar Arrehane by using DArT markers anchored with SSR markers. This map consisted of 54 linkage groups belonging to twenty one bread wheat chromosomes, spanning to a total of 935.629 cM. Total number of markers mapped varied from 5 to 87 depending on the chromosomes. Seventy seven *DArT* markers with unpublished chromosomal locations were mapped on different chromosomes, whereas 23 *DArT* markers were mapped on different chromosomes instead of their published chromosomal locations. Interval mapping was conducted to identify QTLs for agronomic and spike related traits by using four different environments/locations. The QTL analysis led to the detection of 120 QTLs. Several important and novel genomic regions for spike morphology were detected. In addition, some of the QTLs showed pleiotropic effects for different quality traits. Stable QTL clusters influencing the 1000 grain weight, spike length, number of spikelets per spike, number of grains per spike, grain weight per spike, spike compactness and flowering time were identified on chromosomes 1A, 1B, 2B, 3D and 6B. It was concluded that DNA markers might provide an opportunity for increasing the frequency of desirable alleles for spike related traits through marker assisted selection.

Keywords: Bread wheat, QTL, Linkage map, DNA markers, spike traits

ÖZ

DOKTORA TEZİ

BUĞDAYDA (*Triticum aestivum* L.) BAZI ÖNEMLİ TARIMSAL KARAKTERLERİN QTL HARİTALANMASI

Faheem Shehzad BALOCH

ÇUKUROVA ÜNİVERSİTESİ
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: Prof. Dr. Bahattin TANYOLAÇ

Bu araştırma, ekmeklik buğdayda başak ile ilgili kantitatif özellik gen lokuslarının genetik olarak ortaya çıkartılması ve bunların kalıtım şekillerinin anlaşılması amacıyla 2009-2011 yılları arasında sürdürülmüştür.

Türk ekmeklik buğday çeşidi Gerek ile Fas ekmeklik buğday çeşidi Arrehane'nin melezlenmesi ve elde edilen melezlerden tek tohum yöntemiyle oluşturulan rekombinant kendilenmiş hatlarda SSR markerlarla desteklenmiş DArT markerları kullanılarak genetik bağlantı haritası oluşturulmuştur. Bu harita 21 buğday kromozomu üzerinde toplam 935.629 cM uzunluğundaki 54 bağlantı grubundan oluşmuştur. Haritalanan marker sayısı kromozomlara bağlı olarak 5 ile 87 arasında değişmiştir. Farklı kromozomlarda daha önce kromozom üzerindeki yeri bilinmeyen 77 DArT markerı haritalanmıştır. 23 DArT markerının farklı kromozomlar üzerindeki yeri ise daha önce literatürde bildirilen yerlerinden farklı olarak saptanmıştır. Tarımsal ve başak özellikleri ile ilgili kantitatif özellik lokuslarını (QTL) saptamak amacıyla incelenen rekombinant kendilenmiş hatların dört farklı çevrede yetiştirilmesiyle elde edilen morfolojik ve fenolojik veriler ve bağlantı haritalarından yararlanılarak QTL haritalaması yapılmıştır. QTL analizleri sonucu 120 QTL belirlenmiştir. Başak morfolojisi ile ilgili çok sayıda yeni ve önemli genomik bölgeler saptanmıştır. Ayrıca, saptanan QTL'lerin bazıları farklı kalite özellikleri için pleiotropik etki göstermiştir. 1A, 1B, 2B, 3D ve 6 B kromozomları üzerinde 1000 dane ağırlığı, başak uzunluğu, başak başına başakçık sayısı, başak başına dane sayısı, başak başına dane ağırlığı , başak sıklığı ve çiçeklenme zamanını etkileyen stabil QTL grupları tanımlanmıştır. Araştırma sonuçlarına dayanarak, DNA markerlarının başak ile ilgili özellikler için arzu edile allelerin frakansını markera dayalı seleksiyon yoluyla artırma olanağı sağlayabileceği sonucuna varılmıştır

Anahtar Kelimlere: Ekmeklik Buğday, QTL, Bağlantı haritası, DNA markerları, Başak özellikleri

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Faheem Shehzad BALOCH

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LIST OF ABBREVIATONS:

AFLP : Amplified fragment length polymorphism
APS : Ammonium per Sulphate
BARC : Beltsville Agricultural Research Center
BP : Base Pair
CIM : Composite Interval Mapping
CIMMYT : Centro Internacional de Mejoramiento de Maiz y Trigo
cM : Centi Morgan
CSL : Chromosome Substitution Line
CU : University of Cukurova
DArT : Diversity Array Technology
DH : Double haploid
DMF : Di-Metyl-Formamide
Exp : Explanations
GIC : Genotypic Information Coefficient
GWM : Gatersleben Wheat Microsatellites
ICARDA : International Center For Agricultural Research In The Dry Areas
ICC : International Association for Cereal Science and Technology
ISSR : Inter simple sequence repeat
IM : Interval Mapping
ITMI : International Triticeae Mapping Initiative
Kb : Kilo Base Pair
KD : Kernel Dimensions
KL : Kernel Length
KPS : Kernel per Spike
KW : Kernel Width
LOD : Logarithm of the odds ratio
LRT : Likelihood Ratio Test
MAS : Marker Assisted Selection
Mb : Mega Base Pair
ML : Map Length

MD	: Marker Density
MQTL	: Map QTL software
NIL	: Near isogenic lines
NR	: Non Recombinant Haplotypes
PIC	: Polymorphism Information Contents
QTL	: Quantitative Trait Loci
RAPD	: Randomly Amplified Polymorphic DNA
RFLP	: Restriction Fragment Length Polymorphism
RICL	: Recombinant Inbred Chromosome Lines
RILs	: Recombinant Inbred Lines
RSL	: Recombinant Substitution Line
SCRI (SCRs)	: Single-Chromosome Recombinant Lines
SIM	: Simple Interval Mapping
SNP	: Single-Nucleotide Polymorphisms
SSR	: Microsatellites (Simple Sequence Repeat)
SRAP	: Sequence-Related Amplified Polymorphism
STRs	: Simple Tandem Repeats
TBE	:Tris/Borate/EDTA
TCA	:Trichloroacetic acid
TKW	: Thousand kernel weight
TRAP	: Target Region Amplification Polymorphism
USDA	: United States Department of Agriculture
WANA	: West Asia, North Africa.
X^2	:Chi-square test
YrBP	: Years before Present

1. Introduction

Wheat (*Triticum aestivum* L.) is one of the most important food crops worldwide, which feeds about 40% of the world's population (Gupta et al. 2008). Given the current land usage, humans need to increase wheat production at an annual rate of 2%, to meet the growing demand imposed by the population (Gill et al. 2004). Breeding wheat cultivars with increased yield potential can contribute to meet at least half of the desired production increases and the remaining half can come through better agronomic and soil management practices (Reynolds and Borlaug 2006). Therefore, selection for high grain yield is an important focus in wheat breeding programs. Grain yield is a complex trait and usually controlled by a number of quantitative trait loci (QTL) with minor effects. It is influenced by environmental factors, which make it difficult to be manipulated and improved in breeding programs. The improvement is slow due to the lack of genetic information about the number, location and contribution of each QTL to the final expression of yield (Koebner and Snape 1999). Grain yield and agronomic performance are the most commonly measured, but poorly understood crop traits. This is despite the fact that these traits are economically the most important and are complex in nature, exhibiting low heritability. The grain yield of wheat is determined by three yield components, i.e., number of productive spikes per unit area, number of kernels per spike and kernel weight. Its increase is one of the major factors that have contributed to wheat yield improvement, which mainly resulted from increase in number of kernels per spike, or from increase in both number of kernels per spike and number of spikes per unit area.

With the advent of DNA molecular markers, the genetic bases of yield and its related traits have been extensively analyzed in wheat, rice, maize, barley, sorghum and other crops and a great achievement had been gained (Bernardo 2008). Modern strategies for investigating the genetic basis of grain yield and agronomic performance were first established in the 1980s with the use of molecular markers. During the last two decades, both genomic mapping and sequencing methods have advanced significantly to provide tools for scientists to explore genome structure and

function in many organisms. Genetic maps offer a framework for carrying out evolutionary and comparative genomic studies (Ahn and Tanksley 1993). They are also crucial in the search for Mendelian and quantitative trait loci (QTL) (Lander and Botstein 1989), and understanding the organization and dynamics of organism genome such as landscape of linkage disequilibrium (LD) (Flint-Garcia et al. 2003). Genetic linkage maps based on molecular markers have become an important tool for genome analysis, detection of quantitative trait loci (QTL) underlying important traits, physical mapping, map-based cloning and marker-assisted selection. Construction and improvement of a genetic map for wheat is an important task in order to facilitate quantitative trait locus (QTL) analysis and the development of tools for marker assisted breeding. Marker assisted selection (MAS) is an important plant breeding tool that could be used in the introduction of favourable alleles. With the aid of MAS the breeder can focus on introgressing only alleles for the desired quantitative trait loci (QTL) and avoid introgressing those that could have a negative impact on the trait of interest on the general background. The use of phenotypic selection, instead of MAS, tends to be more time consuming, greatly depends on the environment and requires large populations and much field testing (Reyna and Sneller 2001).

A genetic linkage map constructed from a population segregating for a trait of interest is required for QTL identification. Until now many genetic linkage maps and QTL analysis had been published since 1998 (Vaissayre et al. 2012). These authors also elaborated the gap of durum linkage map for gene pool from diverse regions. Genetic and physical maps and QTL analysis conducted till now were mainly belonging to genetic pools of only few geographic regions. Most of the genetic maps published yet mainly belong to North America and some European gene pool.

Bread wheat originated in the Eastern Mediterranean and has been farmed in this region for the last 12 thousand years (Habash et al 2009). This region is located within the Fertile Crescent, a part of area that stretches from Jordon, Lebanon, Syria, southeastern Turkey and Tigris and Euphrates rivers into Iraq and western flanks of Iran. Northern Syria and southeastern Turkey are the core area of plant domestication, where first einkorn and wild emmer wheat were originated and

domesticated near Karacadag Mountain, and is one of the most important diversity centers for bread and durum wheat and contains unique landraces (Ozkan et al 2002). Turkey is main producer of bread wheat in the world. During the last 35 years wheat production in Turkey steadily increased, reaching about 21 million tons/year out of 9 million ha (the seventh largest area in the world; Altintas et al., 2008). Genetic resources from Turkey contributed greatly to the increase of wheat production in many countries. Germplasm exploration and collection missions led to the evaluation of sampled materials in different countries, and several landraces (e.g. Turkey Red) were largely utilized to breed new varieties. In Turkey modern wheat breeding started in 1925. The main goal was to select lines adapted to the different regions of the country from local population. This breeding effort quickly produced many cultivars. In 1967 the National Wheat Release and Training Project was established, with the contribution of international organizations resulting in the Turkish Green Revolution. Since then many cultivars (like Cham1) were introduced from international research centers such as ICARDA, CIMMYT and other foreign countries specifically targeting different areas. The national wheat breeding program, meanwhile, developed over 100 wheat cultivars, most of which had a significant impact on the economy. Unfortunately, genetic structure and QTL analysis of these cultivars is still largely unknown.

Fertile Crescent is the primary center of wheat domestication and diversity. Despite of the importance of gene pool from Fertile Crescent particularly from Syria and Turkey, no efforts had been made to get information about the genetic structure of Anatolian gene pool. Therefore to fill this gap and to provide additional information about the genetic structure and genome distribution of the bread wheat from Anatolia, first time we tried to attempt to construct a genetic linkage map and QTL analysis using mapping population composed of recombinant inbred lines from a cross between Turkish landrace “Gerek” and cultivar “Arrehan”developed by IRRI for Morocco. This population is ideally suited for studies designed to map genes and QTLs controlling traits of agronomic importance in bread wheat grown in the Mediterranean region.

Particularly, it was sought to:

a) Construct a genetic linkage map for Gerek x Arrehan population representative of Anatolian wheat,
b) Identify and locate QTLs linked to agro-morphological characteristics under different environmental conditions and
c) Identify molecular markers for agronomic traits to be used in the Marker-assisted selection program.

2. REVIEW OF LITERATURE

2.1. Importance of wheat

From antiquity, foods from wheat (*Triticum turgidum var durum* L. and *Triticum aestivum* L.) have been the staff of life for Middle East and the Mediterranean region and later for much of the world's population. Wheat is a major food crop worldwide and is used to produce a wide diversity of baked food products. Wheat rank first in the world grain production, and is one of the important crops for most of the population of more than 40 countries of the world, for both human as a staple food and food products, as it is abundant source of energy and protein (Patnaik and Khurana, 2001), and also wheat straw used as a animal feed. Wheat is the most widely grown cereal crop in the world, with an ever increasing demand. It plays a fundamental role in food security, and a major challenge is to meet additional requirements with new cultivars and improved cropping technologies (Pena 2002). Slightly more than 50% of the world wheat is grown in the developing countries (Braun *et al*., 2006). Bread wheat accounts for about 95% of World wheat production, and durum wheat the other 5%.

Wheat is superior to most of other cereal such as rice, maize and barley etc. in their nutritive value. Its grains not only contain starch (carbohydrate content of the wheat is 60-80% approximately), but also source of significant amount of protein (8-14%). Unlike other cereals, wheat contains a high amount of gluten, the protein that provides the elasticity necessary for excellent bread making. Gluten protein present in the seed endosperm give wheat dough stickiness, and its ability to rise when leavened, in other word, unique baking qualities, which make wheat a preferred staple food of most of the traditional farming communities in old world from Atlantic coast of Europe to northern part of Indian subcontinent (Zohary and Hopf, 2000). Hard wheat is high in protein (10-17%) and yields a flour rich in gluten, making it particularly suitable for yeast breads. The low-protein (6 to 10%) softer type yields flour lower in gluten and therefore, better suited for tender baked products, such as biscuits, pastries and cakes. Wheat is used also in the making of beer and whiskey to

a limited extent. Together with rice and maize, it provides more than 60% of the calories and proteins for human nutrition. With 620 million tons produced annually worldwide, wheat provides about one-fifth (20%) of the calories consumed by humans (FAO, 2007). Common wheat is used for making bread, cookies and pastries, whereas durum wheat is used for making pasta and other semolina products. **(**Dubcovsky and Dvorak, 2007**;** Patnaik and Khurana, 2001).

Both durum and bread wheats are often considered very versatile because they can be used for making many different types of food. Bread, pasta, bulgur, couscous, crackers, cookies, cakes, and various breakfast foods are all made with wheat. In the 20th century, global wheat output expanded by about 5-fold, but until about 1955 most of this reflected increases in wheat crop area, with lesser (about 20%) increases in crop yields per unit area. After 1955 however, there was a dramatic ten-fold increase in the rate of wheat yield improvement per year, and this became the major factor allowing global wheat production to increase. Thus technological innovation and scientific crop management with synthetic nitrogen fertilizer, irrigation and wheat breeding were the main drivers of wheat output growth in the second half of the century (Slafer and Satorre, 1999).

The cereal is an important component of the world's diet as they represent the largest protein of world food supplies. In the years ahead, bread and durum wheat, perhaps more than other cereals, can be expected to assume greater importance as a source of protein much of the world's increasing population. Wheat grain contains all essential nutrients; grain contains about 12% water, carbohydrates (60-80% mainly as starch), proteins (8-15%) containing adequate amounts of all essential amino acids (except lysine, tryptophan and methionine), fats (1.5-2%), minerals (1.5-2%), vitamins (such as B complex, vitamin E) and 2.2% crude fibers (USDA, 2006).

2.2. History and origin of wheat

The transition from hunting and gathering to agriculture had revolutionary consequences for the development of human societies. Crops such as wheat, played a crucial role in the establishment of complex civilizations in south west Asia. The

earliest evidence that man collected and used the cereals is from Ohalo II, a permanent site of epipaleolithic (19,000 BP) hunter-gatherers on the southwestern shore of the Sea of Galilee, Israel (Feldman and Kislev, 2007). In the same Area, Kislev et al. (1992) found grains of wild barley and wild emmer, and Piperno et al. (2004) presented evidence for grain processing and baking of flour. About 10,000 BP, hunter-gatherers began to cultivate wild emmer.

Wheat is the universal cereal of old world agriculture and since spread worldwide to become one of the major crops **(**Dubcovsky and Dvorak, 2007). Wild emmer wheat (Triticum dicoccoides) was one of the first cereals to be domesticated in the Fertile Crescent between c. 12,000 and c. 10,000 years ago (Özkan et al. 2011). This step provided the key for subsequent bread wheat evolution. Modern bread wheat (hexaploid) is a true breeding hybrid of its ancestry linked wild grasses still growing in Fertile Crescent (Patnaik and Khurana, 2001), a piece of land that stretches from present Israel, Jordon, Lebanon, western Syria, southeast Turkey, Tigris and Euphrates rivers into Iraq and western flanks of Iran (Özkan et al., 2002) and geographical distributions of wild ancestors of modern cereal species including wild wheat such as *Triticum urartu*, *Triticum boeticum*, *Triticum dicoccoides* and *Aegilops tauschii* interact in this region (Nesbitt Samuel 1996; Zohary and Hopf, 2000)**.** The first wheat to be cultivated successfully was einkorn, a diploid species, whereas durum and common wheat are polyploidy species that originated by interspecific hybridization of two and three different diploid species, respectively (Özkan et al., 2002). The diploid einkorn wheat *T. monococcum* was one of the first crops domesticated in the Fertile Crescent from the wild progenitor species *T. boeoticum.* The domestication of einkorn wheat occurred in the Karacadag mountain range in southeast Turkey (Heun et al. 1997). *T. urartu* contributed the A^u genome to all tetraploid and hexaploid wheats (Dvorak et al. 1993). *Triticum urartu* (AA) come in cross with *Aegilops speltoides*, the hybrid was tetraploid emmer, *Triticum dicoccoides* (AABB). Then domesticated emmer, *Triticum dicoccum* hybridized with *Aegilops tauschii* to form modern hexaploid wheat *Triticum aestivum* (Salamini et al., 2002). Feldman et al (1995) estimated that the latter hybridization event occurred approximately 8,000 years ago. Hybridization events are explained in the Fig. 2.1.

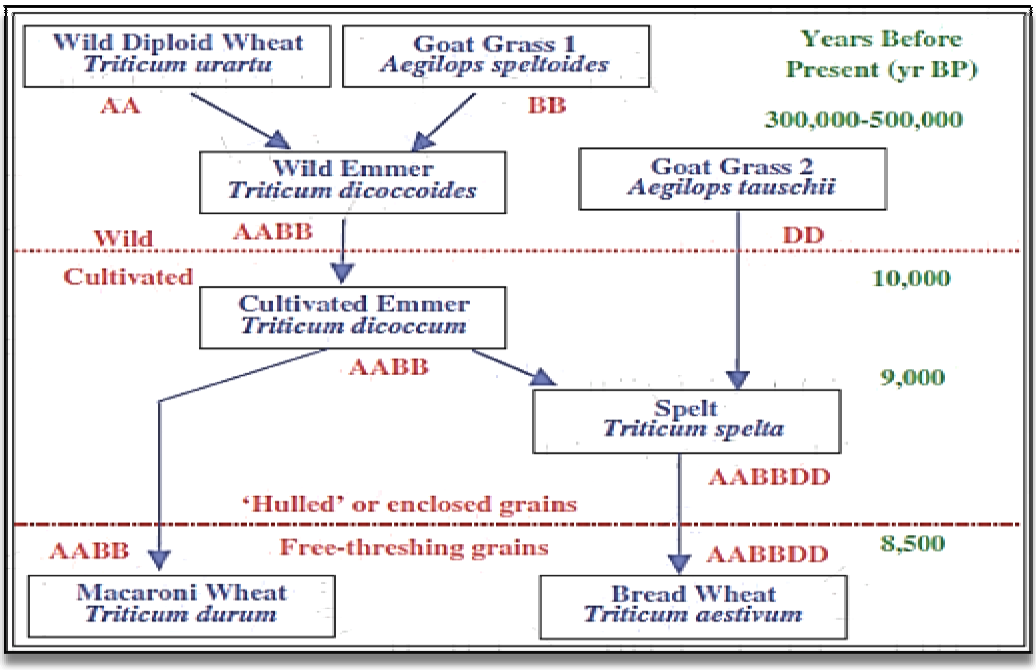

Fig. 2.1. The evolution of wheat from the prehistoric Stone Age grasses to modern durum wheat and bread wheat (Peng et al., 2011).

There are also two wild tetraploid wheat species known as *T. dicoccoides* (AuAuBB) and T. araraticum (AuAuGG). *T. dicoccoides*, wild emmer, grows naturally all over the Fertile Crescent. Emmer is believed to have been domesticated probably in southeast Turkey (Özkan et al. 2002)

Domestication of wheat caused substantial genetic erosion and that erosion was reinforced during modern breeding processes, and thus increased susceptibility and vulnerability to environmental stresses, pests and diseases (Fu and Somers 2009). Hence, its future genetic improvement as a high-quality nutritional food is paramount for feeding the ever-increasing human population. The best strategy for wheat improvement is to utilize the adaptive genetic resources of the wild progenitors, wild emmer *T. dicoccoides* and other wheat relatives (Feldman and Sears 1981).

Lev-Yadun et al. (2000) proposed a ‘‘core area’’ for the origins of agriculture within the Fertile Crescent. This was based on the proposition that wild einkorn and wild emmer from this area are genetically more closely related to the domesticated

crop plants than elsewhere. Therefore wheat genetic resources from this area might contain novel alleles for resistance to insect pest, diseases as well as drought tolerance and these genetic resources could be utilized for future cultivation practices and future breeding for higher yield and better quality.

2.3. Molecular mapping of wheat genome

Wheat (*Triticum aestivum* L.) is not only an important food crop worldwide but also an excellent and complex system for genome mapping and map based analysis. Understanding of wheat genomics and genetics using molecular marker is of great value for genetics and plant breeding purposes. The economic importance of wheat has triggered intense cytogenetic and genetic studies in the past decades that have resulted in a wealth of information and tools which have been used to develop wheat cultivars with increased yield, improved quality and enhanced biotic and abiotic stress tolerance (Carver 2009). The nuclear DNA content often varies somewhat among different cultivars (Bennett 1976). There are different ploidy level i.e diploid, tetraploid and hexaploid found in wheat. Common wheat is disomic hexploid ($2n = 6x = 42$), self-pollinated and segmental allopolyploid, containing three distinct but genetically related genomes: A, B and D; A and D sharing much similarity while B is more diverged, each genome with 7 chromosomes (Gupta et al., 2002), arranged in two way classification with seven homologous groups. The diploid progenitors of these have been established as *T.urartu* for A genome, *Aegilops tauschii* for D genome and *Aegilops speltoides* for B genome. A normal 2n plant has pairs of chromosome 1 of genome A (1A), chromosome 1 of genome B (1B), and chromosome 1 of genome D (1D), and so on for the seven chromosomes. Chromosomes 1B and 6B carry nuclear organizers on their short arms. In general the chromosomes of D genome are shortest. Chromosome 5B is most heterobrachial. The rest of the chromosomes are not substantially different from each other and thus are classified on the basis of total length and the arm ratios. The haploid DNA content of the bread wheat is aproximately 1.7×10^{10} bp (Nalini et al., 2007) with an average of 810 Mbp per chromosomes**.** Thus average wheat chromosomes is 25 fold

longer than an average rice chromosomes and three wheat chromosomes is equal to haploid maize genome and one half of the average wheat chromosomes is equal to haploid rice genome, making wheat one of the largest genome. It is estimated that each of the three wheat genomes contains approximately 40.000 to 50.000 genes. More than 80% of the DNA of bread wheat consists of repeated DNA sequences with transposons and retro transposons representing the highest proportion (Devos et al., 2005 ; Röder et al., 1998**)**.

2.3.1. New genomic tools for Wheat improvement

For the improvement of agronomically and economically important traits, plant breeding generally recombines traits present in different parental lines of cultivated/ wild species. Conventional breeding programs reach this goal by generating an F2 segregating population and then screening the phenotypes of pooled or individual plants for presence of desirable trait. This is followed by a time consuming and costly process of repeated backcrossing, selfing and testing. During this breeder depends on accurate screening methods and availability of lines with clear-cut phenotypic characters. Therefore, combination of complex characters encoded by multiple genes with additive effects (quantitative trait loci - QTL), recessive genes or accumulation (pyramiding) of genes encoding the same trait as different resistances against the same pathogen, is difficult to achieve with classical methods (Beckmann and Soller, 1986). QTL is a single locus from a series of polygenes, which are involved, in a quantitative trait of complex nature
(Paterson et al. 1988).

Use of molecular markers facilitates all these breeding processes, since it can accelerate the generation of new varieties and allow association of phenotypic traits with genomic loci. These properties make molecular markers indispensible for crop improvement. DNA markers also provide means of detecting and resolving complications such as linkage drag (Young and Tanksley, 1989), suppression of recombination and segregation distortion, encountered during interspecific gene introgression.

The first chromosome map was produced by Sturtevant with segregation data derived from studies on *Drosophila* (Crow and Dove, 1988). Markers on this map were phenotypic traits scored by visual observation of morphological characteristics of flies. By the early 1980s, biochemical markers had been employed as a general tool for QTL mapping (Weller et al. 1988). Isozyme genomic maps have been established for several plant species, including wheat (Saha and Stelly, 1994). DNA fingerprinting involves the display of a set of DNA fragments from a specific DNA sample (Vos *et al*. 1995), to study polymorphism at DNA level that can be an indicative of genetic diversity. Plant DNA fingerprinting is also defined as the application of molecular marker techniques to identify cultivars, measure genetic diversity, select parents and warrant marker assisted selection.

2.3.2. Linkage Analysis and construction of genetic linkage map

This section aims to summarize the requirements of genetic linkage analysis, and to give a theoretical background on it and on the methods used to construct linkage maps. Linkage maps display the linear order and the relative distances between loci.

2.3.2.1. Principles of linkage analysis by means of meiotic recombination

When ordinary somatic cells divide, the cell undergoes a procedure named mitosis. During that process two daughter cells are produced that have identical genetic material. However, during the production of gametes, the cell divides according to a different biological pathway called meiosis, that after completion are formed four gametes which are haploid and carry a random set of one copy of each chromosome. In the course of meiosis, homologous chromosomes will pair, form chiasmata and exchange genetic material under the process known as recombination or crossing-over (Figure 2.2)

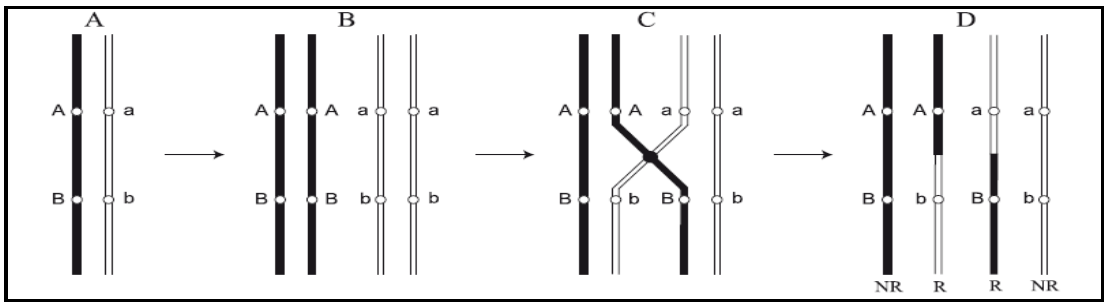

Figure 2.2. Schematic drawing of the process of crossing-over between loci A and B. (A) Two homologous parental chromosomes, one that carries alleles 'A' and 'B', and another that carries alleles 'a' and 'b'. (B) Chromosomes goes through replication, generating two pairs of identical sister chromatids. (C-D) Cross-over occurs between two of the chromatids resulting in two recombinant (R) and two non-recombinant (NR) chromatids (figure adapted from Wahlberg, 2009).

The purpose of linkage analysis is to determine whether or not pairs of genetic markers show independent assortment. To illustrate genetic linkage, let us consider the formation of gametes from an individual that is a double heterozygote (*Aa/Bb)* for two loci (Table 2.1). If the two loci in this example are located on different chromosomes, or far away on the same chromosome, they will segregate independently during meiosis, and thus, there is a 50 % probability that alleles at different loci will be inherited together. The independent segregation of alleles from the two loci would lead to the formation of four different gametes in equal proportions (i.e. *AB, Ab, aB* and *ab*). In contrast, if two loci are located on the same chromosome they tend to show genetic linkage and the proportions of gametes will be distorted and not produced in equal numbers. In this case we have to consider that two alternative *linkage phases* exist; either that allele *A* is coupled to *B* or alternatively *A* could be coupled to *b*. In Table 2.1, we see that the majority of gametes correspond to either 'AB' or 'ab'. Those represent the parental or non-recombinant haplotypes (NR).

Table 2.1. Segregation of alleles in a double heterozygote individual.

Genotypes	AaBb			
Gametes	AB	Ab	aB	ab
Independenat segregation (%)	25	25	25	25
Incomplete Linkage (%)	35	15	15	35
Complete Linkage	50	0	0	50

This Table is adapted from Wahlberg (2009)

In practice, recombination between homologous chromosomes means that the physical linkage between marker alleles on the same chromosome will occasionally be broken up by recombination events and that the linkage between two alleles will be incomplete. The more distant two markers are on a chromosome, the more likely it is that recombination events will break up the linkage between the markers.

As discussed above, recombination between the maternal and paternal chromosomes occurs during meiosis, whereby homologous chromosomes are paired and genetic material is exchanged. At a genomic level the exchange of genetic material between chromosomes produces diversification by shuffling allelic variants, and hence results in novel haplotype combinations that are transmitted to the next generation. Therefore, recombination has considerable influence on the genome diversity and on evolution. At a molecular level, the process of recombination generates a temporary connection between chromosomes (chiasma).

Several approaches have been used to study the frequency and the chromosomal distribution of meiotic cross-over events. Historically, recombination was studied directly by observing chiasmata in cells going through meiotic division using cytogenetic methods. The cytogenetic approach can be used to examine the total number of cross-over events per cell as well as the number or events per chromosome. Whilst this method can reveal general patterns, it fails to provide a detailed view of the distribution of recombination events. Indirectly, recombination can be monitored by the construction of genetic maps to estimate the frequency of recombination between pairs of loci. The resolution of a linkage map is dependent on the marker density and the number of observed meiosis. Fine-scale patterns of

recombination can be studied by observing the degree of linkage disequilibrium (LD) between marker loci in natural populations

2.3.2.2. Mapping Population

In early 20th century, it was demonstrated by Johannsen that quantitative variation results from the combined effects of multiple segregating genes and environmental factors. The parents selected for mapping population should be sufficiently polymorphic at the DNA level so that the recombination events throughout the genome could be detected. There are several types of breeding schemes that can be used to produce a mapping population for QTL analysis or linkage map construction, each having its own advantages and disadvantages. Mostly used populations for genetic mapping or QTL studies are;

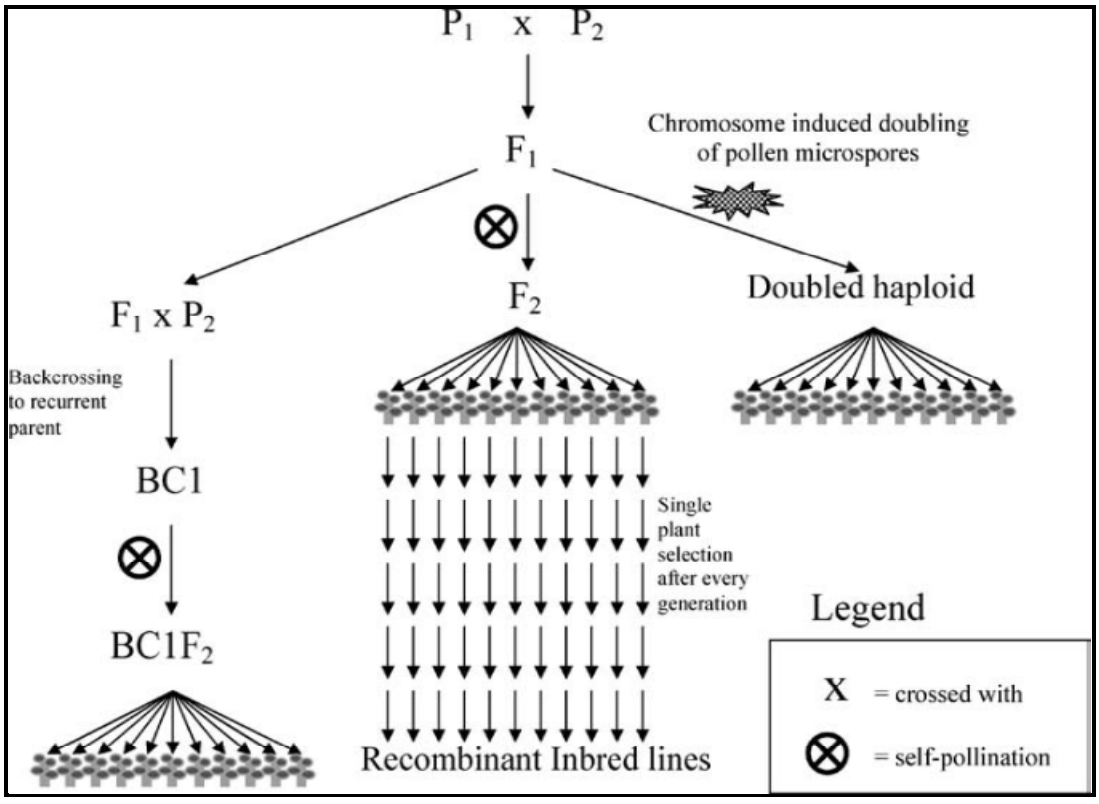

Fig. 2.3 Diagram of main types of mapping populations for self-pollinating species (Picture is taken from Collard et al. 2005)

F_2 segregating population, backcross population, recombinant inbred line (RIL) population, doubled haploid (DH) population (Fig 2.3). F2 and backcross population can be developed by selfing the F1 hybrids, and backcrossing them to one of the parents. The advantages of F_2 and backcross populations are that they are easy to develop, and require short time to produce, although sterility in F1 hybrid may limit some combinations of parents particularly in wide crosses. A F_2 is better than backcross, since QTL with recessive alleles in recurrent parent cannot be detected, when dominance is present (Asin 2002). Major inconveniences of F2 and backcross population is that, these cannot breed true and cannot be replicated (evaluation several times, different environmental conditions, different years and locations etc.), and more crossing is needed to produce sufficient seeds that is difficult and labor intensive. Recombinant inbred lines (RIL) can be produced by single seed descent method through selfing the F_2 progeny, until 6 or more generations. Recombinant inbred lines contain a series of predominant homozygous lines, and due to several meiosis process, each RIL contains a unique combination of different linkage blocks from the original parents. However to produce RIL takes long time and 100% homozygosity cannot be achieved even selfing until 6 or more years. One preferred mapping population used by researchers is doubled haploids. Doubled haploid population in wheat can be derived from F1 generation through anther culture, microspore culture, and hybridization of wheat with maize or pearl millet followed by chromosomes elimination technique, and later doubling the chromosomes by colchicine application. DHs are hundred percent homozygous and require short time to be produce. The main drawback of DH population largely depends upon genotypic response to tissue culture techniques. However, RIL and DH population are considered as a powerful over F2 or back cross population, where only fewer individuals can be screened (and this economically very important while using molecular markers) and can be maintained permanently and evaluated in repeated trials across different years and locations and having a large potential for identification of reliable QTL. Some other populations used for linkage map construction or QTL analysis are advanced backcross QTL population, single chromosomes recombinant lines (SCRL), and single chromosome recombinant-

double haploid lines (SCR-DH). Advanced backcross QTL (AB-QTL) was first time proposed by Tanksley and Nelson in 1996.

Once an appropriate population is chosen, then appropriate population size must be decided. In general large population is suggested, whenever possible. Population less than 50 individuals give poor map to be useful. For QTL mapping, much larger population will be required. Generally, population smaller than 200 individuals is rarely successful in finding QTL and in some cases more than 500 individuals is required (Young 1994). The limited population sizes used in many QTL detection experiments may have led to underestimation of QTL number, over estimation of QTL effects, and failure to quantify QTL interactions (Beavis 1998; Melchinger et al. 1998). The number of lines used in many QTL experiments has been about 100. Beavis (1998) suggested that even 200 individuals may be too few for reliable QTL detection, and addressed issues related to the choice of population size in QTL mapping experiments. In most of the QTL experiments, size of populations is limited because of the cost of marker genotyping and phenotyping traits. If marker genotyping is expensive, then selective genotyping can be used rather to exploit whole population, in which entire population is phenotyped for the traits of interest and lines from extreme tails are selected for marker genotyping.

After the mapping population is created, DNA is extracted from each line in the population and analyzed with molecular markers. The markers used for mapping population must first be tested on the parental lines to confirm that they polymorphic. The genotyping of the whole set of population allow researchers to determine, if each individual is genetically similar to parent A or parent B for specific marker, allowing the segregation to be determined and recombinant events to be inferred.

For QTL analysis, parents and whole set of the population should be screened for morphological traits. For phenotypic evaluation, data should be recorded under multi locations and years. For example, for F_2 or backcross population, morphological characters must be recorded minimum two different locations and repeated at least for two years. If there exist a significant difference between population and parents for particular traits of interest, then they are subjected to identify by DNA markers that reveal difference between parents. It is extremely

crucial that there must be sufficient genetic diversity between parents in order to construct genetic linkage maps or QTL analysis.

2.3.2.3. Molecular markers

The term “molecular markers” is applied to a variety of DNA fingerprinting techniques that assay variations at the DNA level. Ideal markers are stable, abundant and detectable in plant tissues regardless of growth, differentiation and defense status. A number of DNA fingerprinting techniques are available (Karp and Edwards 1997), most of which use polymerase chain reaction (PCR) (Vos *et al.* 1995) for detection of polymorphism, mapping and QTL analysis (Young, 1999). Development of the first map of human genome based on molecular markers (Botstein *et al.* 1980) fueled the development of maps in other genomes.

Molecular markers are powerful tools that can be used for marker-assisted selection and as landmarks for map-based cloning of resistance genes. Any unique DNA sequence can be used in DNA hybridization, PCR or restriction mapping experiments to identify that sequence. They occupy specific position on chromosomes like a gene and reveal site of variation in DNA. They arise from different classes of DNA mutations such as substitution mutation (point mutation), rearrangement mutation (insertion or deletion) or error in replication of tandemly repeated DNA (Paterson, 1996). The first available molecular markers were biochemical markers, which include allelic variants of enzymes called isozymes, protein variants detected by difference in migration on electrophoresis and specific staining. Isozymes markers were extensively used in genetic diversity studies in different crop species including wheat, but for mapping studies, they are insufficient in number. The main drawback of the biochemical markers is that they are limited in numbers, affected by environmental conditions and developmental stage of the plants. In comparison, DNA markers are useful complements to the morphological and physiological characterization of cultivars because they are plentiful, are not influenced by plant tissue or environmental effects, and allow cultivar identification very early in plant development (Manifesto et al. 2001).

In the recent decades, use of molecular markers has become routine in studying genetic diversity, population genetics, understanding evolutionary mechanism among crop species, linkage maps and QTL identification, discovery of genes, and thereby resulted in revolutionization of molecular biology. A number of different molecular markers have been developed to be used in various plant species; however some markers may not be suitable for one purpose but may be not suited for other purpose. Unlike morphological and biochemical markers they are unlimited in numbers. Molecular markers has been classified by Weising et al (1995) as

(1) *Hybridization based*: Classical hybridization based fingerprinting involves the cutting of genomic DNA with restriction enzymes, electrophoretic separation of the resulting DNA fragments according to size and detection of the polymorphic multilocus of the banding patterns by hybridization with labeled complementary DNA sequences, a so called 'probe'.

(2) PCR based markers involve intro amplification of particular DNA sequences with the help of specific primers and thermo stable DNA polymerase, electrophoretic separation of amplified fragments and detection of polymorphic banding patterns by staining with ethidium bromide.

(3) DNA sequence based such as single neocleotide polymorphism (SNP) markers.

DNA based molecular markers have been divided on the basic principal; one which can distinguished between heterozygote and homozygote plant, termed as co-dominant marker, whereas dominant markers indicated as present and absent and cannot differentiate between heterozygote and homozygote plant.

Variety of molecular techniques have been emerged to study the DNA polymorphism for selection of desired parents for improvement of cultivars through breeding and also vastly used in the studying the genetic relationship among species of different crops. Most commonly used markers used in different plant species are; random fragment length fragment polymorphism (RFLP), random amplified polymorphic DNA (RAPD), simple sequence repeats (SSR), inter simple sequence repeat (ISSR), sequence related amplified polymorphism (SRAP), amplified fragment length polymorphism (AFLP) etc.

2.3.2.3.(1).AFLP- marker

A novel DNA fingerprinting technique, called amplified fragment length polymorphism (AFLP^{TM}), has been developed by Zebeau and Vos (1993). The AFLP technology is trade marked by keygene (www.keygene.com). According to Vos et al., 1995, the AFLP technique is based on the amplification of subsets of genomic restriction fragments using PCR. DNA is cut with restriction enzymes, and double-stranded (ds) adapters are ligated to the ends of the DNA-fragments to generate template DNA for amplification. The sequence of the adapters and the adjacent restriction site serve as primer binding sites for subsequent amplification of the restriction fragments. Selective nucleotides are included at the 3' ends of the PCR primers, which therefore can only prime DNA synthesis from a subset of the restriction sites. Only restriction fragments in which the nucleotides flanking the restriction site match the selective nucleotides will be amplified. Using this method, sets of restriction fragments may be visualized by PCR without knowledge of nucleotide sequence. The method allows the specific co-amplification of high numbers of restriction fragments. The number of fragments that can be analyzed simultaneously, however, is dependent on the resolution of the detection system. Typically 50-100 restriction fragments are amplified and detected on denaturing polyacrylamide gels. AFLP are Mendelian markers having a number of appealing features. Thus they provide a novel and powerful tool for DNA fingerprinting of genome of any origin or complexity (Ajmone et al., 1998). However, according to comparative studies on soybean and maize, AFLP is the most efficient marker system with a 10-fold higher efficiency in the time, cost and replicability and resolution, when compared to RFLP, RAPD and SSR (Powell et al., 1996). AFLPs combine the merits of both RFLP and PCR-based multilocus markers (Bai et al., 1999). AFLP can detect multiple loci and are dominant inherited as compared to RFLP, which is a codominant in nature. However, AFLP is a complicated time taking technology, require large amount of DNA, with more labor (Vos et al., 1995). The level of polymorphism is species specific.

The first simplified AFLP method was established by Suazo and Hall (1999) on honey bee (*Apis mellifera* L.). Later on AFLP analysis has been conducted in number of crop plants such as bean (Tohme et al., 1996), rice (Virk et al., 2000), tea (Paul et al., 1997), maize (Ajmone et al., 1998), , soyabean (Maughan et al., 1996), including bread wheat (Liu et al., 2007).

2.3.2.4.(2). Microsattelite markers

Molecular marker technology has gained momentum with the introduction of microsatellites during 1990s (Somer et al. 2004), since the first microsatellites has been described for hexaploid wheat (Devos et al., 1995; Röder et al., 1998). A genome of all eukaryote contains a class of sequence called as microsatellites or simple tandem repeats (STRs) or simple sequence repeats (SSRs) etc. Microsatellites are the stretches of DNA consisting of tandemly repeated short units of 1-6 base pairs in length (Comertpay et al. 2012). In most cases, microsatellite containing dinucleotide, trinucleotide motifs are used for marker development. Microsatellites are abundant and widely dispersed through eukaryotic genomes and has a number of appealing features, such as, they are PCR based (which is much easier to perform than RFLP analysis and is highly amenable to automation), locus specific (which is extremely important in polyploidy species such as wheat) and are typically co-dominant in nature. They detect high polymorphism (particularly in self-pollinated species like wheat, where most of the markers show low polymorphism,) even in closely related germplasm, simple screening requirements and reproducibility ensures that microsatellites are best suited markers system in wheat breeding (Rossetto 2001). However, due to the large genome size, the development of microsatellite markers in wheat is extremely time-consuming and expensive. Another disadvantage of SSRs is the reduced amount of information generated from each amplification reaction and each gel lane. SSRs markers developed for one species are generally of limited utility in other more distantly related species, however is usually successful in closely related species such wheat and its wild relatives. Only 30% of all primer pairs developed from microsatellite sequences is functional and suitable

for genetic analysis. Besides of above mentioned drawbacks, SSRs have received a considerable attention and are probably the current markers system of choice for marker based genetic analysis and marker assisted wheat breeding.

Currently, mapping of approximately 2.000 to 2500 SSR markers on 21 wheat chromosomes has been published in a variety of publications (Ganal and Röder 2007). These are ideal markers for identification of varieties, analysis of germplasm collection and analysis of genetic relationship, construction of genetic linkage maps and QTL analysis in wheat. Microsatellite markers detect a high level of variability. Microsatellites have been extensively used in cereals including wheat in constructing molecular maps and QTL analysis for quality traits and agronomic characters (Prasad et al., 1999; Campbell et al., 2001; Börner et al., 2002; Huang et al., 2004; Nelson et al., 2006; Kunert et al., 2007).

2.3.2.4. (3). Diversity array technology (DArT)

DArT is a complexity reduction, microarray DNA hybridization-based technique that simultaneously assays hundreds to thousands of markers across a genome. Details of the methodology for DArT were first described by Jaccoud et al. (2001). For each individual DNA sample being typed, genomic representations are prepared by restriction enzyme (e.g. PstI and TaqI) digestion of genomic DNA followed by ligation of restriction fragments to adapters. The genome complexity is then reduced by PCR using primers with complementary sequences to the adapter and selective overhangs. The fragments from representations are cloned, and cloned inserts are amplified using vector-specific primers, purified and arrayed onto a solid support (microarray) resulting in a “discovery array.” Labeled genomic representations prepared from the individual genomes included in the pool are hybridized to the discovery array (Jaccoud et al., 2001). Polymorphic clones (DArT markers) show variable hybridization signal intensities for different individuals. These clones are subsequently assembled into a “genotyping array” for routine genotyping (Semagn et al. 2006).

Diversity Arrays Technology (DArT) is one of the recently developed molecular techniques and it has only been used in rice (Jaccoud et al., 2001), barley (Wenzl et al., 2004), eucalyptus (Lezar et al., 2004), Arabidopsis (Wittenberg et al., 2005), cassava (Xia et al., 2005), wheat (Akbari et al., 2006; Semagn et al., 2006), and pigeon-pea (Yang et al., 2006). DArT provides a promising alternative to satisfy the requirements of throughout, genome coverage, with higher reproducibility and transferability (Jaccoud et al. 2001). This technique doesn't need prior sequence information for the species to be studied. DArT preferentially targets low-copy genomic regions, allows automation of data acquisition and is cost competitive. However there are some drawbacks such as DArT are dominant markers and is a microarray-based technique that involves several steps, including preparation of genomic representation for the target species, cloning, and data management and analysis. The latter requires dedicated softwares such as DArT soft and DArTdb. The establishment of DArT system, therefore, is highly likely to demand an extensive investment both in laboratory facility and skilled manpower (Semagn et al. 2006). Although developed some years ago, this marker technology has recently gained increasing attention (Wenzl et al. 2004; Semagn et al. 2006). This technology can genotype agricultural crops with varying degree of efficiency, but they several limitations associated with their capability to quickly developed large number of markers. DArT has recently been used in genetic mapping and fingerprinting studies in cereals such as rice (Jaccoud et al. 2001), and later applied in other crops like barley (Wenzl et al. 2004), and most recently in wheat (Akbari et al. 2006; Semagn et al. 2006; Peleg et al. 2008).

Advanced high-density genetic linkage maps established using molecular markers for a series of economically important crops provide a basis for MAS of agronomically useful traits, for pyramiding of resistance genes and the isolation of important genes by map-based cloning strategies (Tanksley ***et al.,*** 1995). Once the molecular markers closely linked to desirable traits are identified, MAS can be performed in early segregating populations and at early stages of plant development (Zhang ***et al.,*** 2004).

2.3.3. Genetic Linkage maps of wheat

Construction of linkage maps with molecular markers is a key step in the linkage analysis of biologically or agronomically important traits. Linkage maps may be thought of as a 'road map' of chromosomes derived from two different parents (Paterson, 1996). Linkage maps indicate position and relative genetic distance between markers along chromosomes, which is analogous to sign or landmarks along a high way. Linkage maps have been utilized for identifying chromosomal region that contain genes controlling simple traits (controlled by single gene) and quantitative traits using QTL analysis (Mohan et al., 1997). Dense genetic maps of cereals contribute substantially to positional cloning of important genes and provide a tool for evolutionary studies, as well as characterization of germplasm and gene discovery. Still molecular mapping in wheat lagged behind many other crops due to its large genome and low level of polymorphism.

Several maps have been developed for hexaploid wheat, durum wheat as well as wild relative of wheat such as Einkorn wheat, *Triticum dicoccoides*, *Aegilops tauchii* . In bread wheat, a linkage map based on intervarietal cross may be more useful than an inter-specific cross in detecting agriculturally important genes such as grain yield and abiotic stress tolerance and QTLs for the grain quality characteristics were also determined. Some of the most updated and recent genetic linkage maps of diploid, tetraploid and hexaploid wheat have been briefly described in tabulated form (Table 2.2).

Table-2.2. A list of most up-to-date molecular maps developed in wheat.

Class of wheat	Population used for mapping	Type of Marker used	No. of loci mapped	Genetic map length (cM)	Reference
Diploid wheat (D-genome)	F2 [*T. tauschii* (TA1691 var. meyeri x TA1704 var. typica)]		152	1554	Gill et al (1991)
Diploid wheat	F3 (*T.* monococcum ssp. Monococcum DV92 x *T.* monococcum ssp. Aegilopoides G3116).		335	1067	Dubcovsky et al (1995)
Diploid wheat (D-genome)	Meyeri [(TA1691 x *Ae. tauschii* var. typical (TA 1704)]		546	-	Boyko et al (1999)
Durum wheat	RILs (*T. durum* var. Messapia x *T. turgidium* var. MG4343)	RFLP, Glu3B,	213	1352	Blanco et al., (1998)
(Wild emmer X Durum wheat)	F2 (Hermon H52 x Langdon		573	3169 / 3179	Peng et al (2000)
Diploid wheat (A-genome)	RILs (*Triticum boeoticum pau5088 x T. monococcum pau14087)*	SSRs, EST, RFLP	176	1262	Singh et al (2007)
Einkorn wheat	F2s/F3s (*T. Monococcum* ssp . *monococcum DV92 x T. monococcum ssp. Aegilopoides C3116)*	*RFLP*	3335	714	Dubcovsky et al (1996)
Durum wheat	RILs (*T. durum* var. Messenia x *T. turgidium* var. MG4343)	AFLP, RFLP	88	2063	Lotti et al (2000)
	RILs (Kofa x UC1113) *	SSR, SNP	269	2140	Zhang et al (2008)
	RILs (Kofa x Svevo) *	SSR	232	2347	Maccaferri et al. (2008)
	RILs (Colosseo x Lloyd) *	SSR, DArT, SNP	657	2022	Maccaferri et al. (2008)

Class of wheat	Population used for mapping	Type of Marker used	No. of loci mapped	Genetic map length (cM)	Reference
	RILs (Meridiano x Claudio) *	SSR, DArT, SNP	999	2487	Mantovani et al. (2008)
	RILs (PDW 233 x Bhalegaon 4) *	SSRs, ISSR, TRAP, SSP, SCAR, STS	201	2296.8	Patil.,et al (2008)
	RILs (Jennah Khetifa x Cham1) *	RFLP, SSRs, AFLP	206	3598	Nachit et al (2001)
	RILs (Omrabi 5 x *T dicoccoides* 600545)	SSRs, AFLP	312	2289	Elouafi and Nachit (2004)
	RILs (Colosseo x Lloyd) & (Meridiano x Claudio)	SSRs,DArT, SNP	1479	2967	Trebbi et al (2011)
(Durum wheat X wild emmer)	RILs (Messapia X MG4343)	RFLPs,AFLP,SSR	458	3038	Blanco et al (2004)
(Durum wheat X Wild emmer)	RILs [(Desf.) MacKey X (Köm.)] *	SSRs DArT	152	2317	Peleg et al (2008)
Bread wheat	DHs (CM-82036 x Remus), etc.) *	RFLP, AFLP, SSRs	384	1860	Buerstmayr et al (2002)
	DHs (Savannah x Senat) *	SSRs, AFLP	345	2300	Eriksen et al (2003)
	ITMI RILs (W7984 x Opata85)		279	2162	Roder et al 1998
	F5*s*(Arina x Forno)	RFLP, SSRs	396	3085	Paillard et al (2003)
	DHs (Courtot x Chinese Spring)	RFLP, SSR, AFLP	659	3685	Sourdille et al (2003)
	RILs (Synthetic x Opata)		1235	2569	Somers et al (2004)
	RILs (Wangshuibai x Alondra's) *		250	2430	Zhang et al (2004)
	DHs (Frontana x Remus) *	SSR, STS, AFLP	535	2840	Steiner et al (2004)
	RILs (W7984 x Opata85)		1406	2654	Song et al (2005)

Class of wheat	Population used for mapping	Type of Marker used	No. of loci mapped	Genetic map length (cM)	Reference
	RILs (PI 531005 x BR34) *	TRAPs, SSRs	352	3045	Liu et al (2005)
	DHs (Spring x SQ1) *	RFLP, AFLP,SSR	567	3522	Quarrie et al (2005)
	RILs (Dream x Lynx) *	SSR, STS, AFLP	283	1734	Schmolke et al (2005)
	DHs (RL4452 x AC Domain) *	SSR, genes	369	2793	McCartney et al (2005)
	DHs (Fukuho-komugi x Oligoculm)*	SSR, RFLP, RAPD, ISSR	371	4190	Suenaga K., et al (2005)
	RILs (Ning7840 x Clark) *	SSR, AFLP	410	2,223	Marza et al (2005)
	DHs (Kitamoe x Munstertaler)		464	3441	Torada et al (2006)
	DHs (AC Karma × 87E03-S2B1) *	STS, SSR	167	2403	Huang et al (2006)
	DHs (Trident x Molineux) *	SSR, STS, RFLP	251	3061	Williams et al (2006)
	DHs (Cranbrook x Halberd)	SSR, RFLP, AFLP,DArT,STS	749	2937	Akbari et al (2006)
	DHs (Arina x BK93604	DarT, AFLP, SSR	624	2595.5	Semagn et al. (2006)
	RILs (*T. aestivum* L. var. Forno x *T. spelta* L. var. Oberkulmer)	RFLPs, SSRs	230	2469	Messmer et al (1999)
	RILs (Chuan-Mai18 x Vigour18) *		244	3150	Spielmeyer et al(2007)
	RILs (Renan x R´ecital) *	SSR, RFPLs AFLP	265	2722	Groos et al (2003 & 2007)
	DH (Arina x Riband) *	AFLP, SSR	279	1199	Draeger et al (2007)
	RILs (Chuan 35050 x Shannong 483) *	SSR,EST-SSR,ISSR, SRAP,TRAP,Glu loci	381	3636	Li et al (2007)

Class of wheat	Population used for mapping	Type of Marker used	No. of loci mapped	Genetic map length (cM)	Reference
	TA415260 (SHW) X North Dakota (DH)*	SSR,TRAP,RFLP	632	3811,5	Chu et al., 2008
	RILs (Nanda2419 x Wangshuibai)	SSR, EST, RAPD	887	4,223.10	Xue et al (2008)
	RILs (Heshangmai x Yu8679) *	SSR, EST	175	1584.6	Wang et al (2009)
	RILs (Neixiang 188 x Yanzhan 1) *	SSR, SSP	255	3324	Li et al. (2009)
	RILs (Halberd x Cutter) *		170	2256.6	Mason et al (2010)
	RILs (Halberd x Karl 92) *		193	2343.6	Mason et al (2010)
	DHs (Berkut x Krichau) *	SSR, DArT	547	-	Genc et al (2010)
	ILs (Am3 *Triticum carthlicumx* PS5 *Aegilops tauschii*)*	SSR,SSP	168	-	Li et al (2011)
	DHs (Avalon x Cadenza)*	SNP,EST	1054	2990	Allen et al (2011)
	RILs (UC1110 x PI610750) *	SSR,DArT,EST	558	3157	Lowe et al (2011)
	RIL (Ixos and X Primadur)	DArT, SSR, AFLP,ISBP	529	2082	Vaissayre et al. (2012)

* These are framework linkage map prepared for QTL analyses.

2.3.4. Quantitative trait loci (QTL)

Conventional plant breeding methods have made a significant contribution to crop improvement, but were slow in targeting complex traits like grain yield, grain quality and abiotic stress tolerance . In traditional plant breeding, breeders are facing with constraints during selection like;

(i) Large segregating population needs to be screened for a desirable trait e.g., yield and its components, cold tolerance, drought tolerance, disease resistance and quality traits.
(ii) Selection for quantitative traits will not be possible until F_6;
(iii) Environment-influenced traits are difficult to select;
(iv) Can't distinguish contrasting forms at seedling stage, making it necessary to grow population up to the adult stage
(v) It is difficult to undertake pyramiding of resistance genes, since selection of additional genes in presence of an existing resistance gene would be difficult (Duraa, 2009).

One method receiving growing attention is the mapping of chromosomal regions affecting qualitative or quantitative traits through molecular mapping and genomics approaches which offer new opportunities and strategies to dissect major genes and quantitative trait loci (QTL) underlying different interested traits such as quality parameters and abiotic stress tolerance etc (Duraa, 2009). Polygenic characters, which were very difficult to analyze using traditional plant breeding methods, can now be tagged using DNA molecular markers. Molecular markers allow geneticists and plant breeders to locate and follow numerous interacting genes that determine a complex trait. Genetic linkage maps can provide a more direct method for selecting desirable genes via their linkage to easily detectable molecular markers (Tanksley et al., 1989).

Quantitative characters have been major area of study in generics for over a century, as they are a common feature of natural variation in population of all eukaryotes, including crop plants. Traditional genetic studies have concentrated on dichotomous traits such as the presence or absence of a disease resistance in plant.

Such traits are often the result of a mutation at a single gene (Broman, 2009). For most of the period up to 1980, the study of quatitative traits has involved statistical techniques based on mean, variances and covariances of relatives. These studies provide a conceptual base for partinining the total phenotypic variation into genetic and environmental variances, and further analyzing the genetic variances in term of additive, dominance and epistatic effect. From this information it became possible to estimate the heritability of traits and predict the response of trait to selection. It was also possible to estimate the minimum number of genes involved in controlling the trait. However little was known about what these genes, where they are located, and how they control the trait, and there were such segregating in a Mendelian fashion in any given population, and most the cases their effect were additive.

Nilsson-Ehle about 100 years ago assumed that phenotypic variation is due to the combined effect of multiple segregating genes, all of them have a minute but additive effect together with joint action of the environment. It has been surmised that all allelic differences for these genes are small. This is because that all alleles are thought to be different in their efficiencies in contribution to the questioned trait. The genes responsible for these traits were first time called as polygenes by Mather in 1941, and laterally became popular by the term ‘quantitative trait loci (QTL) by Gelderman in 1975. Inheritance of quantitative traits has been subject of numerous investigations, but plant breeders had very little information regarding the number of genes and their location on chromosomes, and relative contribution of each to the final trait expression (Stuber, 1992). Geneticists have always tried to find the mystery of association between phenotype and genotype of an organism, which can provide specific and easily recognizable information about the genetic control of an economically important trait. Basic idea behind QTL mapping is just mapping genes controlling morphological traits that show simple Mendelian segregation, as in classical linkage studies. In 1923 Sax was first one using pigment markers in beans to analyze genes affecting seed size by investigating the segregation ratio of F_2 progeny of different crosses

The number of genes and genetic bases of the variation observed make manipulations and improvements of such traits slow and difficult (Shoemaker et al., 1995).The discovery of biochemical markers increased the use of isozymes to locate QTL but these were not available in sufficient number for thorough scan of entire genome. With the advent of molecular biology in the late 1980s, it is now possible to use molecular marker information to map major quantitative trait loci (QTLs) on chromosomes (Zeng, 1993).

Gene tagging is to locate the genes of interest with the help of linked molecular markers. DNA markers linked to a gene of interest are the milestones. These tags are useful starters for identifying the gene. Chromosome walking to or landing on the site where the gene can be physically found requires information on genome composition and marker orientations. Although cotton has a large and complex genome, however, the physical size in centiMorgan (cM) of cotton does not prohibit map-based cloning. It is estimated that the tetraploid cotton has a genome length of at least 5000 cM, and a physical size of 2246 Mb to 2702 Mb and hence 450 Kb/cM (Arumuganthan and Earle, 1991).

It is safe to say that molecular marker techniques will gain more and more influence on plant breeding in the future and will speed up breeding processes considerably. In view of potential development of new strategies, the future for improvement of polygenic traits through DNA markers appears bright. Moreover, by adopting new and novel marker systems like EST-SSRs, SNPs, DNA chips and microarrays, indeed, some day it may be possible to select best lines for breeding based on RNA expression profiles as much as marker genotypes. Integrating genomics and bioinformatics into the field of molecular breeding may prove to be even more significant than DNA markers and eventually lead to even more profound revolution in crop breeding.

Statistical task of the QTL mapping analysis is essentially a bridge between the trait phenotype and the genotype at the genomic region specified by marker loci. Once genetic linkage map has been constructed, the chromosomal location of the genetic factor(s) controlling the trait of interest can be located on map. A number of methods have been developed to detect and characterize QTL. These include Single

marker mapping (Edward et al., 1989), simple interval mapping (Lander and Botstein 1989), and composite interval mapping (CIM) (Zeng 1993), plus multiple trait mapping (Jiang and Zeng 1995).

2.3.4.1. Single interval mapping

A much better approach for mapping of QTL than the single marker associations discussed above will use information from two linked markers preferably on each side of the QTL (i.e., flanking markers). This is the widely applied method for QTL mapping, first described by Lander and Botstein in 1989 and described in more detail by Van Ooijin (1992), has now become current standard method used successfully by many geneticists for mapping QTL. They used one marker interval at a time to construct a putative QTL for testing by performing a likelihood ratio test (LRT) at every position in the interval. With a fine scale genetic marker map throughout the genome, IM can be performed at any position covered by markers to produce a continuous LRT statistical profile along chromosomes. The position with largest LRT statistics in chromosomes regions is an estimate of QTL position (Kao et al., 1999). Compared with the traditional method (Soller *et al.*, 1976), the interval mapping method has a number of advantages such as IM has more power and require fewer progeny. But it still has several problems; it does not allow precise detection of nearby QTL on same chromosomes. When there are two or more QTLs located on a chromosome, the mapping of QTLs can be seriously biased, and QTLs can be mapped to wrong positions (Haley and Knott., 1992). LOD (logarithm of the odd) score is the log of ratio between null hypothesis (no QTL) and alternative hypothesis (QTL being tested). Large LOD score correspond to greater evidence for presence of a QTL. Different wheat researchers use simple interval mapping for QTL detection by using computer programs eg. MQTL, MQTL (Kato et al., 1999, 2000), MAPMAKER/QTL 1.1, QGENE (Narasimhamoorthy et al., 2006).

2.3.4.2.Composite interval mapping

In 1993, Jansen proposed a combination of interval mapping and regression on markers, which would allow a more precise detection of multiple QTLs. The approach of the CIM is that when testing for putative QTL in an interval, one use other markers as a covariates to control for other QTLs and to reduce the residual variance such that the test can be improved. The main advantage of CIM is that it is more precise and effective at mapping QTLs compared to single marker analysis and interval mapping, especially when linked QTL involved (Collard et al., 2005). Different software for constructing composite interval mapping are available on internet like QTL Cartographer, MQTL, MapManager QT, QTLMaper 2.0, MAPMARKER, PLABQTL WinCartographer V2.5 software, MAP QTL etc.

2.3.5. Mapping agronomic traits in wheat

For the past two decades an intensive amount of molecular research has been conducted wherein chromosome specific DNA sequences or markers have been used to identify genes controlling traits of economic importance in wheat. With the incorporation of marker technology in the breeding programs worldwide, QTL analysis has been used to detect yield and yield related traits. There is need to improve agronomic traits that affects yield and yield components in wheat. Many agronomic traits are correlated with each other. Agronomic traits are among the most important and least understood traits of wheat. Understanding the genetic control of these traits is crucial for the sustained improvement of wheat (McCartney et al., 2005). Grain yield, time to maturity, lodging resistance, plant height and tiller numbers per plant are important agronomic traits of wheat. Among these characters yield is of primary importance in wheat breeding programe. Grain yield in wheat is very complex character and generally controlled by a number of quantitative trait loci (QTLs) and is influenced by environmental factors, and are characterized by low heritabilities, thus making it difficult to manipulate and improve breeding programs (Kato et al., 2000). Yield can be dissected into a number of components such as

number of kernels per spike (KPS), number of spikes per square meter (KPSM), and kernel weight (kw) are considered the major economic traits for wheat improvement (Brancourt-Hulmel et al., 2003; Donmez et al., 2001). These component traits are also under QTL control and the effects of individual QTLs on phenotypic variation are relatively small (Yano and Sasaki 1997). Therefore, while looking for QTLs controlling grain yield, QTLs for yield components should also be determined to provide more useful information. The genetic architecture of a quantitatively inherited trait such as grain yield is typically complex, affected by multiple interacting genes, environments, and the interaction between genes and environments that contribute to the observed phenotypic variation (Falconer and Mackay, 1996). Molecular genetic studies to determine these effects may increase the efficiency of wheat breeding for improved agronomic and morphological traits with high yield. QTL analysis has provided an effective approach to dissect complicated quantitative traits into component loci to study their relative effects on a specific trait (Doerge 2002). In hexaploid wheat (*Triticum aestivum* L.), however, QTL analysis of grain yield and its components using molecular marker systems are more complex than for diploids like barley and rice, and hence are currently limited (Hyne et al. 1994; Araki et al. 1999). Many QTL affecting yield and yield components has been mapped in different studies (Bezant et al., 1997; Kato et al., 2000; Kuchel et al., 2006). As summarized in Table 2.3, a large number of studies have been reported QTLs for yield and yield components in wheat in different environments using different population

Table 2.4. Most of the quantitative trait loci (QTL) for different agronomic traits published previously by different researchers

Trait	Population	Location of QTL	No. of line	No. of env	Reference
Grain Yield	Chinese Spring x Kanto107 (RIL)	4A	98	2	Araki et al., 1999
	Wichita x Cheyenne (RIL)	3A	98	7	Campbell et al., 2003
	Dharwar Dry x Sitta (RIL)	4AL	127	4	Kirigwi et al. 2007
	Milan x Catbird (DH)	4B,4D	98	2	Verma et al. 2005
	RL4452 × 'AC Domain (DH)	2A,2B3D4A,4D	182	9	McCartney ET AL. 2005
	Superb x BW278 (DH)	1A, 2D, 3B, 5A	178	12	Cuthbert et al., 2008
	Récital x Renan (RIL)	2B, 3B, 4A, 4B, 5A, 5B, 7D	194	6	Groos et al., 2003
	Flair X xx86 (shw)	1A,3D4D,5A,5B,6B,6D	111	6	Huang et al., 2004
	Prinz x W-7984 (AB)	1B, 2A, 2D, 5B	72	4	Huang et al., 2003
	ACKarma X 87E03-S2B1 (DH)	5A, 7A, 7B	185	3	Huang et al., 2006
	Trident x Molineux (DH)	1B, 2D, 3D, 4D, 6A, 6D	182	18	Kuchel et al., 2007
	WL711x PH132 (RIL)	1DL, 2DL, 3BL, 4AS, 4DL, 7AS, 7AS	100	6	Kumar et al., 2007a
	Opata85 x W7984 (RIL)	1AL, 2AS, 2DS, 4BL, 6DL	110	6	Kumar et al., 2007b
	Chuang35050 x Shannong483 (RIL)	1D, 2D, 3B, 6A	131	6	Li et al., 2007
	Kofa x Svevo (RIL)	2B, 3B, 7B	249	16	Maccaferi et al.,2008
	Karl92 x TA4152-4 (AB)	2D,7D	190	2	Narasimhamoorthy et al.2006

Trait	Population	Location of QTL	No. of line	No. of env	Reference
	Sunco × Tasman (DH)	2B, 4D		4	Mares and Campbell, 2001
	Ning7840 x Clark (RIL)	1AL, 1B, 2BL, 4AL, 4B, 5A, 5B, 6B, 7A, 7DL	132	5	Marza et al., 2006
	RL4452 × AC Domain (DH)	2A, 2B, 3D, 4A, 4D	182	8	McCartney et al., 2005
	SeriM82 x Babax (RIL)	6D, 7A	194	8	McIntyre et al., 2010
	Chinese Spring X SQ1 (DH)	1AS, 1BL, 2BS, , 4AS, 4AL, 4BS, 4BL, 4DL,5AL, 5BS, 5BL, 5DS, 5DL,6BL, 7AL, 7BS, 7BL	96	24	Quarrie et al., 2005
Plant height	Opata 85 x W7984 (RIL)	1AS, 2DS, 4AL, 6A	144	3	Borner et al., 2002
	Milan x Catbird (DH)	3D, 4B and 4D	98	2	Verma et al. 2005
	Courtot x Chinese Spring (DH)	3D, 4B, 4D, 5A, 5B, 6B, 6D, 7A, 7B	275	3	Cadalen et al., 1997
	Wichita x Cheyenne (RIL)	3A	98	7	Campbell et al., 2003
	RL4452 × 'AC Domain (DH)	2D,4B,4D,5B,7A,7B	182	9	McCartney et al. 2005
	TA415260 (SHW) X North Dakota (DH)	4D,5A	120		Chu et al., 2008
	ACKarma X 87E03-S2B1 (DH)	4BL, 4DS, 5DL, 7BS	185	3	Huang et al., 2006
	ND3338 x F390 (RIL)	1B, 4B, 6A , 6D, 7A	240	2	Liu et al., 2002
	TA4152-60 x ND495 (DH)	4DS, 5AL	120	4	Chu et al., 2008
	CA9613 x H1488 (DH)	1A, 1D, 2A, 2B, 3D, 4A, 6A, 7D	108	4	Hai et al., 2008
	Flair X xx86 (shw)	11A,1D,3A,3B,4A,4B,5A,5B,6A,6D,7A,7D	111	6	Huang et al., 2004

Trait	Population	Location of QTL	No. of line	No. of env	Reference
	Prinz x W-7984 (AB)	2B,4B,4D,6A,7B	72	4	Huang et al., 2003
	Kofa x Svevo (RIL)	1BS, 2BL, 3AL, 3BS, 7BS	249	16	Maccaferri et al., 2008
	Ning7840 x Clark (RIL)	2B, 2D, 3B, 4B, 6A	132	3	Marza et al., 2006
	RL4452 × AC Domain (DH)	2D, 4B, 4D, 5B, 7A, 7B	182	8	McCartney et al., 2005
	SeriM82 x Babax (RIL)	1D, 2B, 4A, 4B, 5B, 5D	194	8	McIntyre et al., 2010
	Courtot x Chinese Spring (DH)	4BS, 4DL, 7AL, 7BL	217	5	Sourdille et al., 2003
	Chuan-Mai18 x Vigour18 (RIL)	2AS, 6AS	460	2	Spielmeyer et al., 2007
	Heshangmai x Yu8679 (RIL)	1D, 2D, 3D, 4D	142	4	Wang et al., 2009
Spikelet number	Milan x Catbird (DH)	2B,6A	98	2	Verma et al. 2005
	Chuang35050 x Shannong483 (RIL)	5D	131	6	Li et al., 2007
	Nanda2419 x Wangshuibai (RIL)	7D, 5A,1B,3B, 5B,2D,7A	136	2	Ma et al., 2007
	Agropyron cristatum (3228) X jing 4839 (F2:3 families)	6A,5B,1A,1B,3B,3D,4D	237		Wang et al. 2011
	TA4152-60 x ND495 (DH)	4DL	120	4	Chu et al., 2008
Spike density	Nanda2419 x Wangshuibai (RIL)	1A,4A,5A,5B,7D,	136	2	Ma et al., 2007
	Ning7840 x Clark (RIL)	1B,4A,7B,7D	132	3	Marza et al., 2006
	TA4152-60 x ND495 (DH)	5A,5B	120	4	Chu et al., 2008

Trait	Population	Location of QTL	No. of line	No. of env	Reference
Number of seeds	Milan x Catbird (DH)	1B,2B,6A	98	2	Verma et al. 2005
	Flair X xx86 (shw)	1D,2A,3D,6A,7A,7D	111	6	Huang et al., 2004
	Heshangmai x Yu8679 (RIL),	1D,2A,2D,3A,4D,6D	142	4	Wang et al., 2009
	Ning7840 x Clark (RIL)	1A,1B,2B,2D,3B,4B	132	3	Marza et al., 2006
	Chuang35050xShannong483	2A,3B,1D,2A,6A,6B	131	6	Li et al., 2007
	Karl92 x TA4152-4 (AB)	3D	190	2	Narasimhamoorthy et al.2006
	Agropyron cristatum (3228) X jing 4839 (F2:3families)	1A,4A,4B,5A,7A,3B,4B,7B	237		Wang et al. 2011
Day to heading	Chinese Spring x Kanto107 (RIL)	4A	98	2	Araki et al., 1999
	Norstar x Manitou (DH)	2B, 4A, 6A	142	1	Baga et al., 2009
	Opata 85 x W7984 (RIL)	2DS, 3AL, 5DS, 5DL, 7DS	114	11	Börner et al., 2002
	TA4152-60 x ND495 (DH)	5AL, 5BL	120	4	Chu et al., 2008
	Superb x BW278 (DH)	1B, 2D, 3A, 5A, 6B, 7B, 7D	178	12	Cuthbert et al., 2008
	TA415260 (SHW) X North Dakota (DH)				Chu et al., 2008
	Renan x Recital (RIL)	2B, 2D, 5A, 5B, 7A, 7D	194	3	Hanocq et al., 2004
	Prinz x W-7984 (AB)	2A, 2D, 3B, 5A, 5B, 6A 7B	72	4	Huang et al., 2003
	Trident x Molineux (DH)	1AL, 2AS, 6DS 7AS	182	18	Kuchel et al., 2006
	Flair X xx86 (shw)	2D,3A,4A,7A,7D	111	6	Huang et al., 2004

Trait	Population	Location of QTL	No. of line	No. of env	Reference
	Nanda2419xWangshuibai (RIL)	1B, 1D, 2B, 2D, 4A, 7B	136	5	Lin et al., 2008
	Kofa x Svevo (RIL)	2A, 2B, 7B	249	16	Maccaferri et al.,2008
	Ning7840 x Clark (RIL)	3BL, 5B, 6B	132	5	Marza et al., 2006
	SeriM82 x Babax (RIL)	1B, 1D, 4A, 5D	194	8	McIntyre et al., 2010
	Karl92 x TA4152-4 (AB)	2D, 3D	190	2	Narasimhamoorthy et al.2006
	WPI219 x Opata85 (RIL)	2D	114	5	Nelson et al., 2006
	Courtot x Chinese Spring (DH)	2BS, 5AL, 7BS, 7BL, 7DL	217	5	Sourdille et al., 2003
	Heshangmai x Yu8679 (RIL)	1B, 2B, 3B, 5D, 6D	142	4	Wang et al., 2009
	Huapei3 x Yumai57 (DH)	1B, 2B, 5D, 6D, 7A, 7D	168	3	Zhang et al., 2009

2.3.5.1. Days to heading

Earliness can be considered as an adaptation trait (Worland 1996). Its control has been one of the main explanations for the gradual extension of wheat cultivation (Law and Worland 1997). Nowadays, earliness remains a major factor of variation for agronomic traits, and one of the critical traits which must be considered when breeding a variety. For this reason, the modern methodologies used to investigate the genome, such as QTL and gene mapping, are useful tools for breeders. One of the main issues regarding these methodologies is to determine reliable markers that can be implemented in the context of marker-assisted selection (MAS) programs (Hanocq et al. 2007).

Common bread wheat is the most widely grown cereals in the World due to its ability to survive under many different environmental conditions. The plant life cycle includes three basic growth stages: germination, vegetative and reproductive growth stage. Flowering is the key component of the reproductive stage and is important for continual cropping and adaptation to target environment. The regulation of flowering time is most important criterion for adaptation to such a wide range of growing conditions. A heading time inappropriate for local environment may subject a crop at critical growth stages to the influences of extreme weather conditions such as frost, drought, heat stress, and result in significantly reduced grain yield. Growth and developmental phases (i.e. tillering, stem elongation, heading, anthesis and maturity) of wheat are primarily determined by vernalization genes ***(Vrn),*** genes that control the photoperiod response ***(Ppd)*** and earliness ***per se*** genes ***(Eps)*** (Dubcovsky et al. 1998). The adaptability of bread wheat to a wide range of environments has been favored by allelic diversity in genes regulating growth habit (*Vrn* genes) and photoperiod response (*Ppd* genes) (Distelfeld et al. 2009). Differences in *Vrn* genes divide wheat into spring, winter and facultative types (semi winter), whereas differences in the *Ppd* genes divide the germplasm into day length-sensitive and day length-insensitive classes. Increasing knowledge about the genetics of growth habit will contribute to a better understanding about the adaptation of wheat and promote their breeding for specific environments (Goncharov 2003). Now

a days earliness remain a major factor of variation in agronomic traits, and one of the critical trait that must be considered when breeding a variety. Therefore modern technologies such as QTL and genome mapping could be usefull for breeders to investigate genome,

Quantitative trait loci (QTLs) for wheat flowering time or heading date were reported in several mapping studies. In addition to the major QTLs that correspond to *Ppd* and *Vrn* genes on homologous group 2 and 5 chromosomes, QTLs with considerable effect in controlling earliness were mapped on the other chromosomes such as 4A and 4B.

Sourdille et al. (1999) mapped QTL for heading date and photperiod response in a doubled-haploid (DH) population derived from a cross between the wheat cultivars 'Courtot' and 'Chinese Spring'. For each trait, 2-4 QTLs were identified with individual effects ranging between 6.3% and 44.4% of the total phenotypic variation. Two QTLs affecting simultaneously heading time and photoperiod response were determined. One QTL located on chromosome arm 2BS near the locus *Xfbb121-2B*, co-segregated with the gene *Ppd-B1* known to be involved in photoperiod response. This chromosome region explained a large part of the variation (23.4–44.4% depending on the years or the traits). Another region located on chromosome arm 7BS between the loci *Xfbb324-7B* and *Xfbb53-7B* also had a strong effect (7.3–15.3%). This region may correspond to a QTL for earliness per se.

Zhang et al. (2009) studied QTL for heading date using a population of 168 doubled haploids derived from cross between two elite Chinese wheat cultivars Huapei 3 x Yumai 57. Two QTLs with main effect and epistatic effects of two pairs of genes were detected for heading date on chromosomes 1B, 2B, 5D, 6D, 7A, and 7D. A highly significant QTL, designated as Qhd5D, was observed within the Xbarc320-Xwmc215 interval on chromosome 5DL, accounting for 53.19% of the phenotypic variance and reducing days-to-heading by 2.77 days. The Qhd5D closely linked with a PCR marker Xwmc215 with the genetic distance 2.1 cM, located on the similar position of vernalization sensitivity gene Vrn-D1, can be used in molecular marker-assisted selection (MAS) in wheat breeding programs.

A recombinant inbred line (RIL) population derived from CI 13227 × Suwon 92 was employed to tag the quantitative trait locus (QTL) for early heading in Suwon 92. Interval analysis revealed a major QTL for heading date, designated as *QHd.pser-2DS*, between AFLP marker *XGCTG.CGCT118* and SSR marker *Xgwm261*. Based on the linkage map, *QHd.pser-2DS* was about 41.2 cM proximal to the distal end of chromosome 2DS, and explained 40.5% of the phenotypic variance across three years. The identified markers associated with the early heading QTL have the potential to be used in wheat breeding programs (Xu et al. 2005).

2.3.5.2. Plant height

Plant height is an important trait in wheat breeding because it is related with plant biomass and lodging resistance. Appropriate plant height is an important trait to achieve the desired level of grain yield in wheat. Tall wheat varieties are more sensitive to lodging, whereas semi dwarf wheat varieties have been credited with an important contribution to yield improvement, both because they permit a more efficient utilization of assimilate and reduce the extent of lodging-induced yield loss. In warm humid environments, taller plants tend to produce less leaf area, and therefore are less effective as photosynthesizers and assimilators (Ahmad and Sorrells, 2002). The various dwarfing genes are different in their effects on height, grain yield and other aspects of agronomic performance (Worland et al., 1998. Understanding of the genetic bases of plant height is useful in wheat improvement. In bread wheat (Triticum aestivum L.), at least 21 major genes dispersed over a number of chromosomes determine the overall height of plant.

A set of 142 recombinant inbred lines deriving from cross of Heshangmai X Yu8679 were tested under four different ecological environments during 2006 and 2007, and they were genotyped using SSR markers. Six QTLs for plant height were identified on chromosomes ID,2D,3D and 4D (Wang et al. 2009).

Maccaferri et al. (2008) evaluated a wheat population consisting of RILs under 16 different environmental conditions of Mediterranean ((10 rainfed and 6 irrigated locations) over two years (Table 2.3). They identified five major QTLs for

plant height on chromosomes 1BS in seven environments, 2BL in nine environments, 3AL in 10 environments, 3BS in 11 environments, and 7AS in 6 environments. The QTLs were inconsistent over environments, especially under different moisture levels.

In another study by Hai et al. (2008) a population of 108 DH lines were evaluated in four environments. They identified 10 QTLs on chromosomes 1A, 1D, 2A, 2B, 3D, 4A, 6A, and 7D. Only one common QTL on 2A was identified across the four environments and it explained more than 23% of the total phenotypic variation for plant height. Similarly, Borner et al. (2002) reported the same QTL locations (1A, 4A, and 6A), in addition to another QTL on 6AS.

McCartney et al. (2005) evaluated a population of 182 DH lines in eight environments and revealed six QTLs controlling plant heigth on chromosomes 2D, 4B, 4D, 5B, and 7A. The strongest plant height QTL was on chromosome 4D. This QTL had a LOD score of 30.9 and an R2 value of 47.5%. QTLs on the 4B and 4D were mapped near the two major genes, Rht-B1 and Rht-D1, respectively.

In a study to evaluate a spring wheat population of 140 RIL segregating at Rht- B1 and Rht-D1 under different moisture levels, Butler et al. (2005) found that the two loci had major effects on plant heigth in all four environments with R2 values of 35.9 to 70.0 %.

QTLs for plant height in wheat (*Triticum aestivum* L.) were studied using a set of 168 doubled haploid (DH) lines, which were derived from the cross Huapei 3/Yumai 57. Four additive QTLs and five pairs of epistatic effects were detected, which were distributed on chromosomes 3A, 4B, 4D, 5A, 6A, 7B, and 7D. Two major QTLs, Qph4B and Qph4D, which accounted for 14.51% and 20.22% of the phenotypic variation, were located similar to the reported locations of the dwarfing genes Rht1 and Rht2, respectively (Zhang et al. 2008).

Yu and Bai (2010), observed similar results in a wheat RIL population, where they detected four QTLs for plant height on chromosomes 3D, 5A, and 4D. The QTL on 4D showed larger effect on plant heigth and explained 40 to 59% of phenotypic variation.

2.3.5.3. QTL for yield and yield Components

Grain yield is a complex quantitative trait controlled by a number of genes, each with a small effect on the final product and is highly influenced by the environment. Studies on mapping QTLs (or genes) of yield components have been reported in wheat. Two genes responsible for tiller inhibition were mapped on 1AS and 3A (Kuraparthy et al. 2008; Spielmeyer and Richards 2004). Shah et al. (1999) mapped a significant QTL for TN on chromosome arm 3AL. Araki et al. (1999) detected QTLs for SNS on 4AS. Kato et al. (2000) detected a minor QTL for TN and SNS on 5A. Li et al. (2002) detected QTLs for TN on 6AS, 1DS and 2DS, and QTLs for spike length (SL) on 1AL, 1BS, 4AL and 7AL. Borner et al. (2002) detected QTLs for SL on 1B, 4A and 5A, and QTLs for grain number per spike (GNS) on 4A and 7D. Huang et al. (2003) detected QTLs for tiller number (TN) on 1B, 2A, 2D, 3B, 4D, 5D, 6D and 7A from wild wheat relatives. Huang et al. (2004) detected QTLs for TN on 1B and 7A, and for GNS on 1D, 2A, 3D, 6A, 7A and 7D from elite winter cultivar. Narasimhamoorthy et al. (2006) detected QTL for TN and GNS on 3B and 3D, respectively. Kumar et al. (2007) detected coincident QTLs for TN on 3BL, 4AL and 6DL, QTLs for SL on 1BL, 2DS, 4AL and 5AL, QTLs for spikelets number per spike (SNS) on 2DS and 5AL, and QTLs for GNS on 1AL and 1BL in two populations. Cuthbert et al. (2008) detected QTLs for GNS on 1A, 2D, 3B, 5A and 7A.

Borner et al. (2002) constructed a high density RFLP map to identify QTLs associated with yield and yield components under a range of conditions. The highest number of QTLs was detected for spike length on chromosomes 1BS, 4AS, 4AL and 5AL, detected in 9 of 11 environments.

Groos et al. (2003) evaluated 194 RILs (Table 2.3) at six locations to detect QTLs for grain yield and kernel weight. Seven QTLs were detected for yield on chromosomes 2B, 3B, 4A, 4B, 5B, and 7D. However, only the QTL on 7D was considered to be stable since it was observed in four of the six environments evaluated. Nine QTL were identified for kernel weight on chromosomes 1D, 2B, 2D, 3A, 5B, 6A, 6D, 7A, and 7D, while only the QTL on 2B, 5B, and 7A were

considered to be stable. QTLs for kernel weight and yield co-located on chromosomes 5B and 7D.

An advanced backcross population was used to identify QTLs for yield and yield components in four environments (Huang et al., 2003). Eleven QTLs were detected for grain yield on chromosomes 1B, 2A, 2D, and 5B, explaining from 9.6 to 21.6% of the phenotypic variation. They also identified seven QTLs for kernel weight on chromosomes 2A, 2D, 4D, 5B, 7A, 7B, and 7D. QTLs for other yield components detected in this study were distributed on chromosomes 2D, 3B, 4D, 5B, 6A and 7B.

Huang et al. (2004) studied seven agronomic traits in a 111 BC2F3 families (Table 2..3). In total, 57 QTLs were detected for yield and kernel weight. Nine QTLs were identified for yield on chromosomes 1A, 3D, 4D, 5A, 5B, 6B, and 6D while 14 QTLs were identified for kernel weight on chromosomes 1B, 1D, 2A, 2D, 3A, 3B, 3D, 4B, 6A, 7A, and 7D.

Lin et al. (2008) established field trials in five seasons with a population of 108 DH lines. Three QTLs for grain weight per spike were identified on chromosomes 1A, 2B, 35 and 2D, respectively. Two QTLs for kernel weight were identified on chromosomes 2B and 7B, explaining nearly 14% of the total phenotypic variance in all environments. Five QTLs for biomass were located on chromosomes 1B, 5B, 5D, 7A, and 7D, respectively, explaining a total of 31% of the phenotypic variance.

Cuthbert et al. (2008) evaluated yield and yield components in a DH spring wheat population. QTL analysis of the mapping population detected 53 QTLs across environments for grain yield, yield components, and agronomic traits. They identified QTLs for grain yield on chromosomes 1A, 2D, 3B, and 5A, thousand grain weight on chromosomes 2D, 3B, 5A, and 7A, and harvest index on chromosomes 1A, 3A, 3B, 5A, and 5B. This study identified five major grain yield QTLs on four chromosomes that were consistent across the environments evaluated and coincident with QTLs for at least one yield component.

A mapping population of 182 DH lines was used to construct a linkage map based largely on SSR makers. It was grown in a total of 18 year-site combinations

(environments) (Kuchel et al., 2007). QTLs significantly associated with grain yield were identified on chromosomes 1B, 2D, 3D, 4D, 6A and 6D in one or more environments. Another QTLs located at 6A, was found to be associated with kernel weight at two environments. They also identified QTL for kernel weight on chromosome 7D.

Ma et al. (2007) evaluated a population of 136 RILs over two environments to determine QTLs for spike length. Five chromosome regions were associated with spike length in this population. A major QTL was detected in two environments on chromosome 7D, which explained 29.7 to 36.3% of the phenotypic variation. Other QTLs were distributed on chromosomes 1A, 2D, 4A, and 5B. The 1A location has been associated with spike length in three different mapping populations (Sourdille et al., 2000; Börner et al., 2002; Marza et al., 2006).

A durum wheat population of 249 RILs was evaluated over 16 environments under different drought conditions (Maccaferri et al., 2008). Two major grain yield QTLs were identified in several environments on chromosomes 2B and 3D, with R2 values up to 44.7%. The QTL on 2B, located in the distal region of chromosome 2BL, had a LOD score of 2.5 in eight environments with R2 ranging from 3.5 to 12.4%. The second major grain yield QTL located on the distal region of chromosome 3BS was detected in seven environments with R2 values ranging from 4.8 to 18.1%.

Wang et al. (2009) evaluated a set of 142 RILs (Table 2.4) in four environments. Twenty-one QTLs controlling kernel weight on chromosomes 1B, 2A, 2D, 3B, 4A, 4D, 5A, 6D and 7D were identified across the four environments. Two common QTLs on 1B and 2A were found across all four environments. The detected QTLs on 2A for kernel weight in this population in the interval *Xbarc1165-Xbarc124* seemed to correspond with the QTL results previously detected by Campbell et al. (1999).

2.3.5.4. QTL for kernel characteristics in bread wheat

The economic value of wheat is determined by the class, which in part depends on morphology, texture and size of grain. Grain morphology and texture particularly kernel weight, kernel size, and grain protein content in wheat are important quality traits because they influence the market value of wheat (Yücel et al., 2005).

Seed size and seed numbers are the major determinants of crop yield in both the cereals and the grain legumes. Seed size was also a target of artificial selection during domestication, where large seeds are generally favored due to ease of harvesting and enhanced seedling vigor (Harlan et al. 1972). In wheat, traits related to grain size and appearance have a large impact on market value and play a pivotal role in the adoption of new varieties

The physical properties of grain have a direct or indirect influence on the milling and baking quality of wheat. Many researchers discovered that grain size had an influence on wheat milling and baking qualities (Marshall et al., 1984; Berman et al., 1996). Millers can obtain more flour per unit of weight from large, round, uniform and well-filled kernels. Similarly maltsters and brewers can obtain more extracts from large kernels (Dziki and Laskowski, 2005). Wheat grains of smaller size are considered harder than larger grain and have inferior milling and baking characteristics, whereas larger wheat grains generally have higher weight, which means more endosperm (Gaines et al., 1997). Marshall et al. (1986) reported that grain size, grain thickness, grain sphericity, endosperm size, and grain density are important factors that directly influence the milling yield. The authors also reported that an increase in grain weight and volume was usually due to an increase in grain length, rather than in grain width or height. The grain length, width, and area were associated with a 40% variation in the milling quality of Australian winter wheat cultivars (Berman et al., 1996). Similarly, selection for increased kernel size resulted in higher flour yield in the studies conducted by Wiersma et al. (2001). Dziki and Laskowski (2005) reported a positive correlation between grain size and grain sphericity. Grain shape and grain sphericity influence individual wheat grain as they

pass through the mill (Marshall et al., 1986). The yield and quality of flour are also strongly related to wheat grain properties, which are taken into consideration during wheat milling evaluation. These properties depend on many factors, among which genetic heritage is most important. Therefore, it would be possible to use this information to increase the value of wheat marketed through breeders in developing new varieties with better grain physical characteristics and improved end use product.

QTL analysis for kernel physical traits had not been studied extensively except 1000 kernel weight. Previous studies reported some QTLs for kernel shape, kernel length, kernel width (Sun et al. 2009, Ramya et al. 2010; Tsilo et al. 2010). QTLs reported in these studies represent only a set of QTL alleles that segregate in germplasm studied. Considering the fact that wheat germplasm varies with different marker classess, QTL studies for wheat kernel physical characteristics for all wheat classess are needed to provide good coverage of QTL alleles, including validation of QTLs in different genetic backgrounds. Another important phenotypic assessment of kernel character that has not been used in QTL analysis is kernel sphericity or kernel size distribution. This is an important character because uniformity of kernel allow for efficient milling process and quality control (Yücel et al. 2009; Tsilo et al. 2010). Uniform kernels are ground more evenly during milling, leading to high flour extraction rate and low ash contents (Yoon et al. 2002)

Table 2.4. Important QTL maps for grain physical characteristics in bread wheat published in the recent years

Trait	Population	Location of QTL	No. of line	No. of env	Reference
1000 grain weigth	Milan x Catbird (DH)	1B, 1D, 2B 2D	98	2	Verma et al. 2005
	Opata 85 x W7984 (RIL)	3AS, 5AL, 6BS, 7DS	114	11	Bömer et al., 2002
	NY6432-18xClark's Cream (RIL)	1AS, 1BS, 3B, 7A	78	6	Campbell et al., 1999
	Superb x BW278 (DH)	2D, 3B, 5A, 7A	178	12	Cuthbert et al., 2008
	CA9613 x H1488 (DH)	2B, 7B	108	4	Hai et al., 2008
	Flair X xx86 (shw)	1B,1D,2A,2D,3A,3B,3D,4B,6A,7A,7D	111	6	Huang et al., 2004
	Prinz x W-7984 (AB)	2A, 2D, 4D, 5B, 7A, 7B, 7D	72	4	Huang et al., 2003
	ACKarma X 87E03-S2B1 (DH)	2B,2D,3B,4B,4D,6A	185	3	Huang et al., 2006
	Récital x Renan (RIL)	1D,2B,2D,3A,5B,6A,6D,7A,7D	194	6	Groos et al., 2003
	Trident x Molineux (DH)	6A, 7D	182	18	Kuchel et al., 2007
	RyeSelection111xChinese Spring (RIL)	3A	100	6	Kumar et al., 2006
	Chuang35050 x Shannong483 (RIL)	1D, 5D, 6A, 7D	131	6	Li et al., 2007
	Nanda 2419 x Wangshuibai (RIL)	2B, 7B	136	5	Lin et al., 2008
	RL4452 × AC Domain (DH)	2A, 3D, 4A, 4B, 4D, 6D	182	8	McCartney et al., 2005
	Opata-85 x W7984 (RIL)	1B, 2D	63	2	Pshenichnikova et al., 2008
	Chara x WW2449 (DH)	6B, 7A	190	2	Raman et al., 2009
	Chuan35050 x Shannong483 (RIL)	1D, 2A, 5D, 6A	131	4	Sun et al., 2009
	Heshangmai x Yu8679 (RIL),	1B, 2A 2D, 3B, 4A, 4D, 5A, 6D, 7D	142	4	Wang et al., 2009
	PH82-2 x Neixiang 188 (RIL)	1B, 4A, 5D, 7A	214	6	Zhang et al., 2009c
	RyeSelection111xChinese Spring (RIL)	1A,1D,2B,2D,4B,5B,6B	185	2	Ramya et al. 2010
	MN98550 X MN99394 (RIL)	7A,5B,6B	139	3	Tsilo et al. 2010
	Agropyron cristatum (3228) X jing 4839 (F2:3 families)	4B,5B,7B,5A,4B,6B,7B,1A,5A,6A,	237		Wang et al. 2011

Kernel weight, one of the three major yield components, is greatly influenced by kernel dimensions (KD), such as kernel length (KL), kernel width (KW), etc. Therefore, it is of utmost interest to obtain more information about the underlying genetic control of kernel dimension traits. With the rapid development of molecular marker technology in wheat, increasing numbers of QTL studies have been conducted in an attempt to dissect the genetic basis of thousand-kernel weight (TKW), and all the 21 wheat chromosomes have now been proven to harbor factors affecting it. To obtain information about genetic relationships between TKW and KD at the QTL level, some researchers performed QTL detection for both TKW and KD simultaneously in the same parental mapping populations

Different QTLs were detected in a diverse germplasm when different methods were used to assess kernel size and shape (Giura and Saulescu 1996; Campbell et al. 1999; Dholakia et al. 2003; Breseghello and Sorrells 2007; Sun et al. 2009). Studies on kernel size showed that kernel length and width are influenced by independent QTLs. Previous studies detected QTLs for kernel morphology on several wheat chromosomes. In the population NY18 _ Clark's Cream, homologous group 3 was the most influential for kernel size (Campbell et al., 1999). In the bread wheat population Chinese Spring _ RS111, a QTL was found on 1A (Varshney et al., 2000) and other significant loci were detected on 1D, 2D and 6B (Ammiraju et al., 2001). In another bread wheat population, the most important QTL was detected on chromosome 2D (Dholakia et al., 2003). QTLs for thousand-kernel weight, stable over six environments, were detected on chromosomes 2B, 5B and 7A (Groos et al., 2003).

3. MATERIAL AND METHOD

3.1. Plant material and mapping population

One hundred and fourteen recombinant inbred lines (RILs) were developed through single seed descent method from cross between two commercial cultivars Gerek and Arrehane. Gerek is a Turkish bread wheat originated from the Anatolian Plateau, released as a cultivar in Turkey in 1967 (Altıntas et. al. 2008). Gerek is a winter bread wheat cultivar and adapted to the high altitude areas; it is drought and cold tolerant; tall and late flowering. Gerek has thinner and shorter grain with poor kernel characteristics. Arrehane is a spring bread wheat cultivar, bred at IRNA (France) and released as a commercial cultivar in Morocco in 1996. Arrehane is a semi dwarf, early heading, and drought susceptible and resistant to Hessian fly. It combines wide adaptation with high yield potential and yield stability (Sharıati et al. 2009). Both of the parents are contrasting for major agronomic traits such 1000 kernel weight, spike characteristics, days to heading, as well as kernel physical characteristics such as grain length, grain width, grain height, grain area etc. Diversity of the spike shape, spike length and texture is clearly shown in the Figure 3.1

Figure 3.1. Difference in the spikes length, shape and texture of two parents a). Gerek and b) Arrehan

Seeds of the F_1 hybrids were bulked and one hundred and fourteen seeds were randomly selected and sown to obtain next progeny. At each corresponding generation (F_2-F_6), two seeds again randomly selected from the harvested lot of spikes of each RIL and sown separately in the pots. After germination of two seed per each RIL, one seedling having strong vigor was left while other seedlings were discarded. Diagramatic scheme for the development of recombinant inbred lines population is briefly illustrated in the Figure 3.2 below. Seeds of the two parents and 114 RILs population were kindly provided by Dr M. Miloudi Nachit (durum wheat breeders-ICARDA).

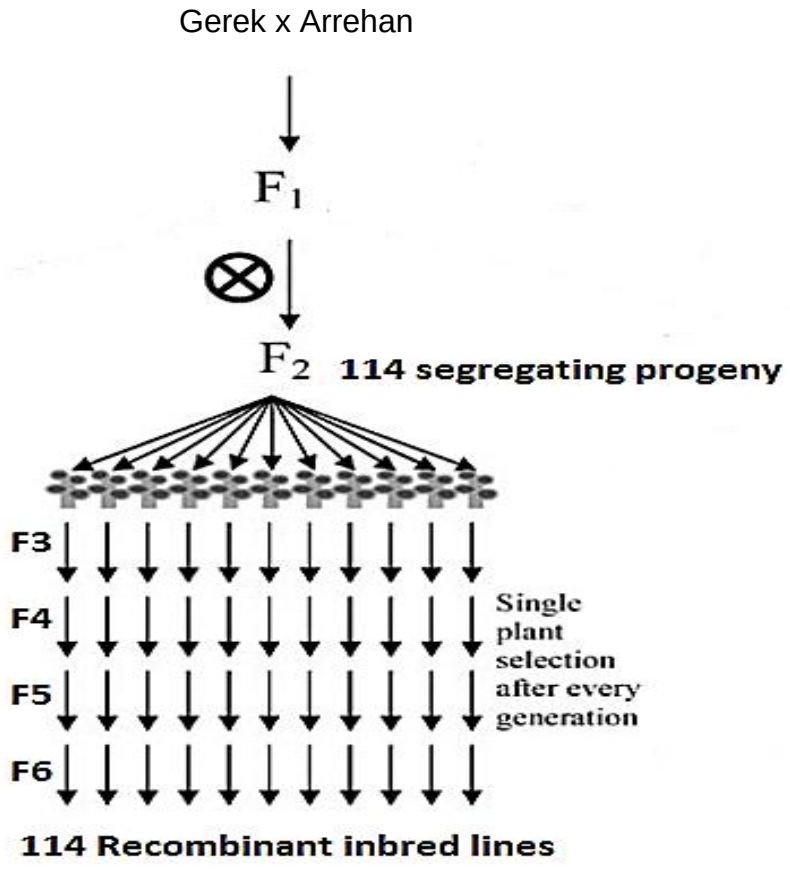

Fig.3.2. Schematic diagram of 114 RIL populations (F_6) developed from the cross of Turkish landrace “Gerek” and Morrocon cultivar “Arrehane”

3.2. Field trials

In 2008-2009, seeds of the all RILs were grown at the Research and Implementation Area of Field Crops Department, University of Cukurova, for multiplying the seeds. After multiplication of seeds, all RILs along with parents were grown in Adana during growing season of 2009-2010 and in three locations during the growing season of 2010-2011. Some characteristics and abbreviations for each location are given in Table 3.1..

Table 3.1. Detail about the location/environment used for growing the population along with years and abbreviations.

Year	Environment-Location	Abbreviation
2009/2010:	Rainfed; Çukurova Uni station-Adana-Turkey	ADA-10
2010/201	Rainfed; Çukurova Uni station-Adana-Turkey	ADA-11
	Rainfed with partial irrigation-Adıyaman-Turkey	ADM-11
	Rainfed; Konya/Ereğeli-Turkey	KON-11

The field experiments were conducted according augmented field design. Each block consisted of 24 genotypes with 5 control cultivars (Federer, 1956). The names of the control varieties were 'Mrb5, Hau, Krf, Waha and Gidara-2'. Total 4-year-site combinations (environment) were utilized to assess the agronomic performance of population. The details of each field experiments at each location are given below:

For 2009/2010 Season:

a) In the field trial at Adana, the seeds of RILs along with parents were sown on well prepared seed bed with hand sowing on 23 November, 2009 (Figure 3.3). Each genotype was sown in a plot consisting of 3 rows, each 2.5 m in length. The distance between rows was 15 cm (Figure 3.2). All genotypes were fertilized in same manner, as recommended 180 kg/ha nitrogen unit (NH_4NO_3), 60 kg/ha of (P_2O_5).

Fig 3.3. A view of the field experiment (2009-2010) conducted at the research and implementation area Çukurova University at seedling stage.

For 2010/2011 Season: In 2010-2011, field experiments were conducted at same location plus two more locations within Turkey.

a) Adana Location

First field trial was conducted at Research and Implementation Area of Field Crops Department of Çukurova University Adana, Turkey, under natural precipitation. Each RIL along with parents were sown in 4 rows with each of 2.5 meter length on 3 December, 2010. The distance between rows was adjusted as 20 cm (Figure 3.4). The amounts of fertilizers used were: N (180 kg/ha) and phosphorus (60 kg/ha).

b) Ereğli/Konya Location

Second field trial was established at Ereğli/Konya. All the RILs along with parents and control cultivars were hand sown on 7 November, 2010. The trial was sown in three rows of 2.5 m in length spaced by 25 cm for each of RIL and parents (Figure 3.5). The amount of fertilizers was given as local

recommended doses as follow: 2/3 (40kg/ha) of (NH_4NO_3) and full dose of phosphorous (40 kg/ha) were given at the time of sowing, and 1/3 (20kg/ha) dose of remaining NH_4NO_3 was added before heading time.

c) Kahta/Adıyaman Location

Third field experiment was conducted at Experimental Station of Kahta Vocational School, University of Adiyaman, Kahta/Adıyaman-Turkey. Adıyaman is located at the South-Eastern Turkey. Each RIL was sown in four rows, each in 2.5 m in length and row to row spacing was adjusted as 20 cm. Two rows were left empty after each genotype (Figure 3.6). The amount of fertilizers were those of locally recommended as follow 2/3 (40kg/ha) of (NH_4NO_3) and full dose of phosphorous (40 kg/ha) were given at the time of sowing, and 1/3 (20kg/ha) dose of remaining NH_4NO_3 was added before heading time.

All trials kept free of weeds and diseases by spraying herbicides and fungicides respectively. Agronomic and plant protection measures were kept normal during the entire experiments.

Fig 3.4. A view of the field experiment (2010-2011) conducted at the research and implementation area Çukurova University.

Fig 3.5. A view of the field experiment (2010-2011) at Konya-Ereğli experimental station

Fig 3.6. A view of the field experiment (2010-2011) at Adıyaman experimental station

3.3. Climatic Characteristics of Experimental Locations

3.3.1. Adana Location

This cites is located at the Research and Implementation Area of Field Crops Department of Çukurova University Adana, Turkey ($37^0$21'N latitude, 35^0 10' E longitude 200 m above the sea level). Adana location possesses rich soil and characterized favorable climatic conditions for local farming system and therefore called as richest agricultural zone of Turkey. This location is different from Ereğli and Kahta locations, because Adana Province is located at the South of Turkey with typical Mediterranean climate with high precipitation in winter but having high temperature and drought condition during grain filling period of wheat. The Total precipitation amounts during the field-experiments in 2008-2009 and 2009/2010 seasons were 578 mm and 606 mm respectively (Turkish Meteorological Service, 2012). The metrological record about this location is given in Figure 3.7 and 3.8.

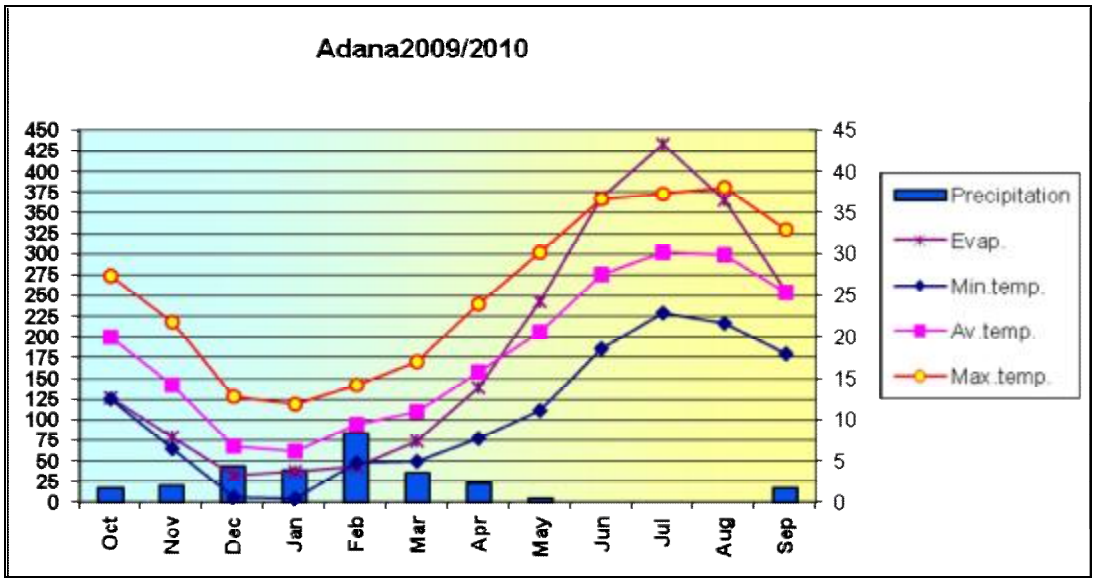

Figure 3.7. The amount of precipitation, average, minimum & maximum temperature and evaporation rate during 2009-2010 at Adana experimental field station

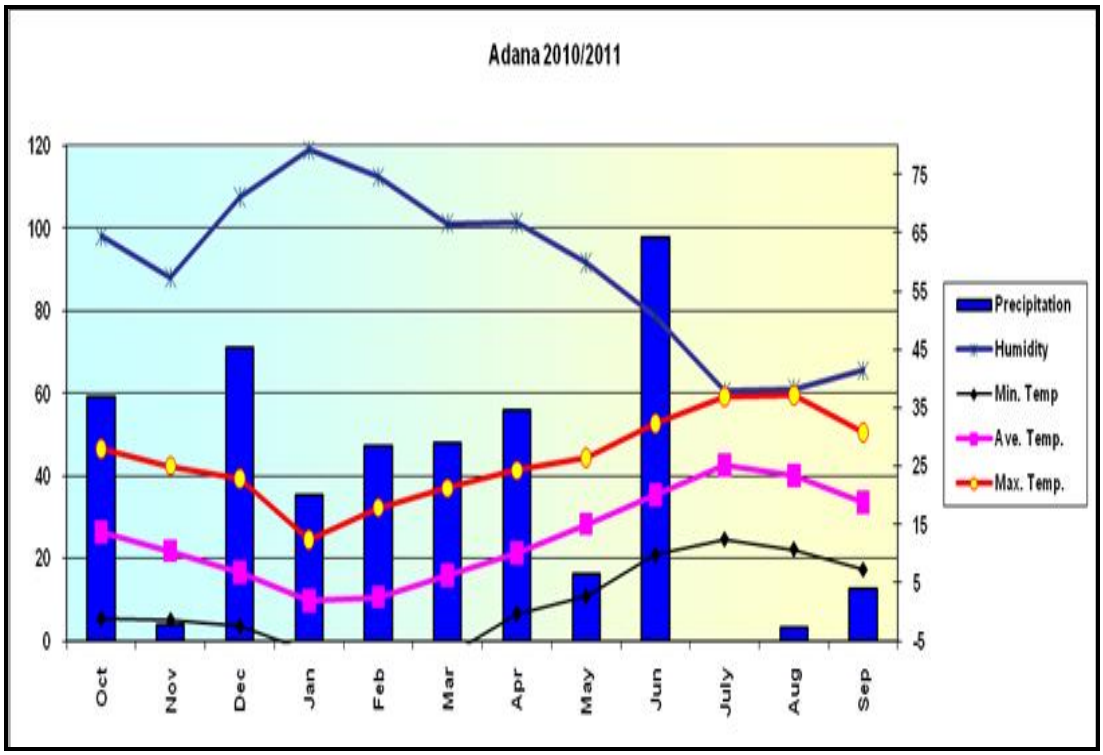

Figure 3.8. The amount of precipitation, average, minimum & maximum temperature and evaporation rate during 2010-2011 at Adana experimental field station

The soil of Adana location was classified as a clay soil in the upper 0-30 cm profile, which contains an average 1.3% organic matter with pH 7.11, 478 mg kg^{-1} of K, 15 mg kg^{-1} of P, 0.69 mg kg^{-1} of Zn, 12.4 mg kg^{-1} of Mn, 1.26 mg kg^{-1} of Cu, and 9.6 mg kg^{-1} of Fe.

3.3.2. Ereğli Location

This experimental cite is located in the Central Anatolian region of Turkey (Latitude; 37° 30' 0" N; Longitude: 34° 3' 0" E and elevation 1044 meters). The Konya Basin is one of the drought regions of Turkey. By the Koppen Classification the climate is semi-arid, with cold moist winters and hot dry summers. Evaporation exceeds total precipitation largely. Ereğli has hard winter with heavy snow and frost. Throughout the Central Anatolia, main crop is winter wheat. This location was selected to check the adaptation of population to hard winter conditions. Moreover,

one of the parent cultivars Gerek was widely grown in central Anatolian region of Turkey. The total annual precipitation amount for 2010 and 2011 338.8 and 390.3 mm (Turkish State Meteorological Service, 2012). The details of precipitation, humidity, average, minimum, and maximum temperatures during the 2009/2010 cropping season are illustrated in detail in Figure 3.9.

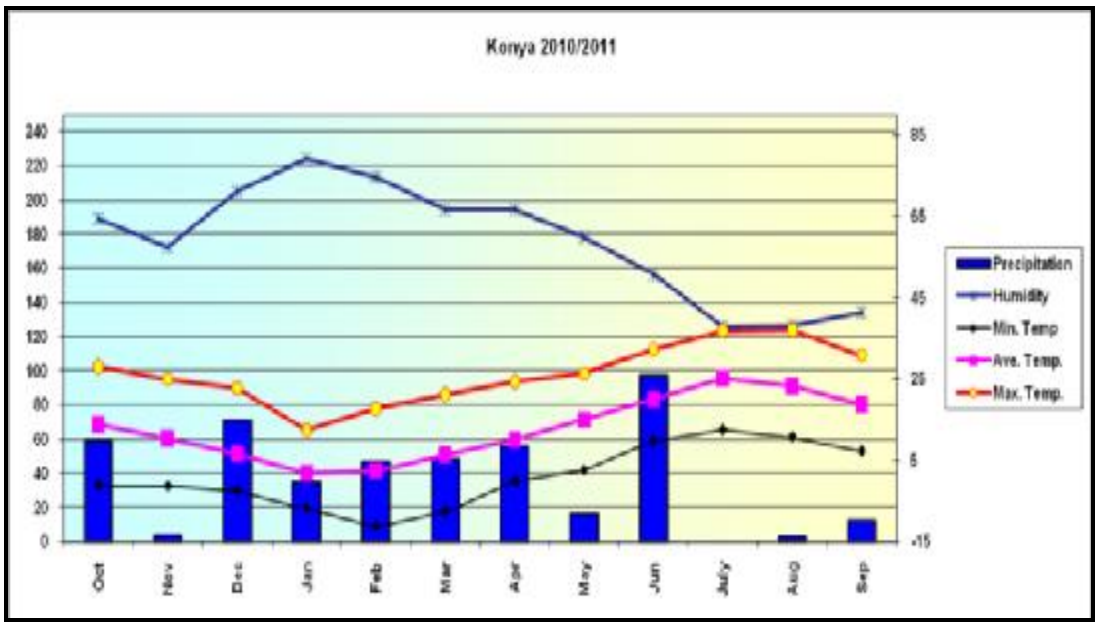

Figure 3.9. The amount of precipitation, average, minimum & maximum temperature and relative humidity during 2008/2009 at Eregli Location

3.3.3. Kahta Location

Kahta is a town of province Adıyaman, about 25 km away from the province Adıyaman. Summers in Adiyaman are hot and dry, winters cool and wet. As such Adıyaman's climate is a hybrid of the mild Mediterranean and the continental climate of eastern Turkey. The effects of the large artificial lake created by the Ataturk dam are still under study. The hottest measured day in Adiyaman was on 7/20/1998 when the temperature hit 45 °C. The coldest recorded temperature was on 2/20/1998 when it got down to -3 °C. The average annual temperature is 17 °C , with 76 rainy days in a year (Turkish Stat Meteorological Service, 2012). On the average

Adiyaman gets snow 7 days in a given year. The amount of precipitation, maximum and minimum temperature and relative humidity is explained in the Fig. 3.10.

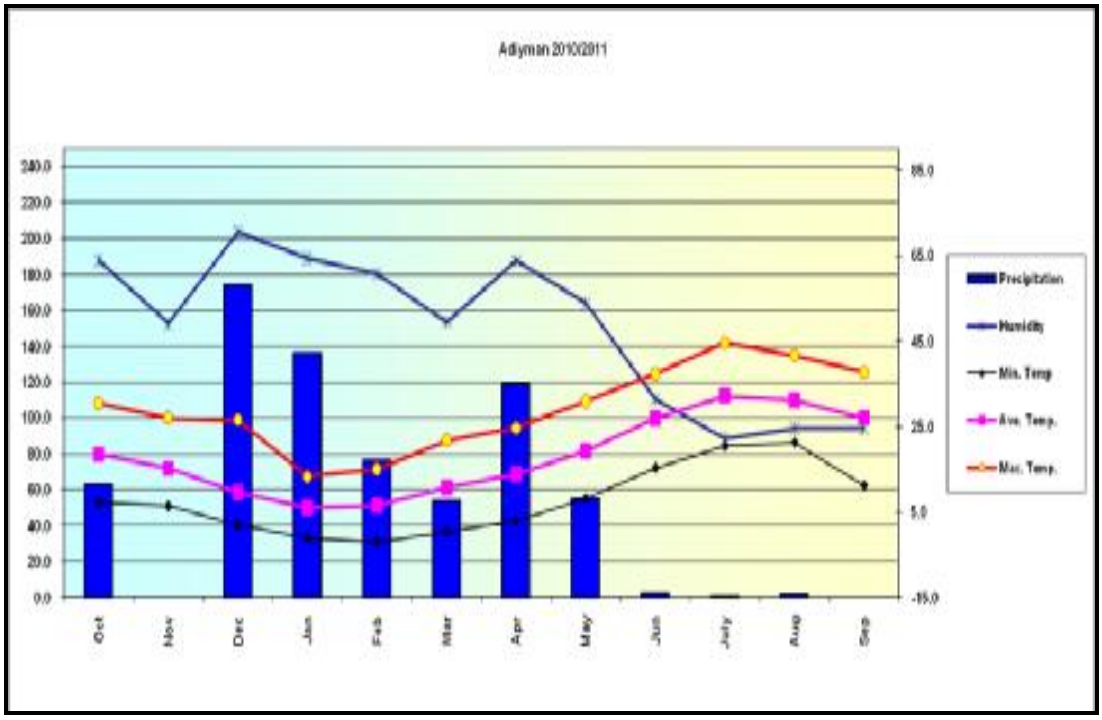

Figure 3.10. The amount of precipitation, average, minimum & maximum temperature and evaporation rate during 2008/2009 at Kahta Location

3.4. Agronomic evaluation of RILs and parents

Data on different agronomic characteristics of the genotypes studied were recorded. Before heading time, the main shoots of 30 plants from each plot were marked with plastic clips. Twenty competitive plants from the parents and RILs as well as the control varieties were sampled randomly from the 30 marked plants from each plot. Plants were harvested when grain moisture was about 13 (Zadoks–94) by hand. A brief detail for each observed trait is given below.

3.4.1. Days to heading (days)

Days to heading was calculated from the date of germination to the emergence of spikes. The days to heading was recorded when 75 % plants from each

RIL were headed. Days to heading was measured only for three locations except Ereğli location.

3.4.2. Plant Height (cm)

Plant height was recorded at maturity one week before harvest from twenty randomly selected plants from each genotype. Plant height was measured with the help of meter rod from ground level to highest growing point.

3.4.3. Spike traits.

To record data on different spike characteristics, twenty spikes were randomly selected from marked 30 main stem spikes from each experimental unit from each location/year. Spikes of all twenty main stem plants were removed with the help of sickle. Data on different spike characteristics were recorded and detail is given below.

3.4.3.1. Spike length (cm)

Spike lengths were measured from twenty main stem spikes with the help of meter rod from the basal of the lowest spikelets to upper most point of spike without awn length.

3.4.3.2. Number of spikelets per spike

Data for number of spikelets per spike were recorded from counting the number of spikelets per spike and then averaged.

3.4.3.3. Number of grains per spike

All twenty main stem spikes were threshed manually with hands one by one

and grains obtained were counted and averaged to number of grains per spike.

3.4.3.4. Grain weight per spike (g)

Grain weight per spike was measured by weighting the all grains of each spike separately on a sensitive balance.

3.4.3.5. Spike compactness (number/cm)

Spike density or spike compactness (number of spikelets per each cm of spike) was calculated as a ratio of number of spikelets per spike and spike length.

3.4.5. 1000 grain weight

Thousand grain weights were inspected by counting of four samples of hundred kernels taken randomly from harvested grain of each plot, and then averaged to 100 grains weight, multiplied by 10 to get 1000 kernels weight.

3.5. Molecular Analysis

3.5.1. DNA extraction

Young leaves from each RIL along with parents were collected from two weeks old seedling. The tissues were transferred to the laboratory on the same day in liquid nitrogen and stored at -80°C. DNA extraction was performed according to CTAB protocol (Doyle and Doyle, 1996), with some modification of Özkan et al. (2005). Frozen plant tissues (about 3-5 g of fresh leaf) were ground by mortar and pestle to a fine powder and suspended with 800 µl CTAB buffer. Samples were incubated at 65^{o}C for 1.5 hour with constant stirring after each 10-15 minutes by hands. An equal amount of chloroform/isoamylacohol (24:1 v/v) was added and tubes were centrifuged at 14000 rpm for 10 min. The supernatant was transferred to

new tube of 1.5 ml. 2/3 volume of the isopropanal was added to each tube. The tubes were gently inverted several times by hands to compact the DNA and left at -20 ^{o}C for few hours or overnight. Then DNA was centrifuged at 8000 rpm for few seconds. The DNA was fished out and washed twice in 10-25ml of 70% ethanol: ammonium acetate by stirring it for 10 minutes on the electronic stirrer then dried by putting the tubes over tissue paper at room temperature. 50-100 µl of ddH_2O was added to each tube and DNA template was completely dissolved in the ddH_2O.

3.5.2. Measurements of DNA concentration

The quantity of the total DNA extracted from each plant was measured by quantitatively and qualitatively by 0.8 % Agarose gel electrophoresis. DNA concentration was estimated and standardized against known concentration of ʎ DNA. Before using DNA molecular analysis, the DNA diluted to a concentration of 20ng/µl for SSR applications and DArT analysis.

3.5.3. Simple sequence repeats (SSRs) or microsatellite analysis

SSR markers used in this study were selected from the previous published literature (Röder et al. 1998). Origin of SSR markers used in this study is given in Table 3.2. Total 317 SSR primer pairs were screened for polymorphism between Gerek and Arrehane. Only 65 SSR primers showed polymorphism among parents, 45 primers gave no amplification product or poor amplification, whereas rest of the 207 markers was monomorphic. First of all, M13 tailed-primer PCR amplification of SSR was performed according to Schuelke (2000) with some modification of Comertpay et al. (2012). M13 tailed-SSR markers produced some nonspecific amplification profile. To reduce or eliminate the nonspecific amplification of M13 tailed-SSR markers, different concentrations of the M13 primer (0.2pm, 0.4pm, 0.6pm, 0.8pm and 1,0 pm) and $MgCl_2$ (0.2, 0.4 and 0.6 ul; 2.5mM) were used. 0.6pmol concentration of M13 primer and 0.6 µl (2.5mM concentration) of $MgCl_2$ showed best results with minimum nonspecific amplification.

Table 3.2. Summary of wheat obtained different source microsatellites used in Gerek/Arrehan mapping population

Microsattelite developer	Abbreviation for SSRs	SSR used	Publishing Reference
Marion Röder (IPK)	GWM	114	Röder et al. (1998)
Pestova	GDM	3	Pestsova et al. (2000
Dr. P. Isaac	WMC	116	Somer et al. (2004)
Perry Cregan (USDA)	BARC	9	Song et al. (2005)
Pierre Sourdille (INRA)	CFA	21	http://wheat.pw.usda.gov
Pierre Sourdille (INRA)	CFD	54	http://wheat.pw.usda.gov

The PCR mixture consisted of total volume of 12 µl PCR containing 1× PCR buffer (1.2 ul), 2.5 mM dNTP (0.96), 25mM $MgCl_2$ (0.4 µl), 0.6 pmol universal M13 sequence tailed forward primer, 5pmol (0.12 µl) of forward and 0.48 µl reverse primer, , 0.12 U/µl Taq DNA polymerase, and ~15 ng genomic DNA. PCR cycling program consisted of initial denaturation of 94°C for 5 minutes, 30 cycles consisting of 94°C for 1 min, annealing temperature for 1 min, 72°C for 1 min, followed by 8 cycles for M13 labelled primer consisting of 94°C for 30sec, 53°C for 45sec and 72°C for 45 sec, and final extension at 72°C for 15 min.

3.5.3.1. Polyacrylamide gel electrophoresis for resolving microsatellite DNA fragments at LICOR DNA analyzer

Polymorphism between the parents and segregation of polymorphic alleles were revealed when electrophoresed on a denatured polyacrylamide gel using LICOR DNA analyzer (Model 4300). Protocol for preparation of the polyacrylamide gel for LICOR is given in the table 3.3

The PCR product was mixed with fuchsine dye and denatured for 5 minutes at 94^oC and waited for few minutes on ice before loading. The amplified fragments were separated on 6% denaturing Acrylamide gel (19:1) and photographed on LI-COR Model 4300 DNA Analyzer.

The primers that produced polymorphism between the two parents were used to survey the DNA of F_6 RIL population, and data was collected about the polymorphic fragments/bands in F_6 population.

Table 3.3. Amount of reagents required for preparing one polyacrylamide gel at LICOR DNA analyzer

Reagents	Concentration
Gel casting solution (high resolution sequa gel)	16ml
Buffering reagent (urea gel)	4ml
10 % APS (ammonium per sulphate)	160 ul

3.5.4. Diversity Array Technology (DArT) Analysis

Diversity Arrays Technology (*DArT*) marker genotyping, genomic DNA of two parental lines and 114 RILs were sent to 'Triticarte^{TM} Pty Ltd (Whole genome genotyping service for wheat and barley)'. (http://www.triticarte.com.au/). For each of these arrays, a genomic representation was generated from a mixture of wheat cultivars using the *Pst*I-based complexity reduction metho described by Wenzl et al. (2004). The procedure involved digestion of a mixture of DNA samples with *Pst*I and one of the following frequently cutting restriction enzyme: *Mse*I, *Rsa*I, *Taq*I or *Bst*NI (NEB, Beverly, MA, USA), ligation of a *Pst*I adapter with T4 DNA ligase (NEB), and amplification of small, adapter-ligated fragments (Wenzl et al. 2004). A library was prepared from each of the amplification products, also called genomic representations, as described by Jaccoud et al. (2001) with the modifications of Wenzl et al. (2004). The cloned representation fragments were amplified (Jaccoud et al. 2001), dried at 37°C and dissolved in a spotting buffer which was initially either 50% (v/v) DMSO or a 1:1 mixture of 50% (v/v) DMSO and the printing buffer of the Vanderbilt Microarray Shared Resource (Nashville, TN, USA). Depending on the number of clones, two or three replicate spots per clone were printed on the arrays. After printing, the slides were heated to 80°C for 2 h (unless spotted with the new buVer), denatured by incubation in hot water (95°C) for 2 min, dipped in 95% (v/v) ethanol (unless spotted with the new buffer) and dried by centrifugation (Please see Akbari et al. (2006) for detail information about DArT proceedure).

The marker names e.g. "wPt" were described according to Akbari et al. (2006), where 'w' was designated if the clone was derived from a wheat library and 'Pt' stands for *PstI* and *TaqI* restriction enzymes used to generate clones. The version

2.0 wheat DArT array of 5,137 clones was used for genotyping RILs using the methods previously described by Akbari et al. (2006).

3.6. Statistical Analysis

One hundred fourteen RILs, their parents and five checks were tested using the augmented design (Federer, 1956). The morphological data for all traits was adjusted according to control cultivars using computer software program TARIST (Açıkgöz, 1990). The standard deviation, maximum, minimum and mean values for all traits were calculated using SPSS computer software program. Associations among traits were calculated using the Pearson correlation procedure implemented in SPSS. Pattern of distributrion among RIL was analysez by conducting histograms for all studied agronomic traits using SPSS program.

3.6.1 DArT analysis

An ANOVA based estimate of marker quality, which reflects how well the two phases (Present = 1 vs Absent = 0) of the marker are separated in this dataset. From our experience, markers with P value above 80 are scored very reliably. We are now reporting markers with lower P value since many could be mapped, but they may contain more scoring errors. Because P is based on ANOVA, it is not an appropriate quality measure for markers with a very asymetric distribution of 0 and 1 scores (low PIC). This is why some markers with much lower P but high call rate, low discordance rate and low PIC are also reported (Triticart Pyt. Ltd).

Tentative assignment of chromosomal location of the each DArT marker were also provided by Triticart Pyt. Ltd based on based on many well-curated genetic maps, built and analysed by Triticart team. Image analysis and polymorphism scoring was done. using software DArTsoft version 7. DArT software automatically computed several quality parameters for each marker such as

a) ***Call rate***: Percentage of valid scores in all possible scores for a marker,

b) ***PIC value***: Polymorphism information content (PIC): a maximum of 0.5 when a marker scores 50% 0 and 50% 1,

c) ***One ratio***: Number of score 1 divided by number of scores.

d) ***Reproducibility of each DArT marker:*** Measure in % how reproducible the scoring for replicated samples is. (100 means 100% reproducible). A small number of markers were also analysed in duplicate.

3.6.2. Chi square test

The $\chi 2$ test was applied to identify DArT and SSR markers with a distorted ($P<0.01$) segregation ratio from the expected Mendelian ratio.

3.6.3. Linkage analysis

The genetic linkage map was constructed using JointMap V5.0 (Lander et al., 1987; Lincoln et al., 1993), with an LOD threshold of $\geq$ 3.0. Recombination distances were determined using the Kosambi mapping function (Kosambi, 1944). Chromosome assignment was determined by comparing this map with the previously published wheat microsatellites and DArT maps

3.6.4. QTL analysis

A widely applied QTL mapping method is “conventional” interval mapping, first described by Lander and Botstein (1989) and successfully applied in a number of case studies (Jansen and Stam, 1994). The implemented QTL mapping procedure is a maximum likelihood approach to the segregation of a mixture of probability distribution. Under the hypothesis that single QTL is segregating the mixture consists of Q distributions, one for each QTL genotype for RILs Q =3. The component distributions are assumed to be normal, with means μ_q and common variance σ^2. In

the mixture model, the mixture density $f(x_n)$ for individual n=1...*N* is the sum of the products of the component densities $f_q(x_n)$ with their probabilities π_q :

$$f_q(x_n) = \pi_q f_q(x_n),$$

Thus, the likelihood for the population under the hypothesis that a QTL is segregating, L_1, is:

$$L_1 = \prod_{n=1}^{N} f(x_n) = \prod_{n=1}^{N} \sum_{q=1}^{Q} \pi_q f_q(x_n).$$

q= component probabilities

With the EM algorithm, the likelihood, or actually its logarithm, is maximized and the parameters π_q and σ^2 are estimated (Dempster et al., 1977). The L_1 likelihood is compared to the likelihood under the null-hypothesis, L_0, which is similar to L_1expcept that there is just a single component in the mixture. The compression is done using the so-called *LOD* score (LOD=log of odds; Branard, 1949) as a test statistic:

$$LOD = {}^{10}\log (L_1/L_0).$$

In order to quantify the genotypic information across the genotypic *information coefficient (GIC)* is defined, the *GIC* can have a value in the range from 0 to 1, zero meaning there is no marker information at all, one meaning that there is complete or maximum marker information. The derivation of the *GIC* is as follows.

A QTL generates an amount of genotype variance V_Q. This variance can be partitioned into the variance to be explained by genotypic (marker) information V_M, and the variance remaining due to uncertainty in the genotypic (marker) information V_R. From this partitioning the *GIC* is defined as:

$$GIC = V_M / V_Q = (V_Q - V_R) / V_Q .$$

For RIL population the GIC has the following formula:

$$GIC = 1 - 2\sum_{n=1}^{N} \left(\pi_A + \pi_B - (\pi_A - \pi_B)^2\right) / N .$$

4. RESULTS AND DISCUSSION

4.1. Linkage Analysis and Genetic Map Construction

Turkey is one of the most important gene center for bread wheat as well as other cereal and legume crops. Despite of the importance of genetic resources from Turkey and other Fertile Crescent countries, any effort has not been attempted to create a genetic linkage map of bread wheat from this region. To our best of knowledge, this is the first genetic linkage map of bread wheat using Turkish cultivar "Gerek". In this study, the genetic linkage map of bread wheat was constructed using the 114 recombinant inbred lines (RILs) derived from cross between one Turkish cultivar "Gerek" and Moroccan cultivar "Arrehane" using Diversity Array Technology (DArT) markers anchored with SSR markers.

4.1.1. Evaluation of Markers

4.1.1.1. Diversity Array Technology (DArT) Analysis

DArT platform (Wheat v3 7000 markers for high density) was performed by Triticart Pty. Ltd (Canberra, Australia) according to description of Akbari et al. (2006). Totally 7000 DArT for high density map was screened for polymorphism among parents. Out of 7000 markers screened, 1109 markers showed polymorphism among parents and were applied to whole set of RILs by Triticart Pty. Ltd (Canberra, Australia). *DArT* markers showing identical banding pattern in both parents either as 0,0 and/ 1,1 were discarded. Later *DArT* markers with missing values in either of the two parents (1,-; 0,- and/or -,1; -,0) were also eliminated for linkage map construction. Two parameters were used eliminated problematic markers for linkage analysis. Clones with a P value (an estimate of marker quality, which reflects how well the two phases "--Present = 1 vs. Absent = 0--"of the marker are separated in this dataset) higher than 80 and a call rate (Percentage of valid scores in all possible scores for a marker) of at least 80% or higher than 80% were initially selected for

mapping analysis, later clones with P values between 71 and 80% were also integrated into linkage mapping analysis. Similarly, *DArT* markers with a polymorphism information content (PIC) value less than 0.304 were also removed from the linkage map analysis (Table 4.1). PIC value equal to 0.5 means equal number of "1" and "0". Therefore, *DArT* markers with extremely lower PIC values (between 0.0 and 0.3) were also discarded from the analysis.

Table 4.1. Polymorphism Information contents (PIC) values of 1109 DArT markers used to construct genetic linkage map of Gerek x Arrehane mapping population

PIC value	Number of DArT markers	% of Total DArTs
0.4-0.5	878	79.1
0.3-0.4	155	14
0.2-0.3	12	1.1
0.1-0.2	23	2.1
0.0-0.1	41	3.7
Total	**1109**	**100**

Finally linkage analysis was conducted with total of 883 *DArT* markers. Out of 883 *DArT* markers used for mapping, 827 were mapped on different linkage groups whereas rest of the 56 *DArT* markers were not mapped and remained unlinked.

Most of the *DArT* markers were mapped according to their chromosomal location provided by Triticart Pyt. Ltd. and published *DArT* literature. Out of 827 *DArT* markers, 23 *DArT* markers were mapped on different chromosomes instead of their known chromosomal location reported in the published literature (list is given in the Table 4.2).

Similarly seventy seven *DArT* markers (9.3%), of which chromosomal locations were until now not reported in the literature, were clustered with *DArT* or SSR markers on different chromosomes (Table 4.3).

Table 4.2. DArT markers mapped on the chromosomes different from ones reported in the published literature

DArTs	Reported chromosomal location	Assignment in G x A map
wPt-732950	3D	3B
wPt-732795	3D	3B
wPt-5313	1B/3D	3B
wPt-731250	6A	6B
wPt-7427	4A	6B
wPt-6748	4A	6B
wPt-7524-	4A	6B
wPt-9445	4A	6B
wPt-7001	4A	6B
wPt-0357-	6B	6A
wPt-7567-	1A	2B
wPt-3657	6B	7D
wPt-663764	6B	6D
wPt-2741	6A	6D
wPt-664603	6A	6D
wPt-672029	6A	6D
wPt-665972	2A	6D
wPt-2861	1B	1D
wPt-8637	5B	1D
wPt-2316	7A	7B
wPt-7947	7A	7B
wPt-7763	7A	7B
wPt-742929-	2B|6B	1B

Table 4.3. Number of DArT markers of which chromosomal locations were until now unknowns, and their positions in Gerek x Arrehane map

Number of DArTs	Position in G x A map
3	1A
3	1D
2	2A
2	2B
4	2D
2	3A
4	3B
19	3D
2	4A
6	4D
1	5B
4	5D
2	6A
2	6B
11	6D
1	7A
3	7B
6	7D

4.1.1.2. SSR Analysis

Microsatellite markers were used as anchor primers for chromosomal arms. They allow the construction of a high-confidence framework map. Microsatellite markers are genome and chromosome specific and/or locus specific. Their inclusion in the linkage map will allow more precise and accurate identification of the genomic region linked. Some SSRs were included in the present genetic map in order to confirm the *DArT* markers position on the chromosomes.

Totally 317 SSR markers were screened for polymorphism among two parents (Figure 4.1), previously mapped in genome A, B and D (Material and Method section; Table 3.2). Out of 317 SSR markers screened for parents, only 64 markers (20%) showed polymorphism among the whole set of 114 RILs (Figures 4.2-4.6). Polymorphisms among parents have been reported as 10-40 % in earlier mapping studies. Polymorphism around 40 percent was reported among durum wheat

parents (Alsaleh 2011). However Vaissayre et al. (2012) did also found polymorphism about 16.0 % among parents (only 67 markers out of total 420 SSR markers). In the present genetic map construction, SSR markers were selected to anchor the DArT markers on all 21 chromosomes of bread wheat but unfortunately some of the chromosomes were remained without SSR markers due to lack of polymorphism in SSR markers for those chromosomes. Out of 64 polymorphic SSR markers, only 39 SSRs were mapped on the different chromosomes whereas rest of the 25 SSR markers were not linked and unmapped at all. Some SSR markers were clustered with DArT but were not mapped on the linkage groups due to week or insufficient linkage

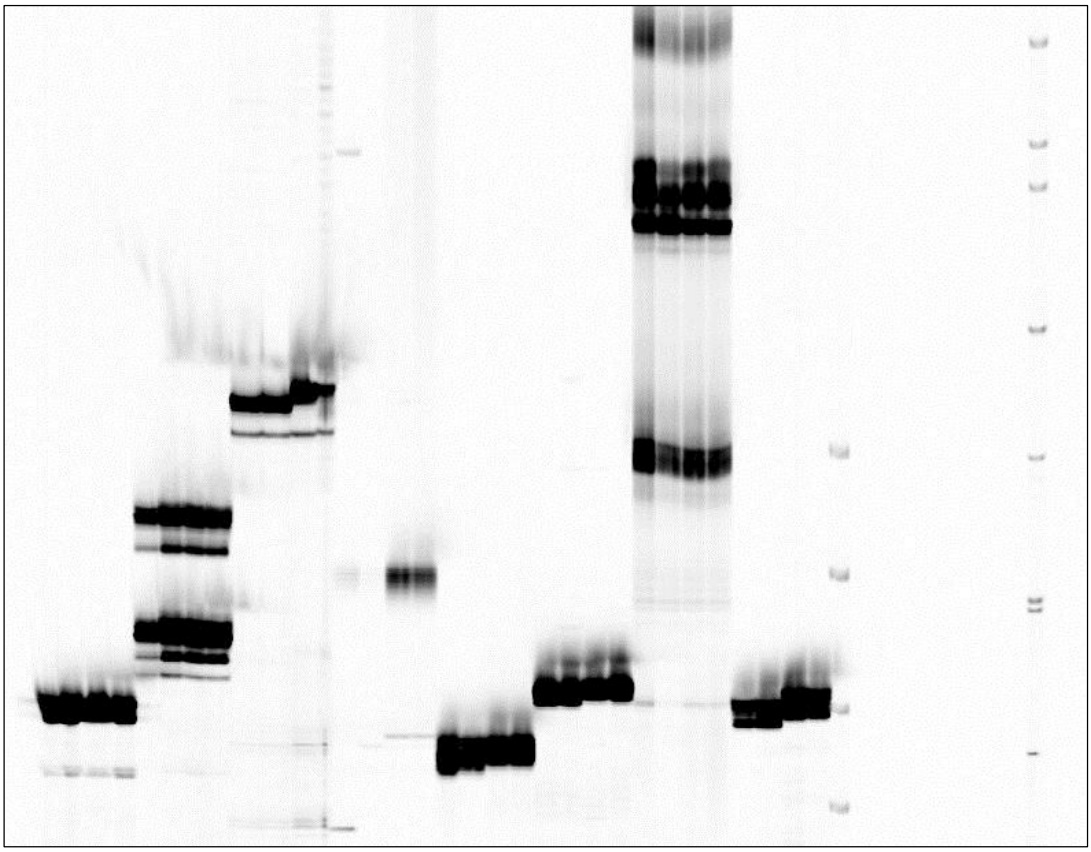

Fig 4.1. Screening of the parents with 8 SSR markers. Arrow showed the polymorphic markers among parents

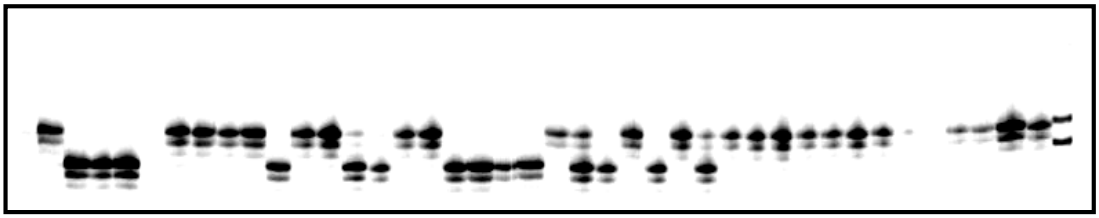

Fig. 4.2. Polyacrylamide gel profiling of SSR marker cfd84 using Licor DNA analyzer

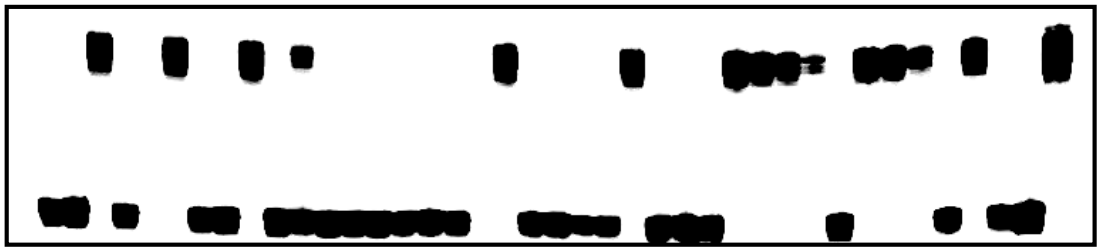

Fig. 4.3. Polyacrylamide gel profiling of SSR marker gwm499 using Licor DNA analyzer

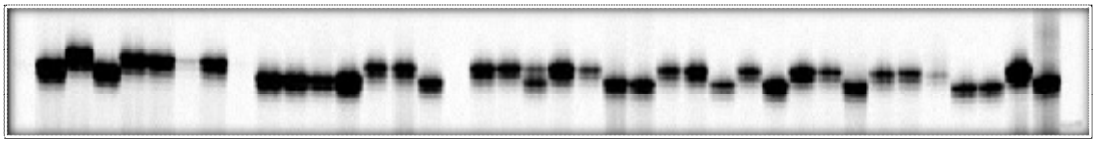

Fig. 4.4. Polyacrylamide gel profiling of SSR marker cfd92 using Licor DNA analyzer

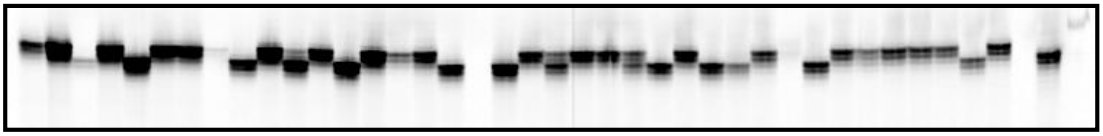

Fig. 4.5. Polyacrylamide gel profiling of SSR marker wmc231 using LICOR DNA analyzer

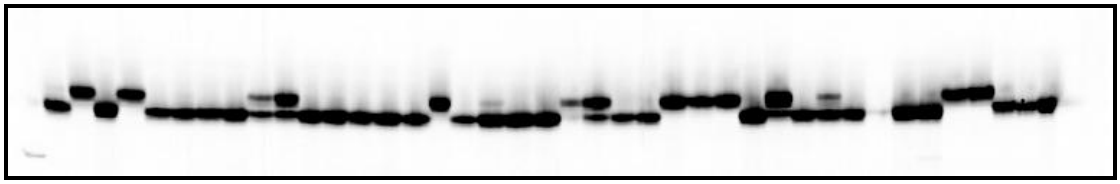

Fig. 4.6. Polyacrylamide gel profiling of SSR marker wmc283 using LICOR DNA analyzer

between SSR and DArT markers. This reinforce to include higher number of SSR marker and as well as to include also other type of markers (non DArT markers) in the map in order to cover all possible genomic regions. High Segregation distortion among DArTs could be also one of the possible reasons for unlinked SSR markers. Elouafi and Nachit (2004) also reported the unmapped SSR and AFLP markers in genetic linkage map of *Triticum durum* x *Triticum dicoccoides* backcross population. All of the microsatellite markers generated only one polymorphic fragment. These findings confirmed earlier results showing that the majority of microsatellite markers are genome-specific and usually amplify only a single locus (Röder et al., 1998; Korzun et al., 1999; Elouafi and Nachit, 2004). Most microsatellite markers mapped are in good agreement with the previously published wheat maps. However two SSR markers were mapped on the chromosomes different from ones reported in the published literature (Table 4. 4)

Table 4.4. SSR markers mapped on the chromosomes different from ones reported in the published literature

SSR Marker	Location in G x A map	Known location location	Reference
Cfd5	6D	5B	Somer et al. 2004
Cfd48	1B	1D	Somer et al. 2004

4.1.2. Genome coverage

Final genetic map consisted of total 866 markers including 827 *DArT* and 39 SSR markers, assigned to all 21 chromosomes, spanning a total map length of 935.629 cM. Total number of markers mapped varied from 5 to 87 depending on the chromosomes (Table 4.5). Chromosomes 5D and 4D contained least number of markers (5 and 6 markers respectively) whereas chromosome 3B contained highest number of mapped markers (87 markers). The average number of mapped markers per chromosomes was 41.2 markers/chromosomes. The map length of the chromosomes also varied from 3.252 to 81.367 cM. Chromosome 4D was one with the shortest map length (3.252 cM) whereas chromosome 3B was the chromosome

with longest map length 81.367 cM, with average map length of 44.54 cM.

There was also variation for marker density per chromosome (Table 4.5). The density of the markers on the map also varied from 0.465 cM/markers on 4D to 2.459 cM/markers on the chromosome 2D, with an average of 1.080 cM/marker.

Distribution of molecular markers, assignment, and cM coverage across the 21 bread wheat chromosomes in the A, B and D genomes is given in Table 4.5 Genome B contained more markers (359 markers) compared with genome D with total 259 markers and genome A with markers 248 markers. Total map length of genome B is 402. 892 cM, which higher compared with genome D (273.983 cM) and genome A (258.754 cM). Genome D contained 20 SSR markers, whereas 10 and 9 SSR loci were assigned to genome B and genome A respectively.

Table 4.5 Distribution of molecular markers, assignment, and cM coverage across the 21-bread wheat chromosomes, 7 homologous groups and in the A, B and D genomes

Chromosomes	No of Markers	ML (cM)	MD	No of SSR markers	No of DarT markers
1A	75	74.453	0.993	3	72
1B	33	43.738	1.367	2	31
1D	53	57.797	1.070	2	51
2A	16	22.422	1.401	0	16
2B	78	70.442	0.903	0	78
2D	32	78.681	2.459	9	23
3A	24	38.178	1.591	0	24
3B	87	81.367	0.935	2	85
3D	60	28.292	0.472	1	59
4A	21	19.599	0.933	1	20
4B	16	26.537	1.659	2	14
4D	7	3.252	0.465	0	7
5A	11	26.022	2.366	3	8
5B	30	52.275	1.743	2	28
5D	5	5.16	1.032	1	4
6A	79	32.046	0.406	0	79
6B	70	54.824	0.783	0	70
6D	36	70.744	1.965	4	32
7A	22	46.034	2.092	2	20
7B	46	73.709	1.602	3	43
7D	65	30.057	0.462	2	63
A genome	248	258.754	1.043	9	239
%	28.72			23.68	28.80
B genome	359	402.892	1.122	11	349
%	41.55			26.32	42.53
D genome	259	273.983	1.058	19	239
%	30.96			50	28.67
Group 1	161	175.988	1.093	7	154
Group 2	126	171.545	1.361	9	117
Group 3	171	147.837	0.865	3	168
Group 4	44	49.388	1.122	3	41
Group 5	46	83.457	1.814	6	40
Group 6	185	157.614	0.852	4	181
Group 7	133	149.86	1.126	7	126
Total	866	935.629	1.080	39	827

ML: Map length, MD: Marker density. * Average marker density.

There were also high differences for number of markers, length of chromosomes and markers density on the basis of homologous groups (Table 4.5). Highest number of markers was placed on homologous group 6 with 185 markers, followed by homologous group 3 (171 markers), homologous group 1 (161 markers), homologous group 7 (133 markers), homologous group 2 (126 markers) whereas the lowest number of markers was grouped on homologous group 4 with 44 markers, followed by homologous group 5 (46 markers). Homologous group 4 was the group with the shortest map length (49.388cM) followed by homologous group 5 (83.457cM). On the other hand, homologous group 1 have highest map length (175.988cM) followed by homologous group 2 (171.545 cM). The lowest marker density was noted in homologous group 6 with marker density of 0.852 cM/marker, whereas highest marker density was recorded in homologous group 5 with marker density of 1.814 cM/marker followed by homologous group 2 with marker density of 1.361 cM/marker. Highest number of SSR was mapped on homologous group 2 having 9 SSR markers followed by homologous group 1 and 7 each having 7 SSR markers. Homologous group 5 has 6 SSR markers. Homologous groups 3 and 4 each have 3 SSR markers whereas homologous group 6 has 4 SSR markers.

The length of the map was shorter than previously published maps for bread wheat (Paillard et al. 2003; Semagn et al. 2006; Akbari et al. 2006; Quarrie et al 2005; Li et al. 2007; Huang et al 2006). There could be several reasons for shorter map length in this study. The DArT markers amplify some genomic regions and hybridized with the amplified clones to produce hundreds of fragments. But these hundreds of fragments are resulted from only few genomic areas resulting in very close genetic distances. In our study, average distance between two *DArT* loci was 0.6cM. The low average distances between two consecutive *DArT* loci have been reported in many species including bread wheat. Our results have been supported by Semagn et al (2006), who also observed an interval < 0.5cM between 2 consecutive DArT loci with high correlation coefficient ($r > 0.98$). This map is one of the most dense map published for bread wheat with low genome coverage but density (in term of number of markers per cM).

4.1.3 Linkage groups

Linkage mapping in the present study identified fifty four linkage groups, many more than the twenty one haploid chromosomes of hexaploid wheat. Number of linkage groups varied among different chromosomes and genomes. Number of linkage groups per chromosomes varied from 1 for 1B, 1D, 3D, 4D and 5D, to 5 for 7B. There could be several reasons for higher number of linkage groups per chromosomes. First, these large numbers of linkage groups compared with haploid chromosomes number suggest that several genomic regions remain undetected with the present set of markers. The requirement of large number of markers or mapping population to reduce the linkage groups to haploid chromosomes number has been emphasized in previous published literature (Sharma et al. 2002; Semagn et al. 2006). However in the present study, linkage map was constructed with total of 866 markers that is higher than most of the previous published maps (Semagn et al. 2006; Nalini et al. 2007; Suenaga et al. 2005). In spite of large number of markers used in the present study, several genomic regions remained undetected. Use of the DArT markers could be one of the possible causes of the large number of linkage groups. Iqbal et al. (2012) reported that *DArT* markers could be biased to certain genome regions, suggesting that large number of markers amplified on the same genomic region. Most of the markers amplified very close to some genomic regions resulting in the gap between the markers ultimately resulting in the large number of groups. Semagn et al. (2006) constructed the bread wheat map using 624 markers with 189 DArT, 165 AFLP, 270 SSRs and reported 58 linkage groups. These authors also reported that the large number of linkage groups in their study was not due to poor data quality; they downloaded the ITMI (International Triticea Mapping Initiative) population data from the grain gene website and constructed the genetic linkage map. In contrast to 21 linkage groups reported by Song et al. (2005), they found 56-63 linkage groups in ITMI population.

Theoretically the number of linkage groups should be equal to the number of haploid chromosomes, so the whole genetic linkage map should include 21 linkage groups. However this map has 54 linkage groups with 33 extra groups. Few linkage

groups have only small number of markers. This enforces to include larger number of markers to saturate the linkage map. Similarly higher number of linkage groups was also reported in durum wheat, for example, 21 linkage groups at JK x Ch1 (Nachit et al. 2001), 18 at MDM (Elouafi and Nachit, 2004), 23 at K/S map (Maccaferri et al, 2008), 25 at Meridiano x CLaudio, 20 at Colosseo x Lloyd (Trebbi et 2011) and 26 at consensus parental maps (Vaissayre et al 2011), instead of 14 linkage groups of durum wheat.

4.1.4 Segregation disorder

Segregation distortion (SD) is defined as the deviation of genetic segregation ratios from their expected Mendelian fraction (Lyttle 1991). Chi-square tests of the number of markers mapped to each linkage group indicated a significant deviation from that anticipated based upon linkage group length. The RILs can be classified as three categories according their genotypes: families with same parental allele frequency (201: 21.22 %), families with dominant maternal allele frequency (605: 63.88 %), and families with dominant paternal allele frequency (141: 14.89). Among total of 947 markers, 746 markers loci (78.78 %) were deviated highly from the 1:1 Mendelian segregation ratio in the progeny mapping population (Table 4.6)

Table 4.6. Numbers and Percentages of DArT and SSR markers with different level of segregation distortion in Gerek x Arrehane map

Number of DArTs	%	χ^2	Significance level
60	8.0	2.75-5.97	*
154	20.6	3.85-9.07	**
50	6.7	6.69-10.17	***
115	15.4	8-13.5	****
34	4.6	11.04-14.88	*****
69	9.3	12.13-17.48	******
264	35.4	15.14-42.75	*******
Total 746	100.0		

Segregation distorted markers were distributed on all three genomes spanning to all 21 chromosomes. Segregation distortion has been reported in linkage maps

using DArT markers (References). It is known that genomic representations obtained with *PstI* reflect the methylation status of the genomic DNA and produce markers preferentially mapping in the hypomethylated gene-rich regions (Van Os et al. 2006; Mantavoni et al. 2008).

The phenomenon of SD and its causes had been poorly understood (Jenczewski et al. 1997). A single or a combination of different mechanisms may be responsible for SD in any particular case. SD has been frequently detected in polyploid species such as hexaploid and tetraploid wheat mapping populations. Deviations from Mendelian segregation ratios were widespread in plants. Deviations from Mendelian segregation ratios had been reported in all plants including several *Triticeae* species (Heun et al., 1991; Liu and Tsunewaki, 1991; Blanco et al., 1998; Nachit et al., 2001, Elouafi et al., 2001). In our study, most of the distorted loci (63.88 %) skewed towards maternal parent (Gerek), which implied that segregation distortion was much more frequent in the female than in the male parent. This is in agreement with the hypothesis that megasporocytes are more tolerant of genetic imbalance than microsporocytes, suggesting the occurrence of some form of selection at the female gamete level. The deviation of markers from expected segregation ratios seemed related to passage through male or female gametes rather than depending on a specific genotype.

Genetic control of segregation disturbance has been reported in rice (Xu et al. 1997). Castro et al. (2011) reported segregated distorted loci located on linkage group 4 of the chickpea genetic map, pointing towards those genetic factors responsible for segregating distortion. Combining ability of the two diverse parents might be one of the most probable reasons of the segregation distortion. Eujayl et al. (1997) also concluded that segregation distortion was not because of the marker technique but rather because of a segregation distortion of the gametes or zygotes leading to the F2 progenies. The apparent preferential transmission of one parental genotype towards some areas of the genome suggests that these areas may carry genes that effect gamete viability.

The phenomenon of segregation distortion can be an important limitation in map construction as it may affect both the establishment of linkage groups and

estimation of recombination frequencies. In this study, first of all we eliminated markers showing segregation distortion and constructed the linkage groups with only solid markers following the 1:1 Mendelian segregation order. Total 201 markers with normal segregation were used in preliminary linkage map construction. Later we subsequently added the distorted markers with low level of segregation disorder. When distorted markers were integrated into the map framework, their introduction did not affect the previous statistical confidence of marker order, and distributed along the linkage groups with normally segregating loci. At the end we included whole set of markers. Our result was consistent with that of Rosa et al. (2003), who also illustrated that the inclusion of skewed markers did not affect the linkage arrangement. Chen et al (2011) also found significant segregation disorder in kenaf (*Hibiscus cannabinus* L.) using ISSR, SRAP and RAPD markers and reported that inclusion of distorted loci did not cause any negative effect on the placement of normal markers as well as on linkage groups. Anhalt et al. (2008) reported that it is questionable if segregation distorted markers in a mapping population can be ignored for further analysis or can be eliminated from further calculation. These markers distort the distances of genetic markers on a map and can lead to underestimation of required markers number for fine mapping studies. However segregation disorder in our population was higher than other plant species as well as durum and bread wheat (References). However, in the ryegrass (*Lolium perenne*) 'VrnA' F2 mapping population (Jensen et al. 2005a) 60% of the marker loci showed segregation distortion.

Segregation distortion is most commonly observed in interspecific crosses; however, previous studies showed that distortion phenomenon also occurs in intraspecific wheat crosses (References). While segregation distortion is a common phenomenon in different types of mapping populations, for example F2, RILs or doubled haploids (DHs), RIL populations have the highest potential for such distortions due to repeated generations of selection forces (Singh et al. 2007), which can be accentuated by loss of vigor with enforced inbreeding. According to Anhalt et al (2008), population structure seems to be important factor for segregation disorder and can lead to variation in the proportion of distorted markers. Xu et al. (1997)

published a study based on six genetic linkage maps of rice with different population structure and reported that recombinant inbred lines (RILs) had highest frequency of markers having segregation disorder. Lu et al. (2002) made similar observation and described higher SD in RIL population than in doubled haploid (DH), backcross, and F2 population. An explanation of SD in RIL population could be inbreeding depression because of an increase of homozygote genotypes over heterozygote. All of the above mentioned researchers indicated that SD most likely accumulates along with additional generation of meiosis in an inbreeding context. Further, Nachit et al. (2001) reported that selective survival of RILs due to the single-seed descend method could be one of the possible cause of the segregation distortion in the RIL population. These segregation distortions may also be due to chromosomal rearrangements (Tanksley, 1984); alleles inducing gametic or zygotic selection (Nakagarha, 1986), reproductive differences between the two parents (Foolad et al., 1995), lethal genes (Blanco et al., 1998), sterility induced by the distant genetic parental background. Moreover the diverse genetic background could be one of the possible reasons for segregation disorder, as Gerek and Arrehane are genetically distant parents, and segregating progeny were also derived from cross of spring and winter wheat varieties.

4.1.5. Map results and Comparison with published literature

The position of 827 DArT and 39 SSR marker loci mapped in the present study was compared with the already published maps of bread and durum wheat. Although there are several published maps for both tetraploid and hexaploid wheat (Röder et al. 1998; Gupta et al. 2002.Pailllard et al. 2003; Nachit et al. 2001; Eloufi and Nachit 2004; Somer et al. 2004; Song et al. 2005; Akbari et al. 2005; Mantavoni et al. 2008), consensus bread wheat maps published by Akbari et al. (2006), Semagn et al. (2006) and Crossa et al (2007), Mantovani et al. (2008) are most comprehensive maps and were used to compare our results for DArT markers. SSR markers were referred to bread wheat consensus map published by Somer at al. 2004) and ITMI map published by Song et al. 2005 sa well as bread wheat map of

Zhang et al. (2008). A total of 150 DArT out of 827 DArT markers and all SSR markers on Gerek x Arrehane map were present on one or more of the already mentioned bread and durum wheat maps. Below map results and position of the DArT and SSR locus on each chromosome is discussed separately. Linkage groups of each chromosome and as well as homologous groups are shown in Figure 4.7-4.13.

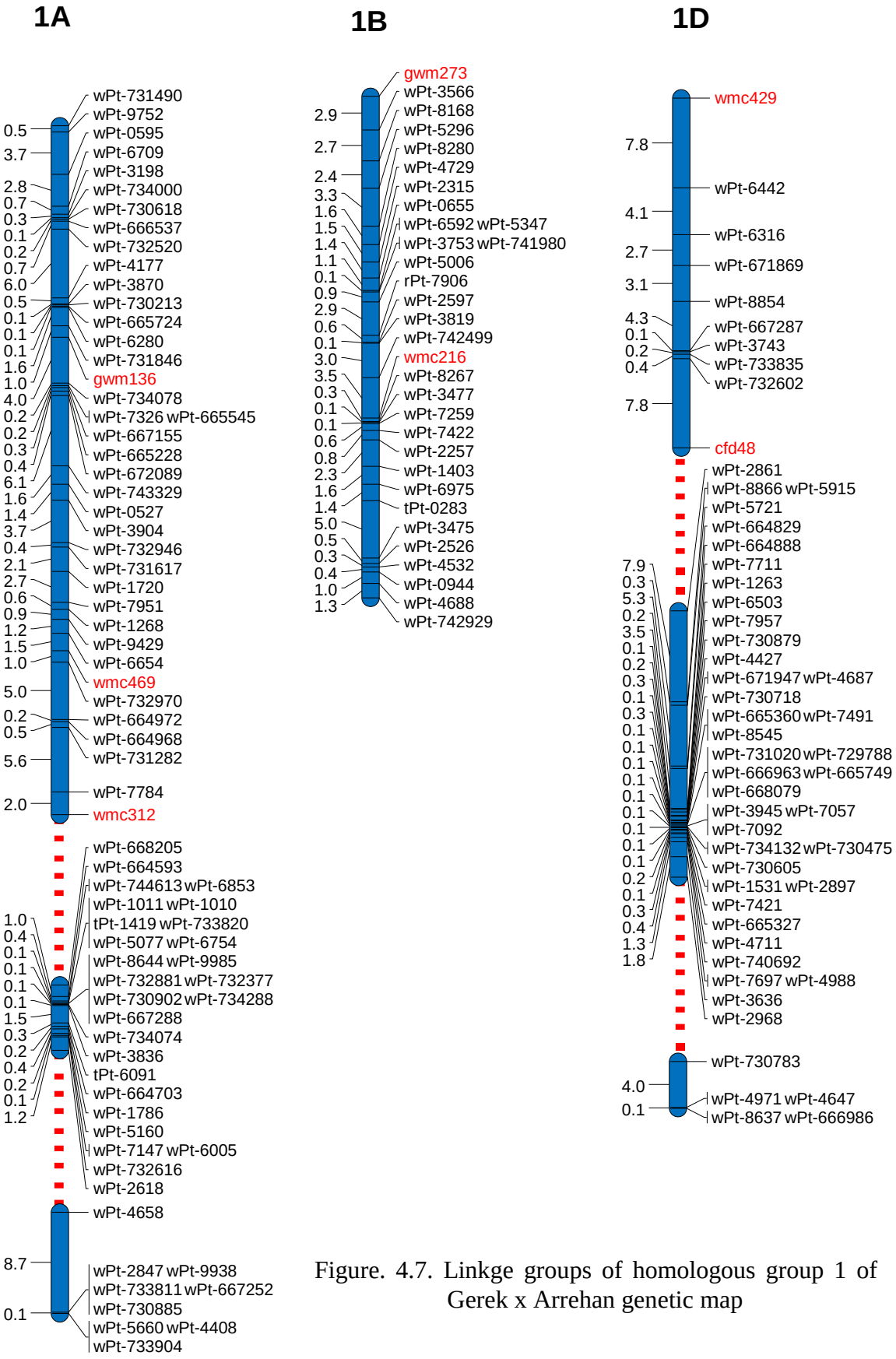

Figure. 4.7. Linkge groups of homologous group 1 of Gerek x Arrehan genetic map

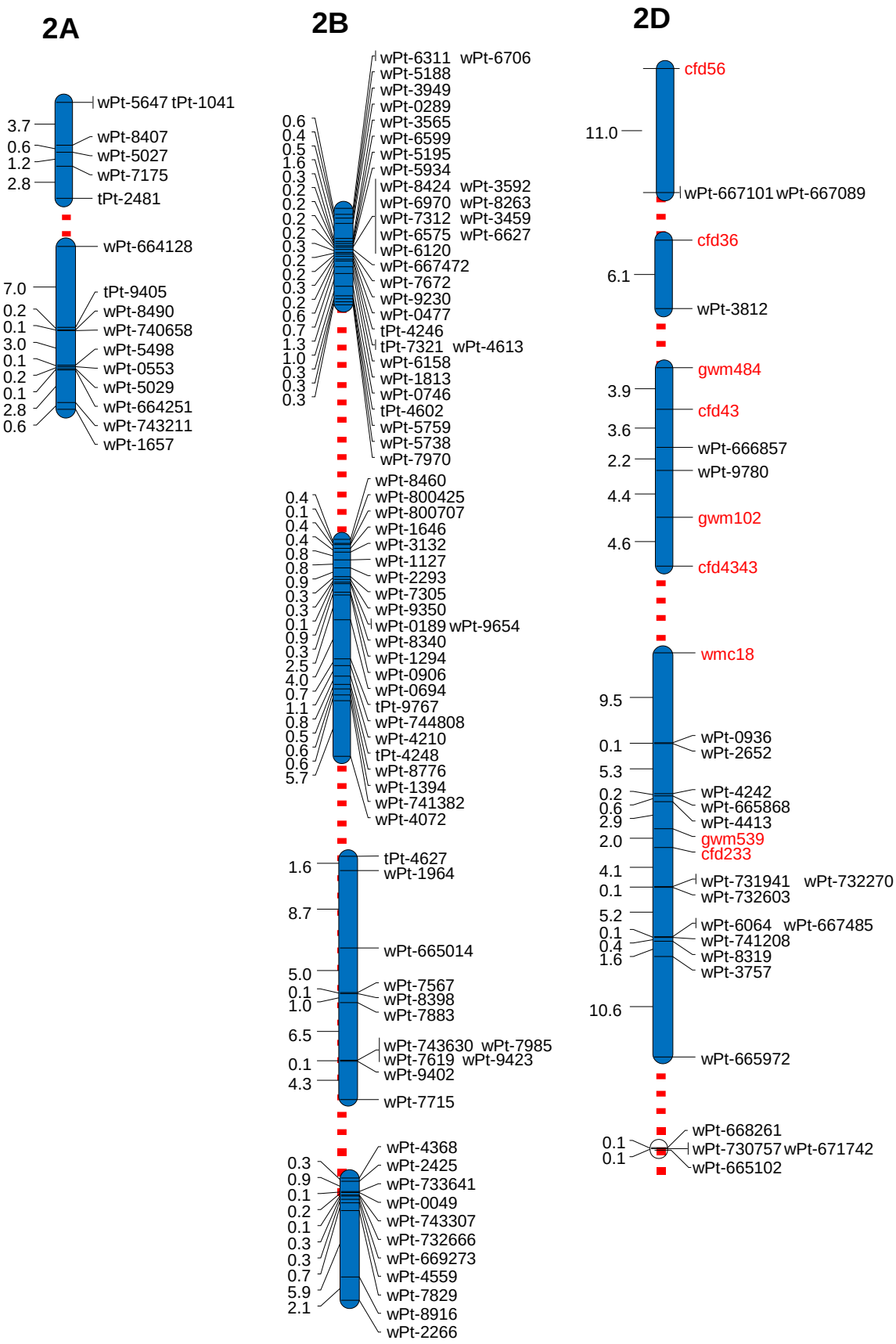

Figure. 4.8. Linkge groups of homologous group 2 of Gerek x Arrehan genetic map

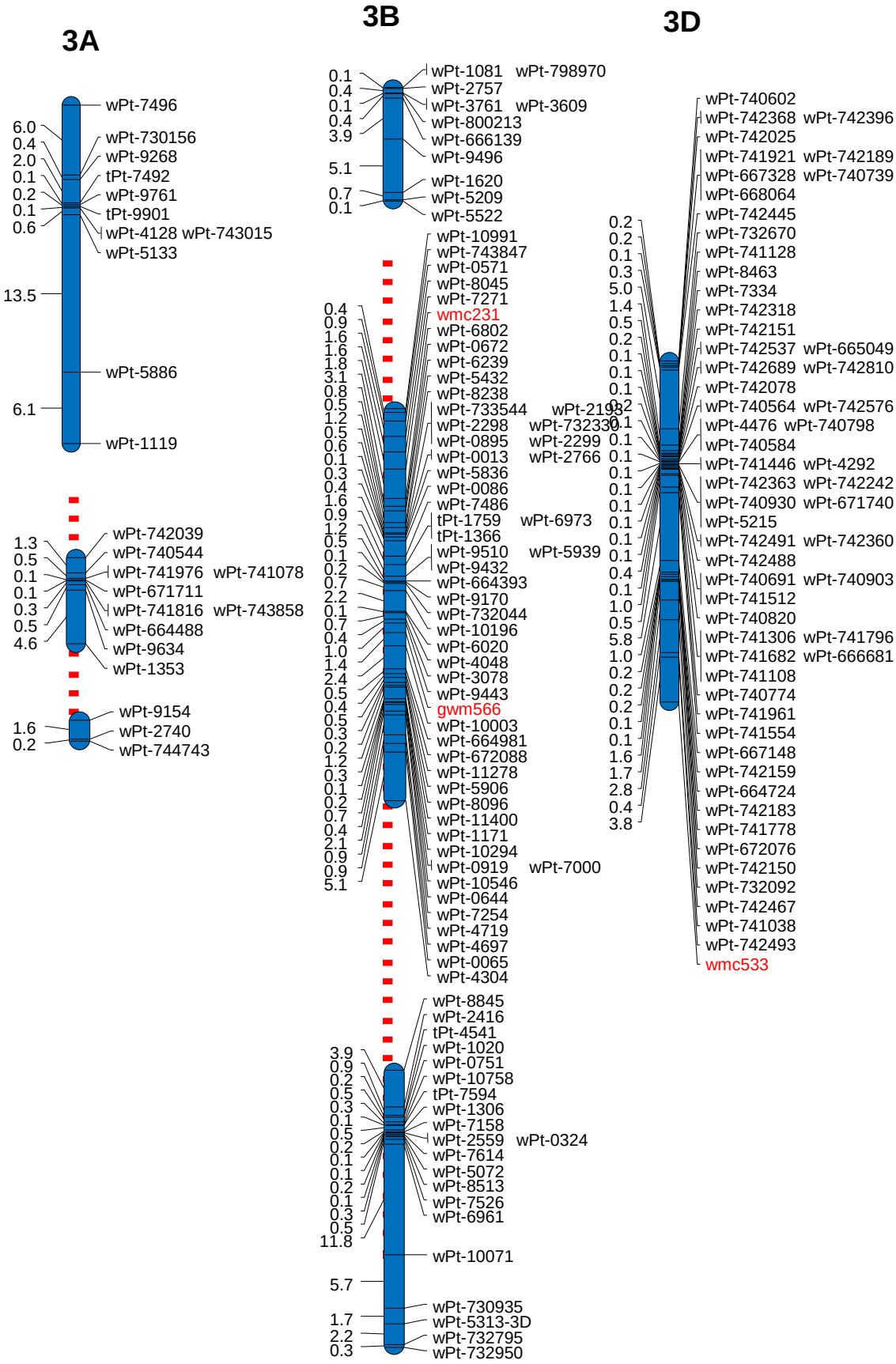

Figure. 4.9. Linkge groups of homologous group 3 of Gerek x Arrehan genetic map

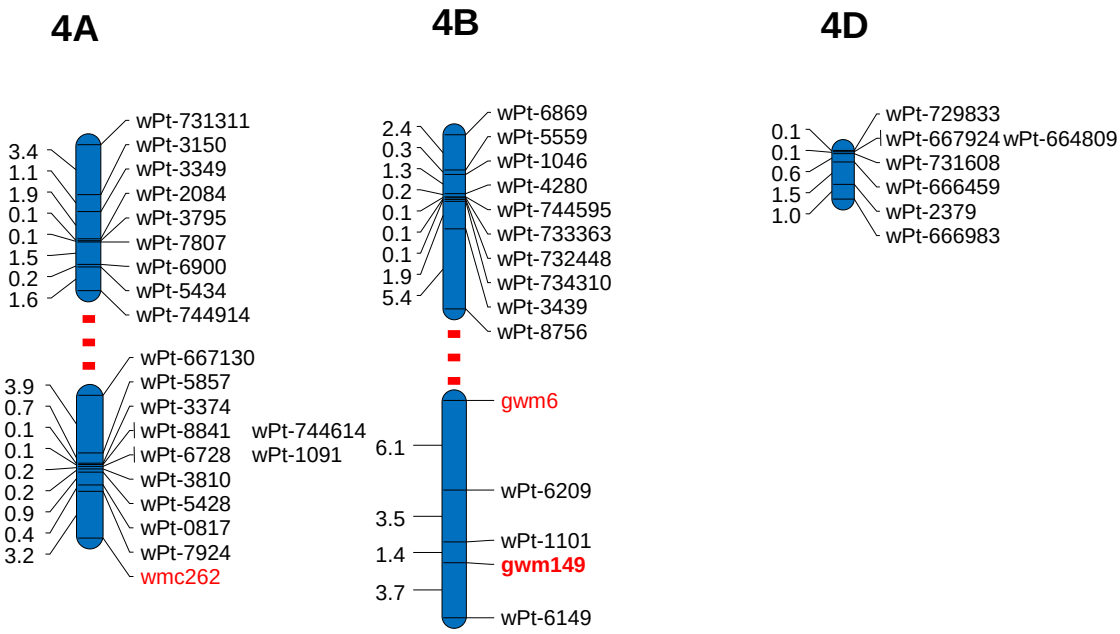

Figure. 4.10. Linkge groups of homologous group 4 of Gerek x Arrehan genetic map

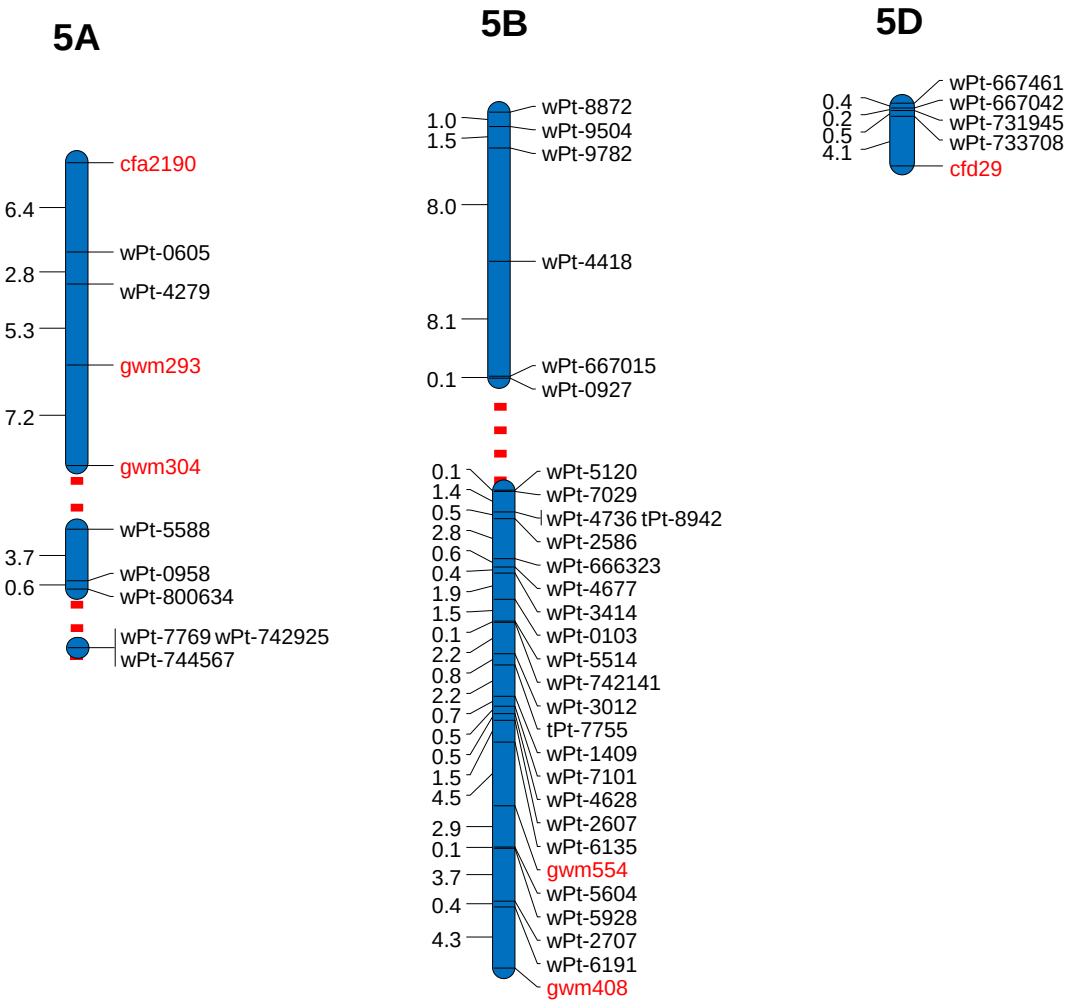

Figure. 4.11. Linkge groups of homologous group 5 of Gerek x Arrehan genetic map

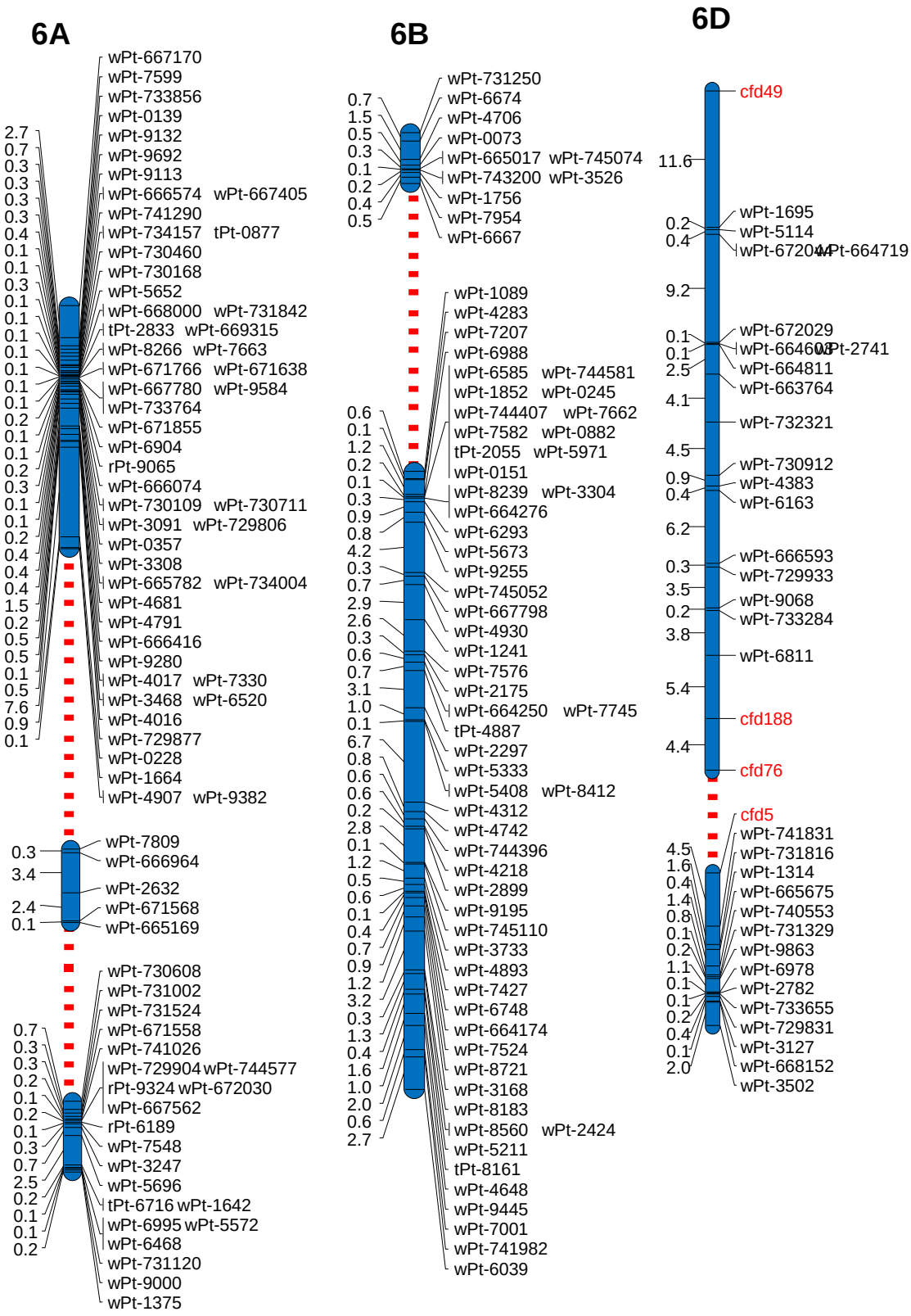

Figure. 4.12. Linkge groups of homologous group 6 of Gerek x Arrehan genetic map

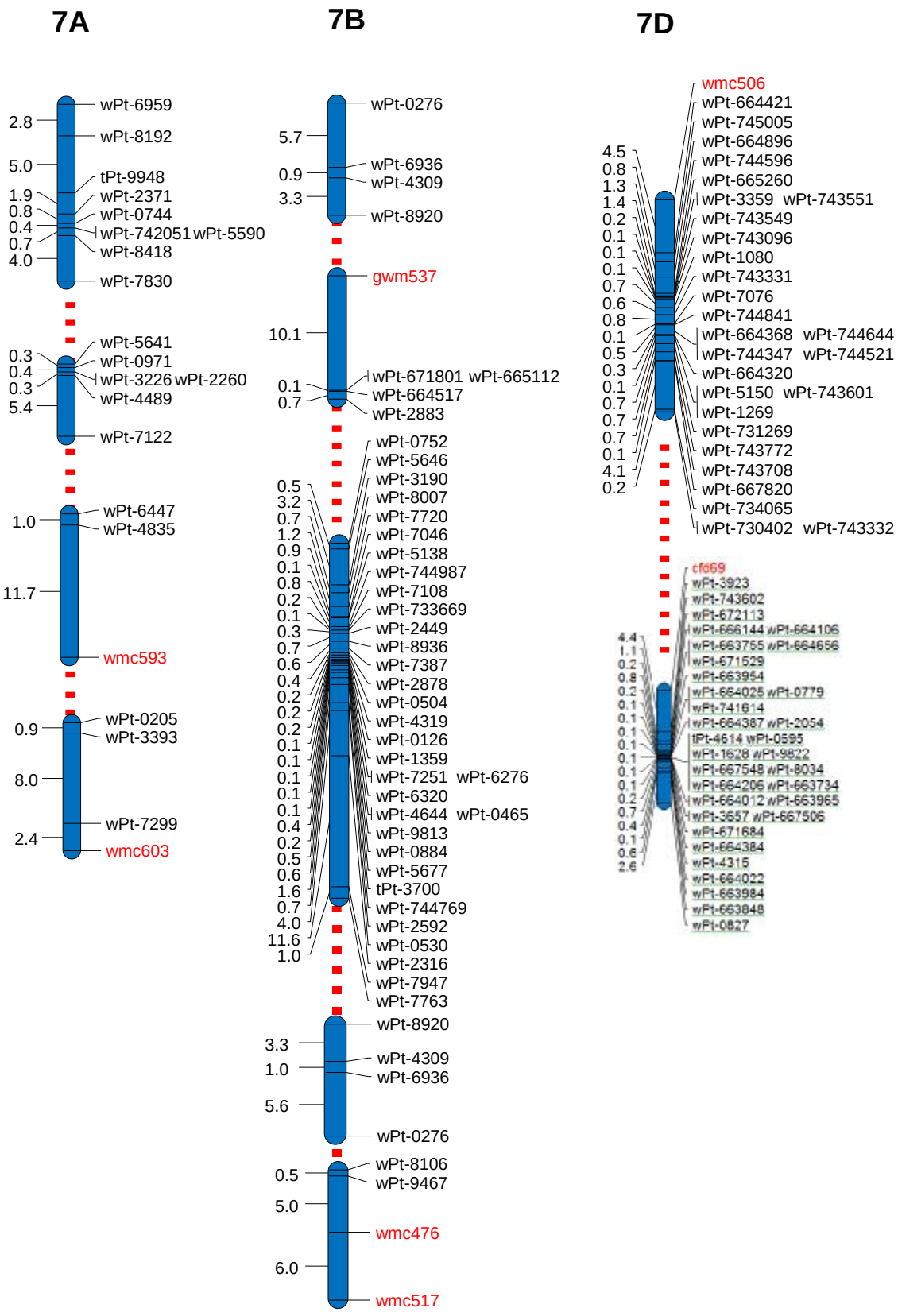

Figure. 4.13. Linkge groups of homologous group 7 of Gerek x Arrehan genetic map

Chromosome 1A: Chromosomes was constructed with total 75 markers (8.66 % polymorphism) anchored with 3 SSRs markers, with 0.993cM per marker. . 6 DArT markers were similar with markers on IA genetic linkage map of Akbari et al. (2005). All of the markers were in good order agreement with the chromosomal location provided by Triticart Company and with Akbari et al. (2005), except one marker order, wPt-8644 which is placed after wPt-6754. Three DArT markers (wPT-9752, wPT-3879 and wPT -4658) were matched and their order was in good agreement with Mantavoni et al. (2008). Three SSR markers mapped on 1A chromosomes were in agreement with order of markers published in consensus map of Somer et al. (2004) and, ITMI map (Song et al. 2005).

1- **Chromosome 1B**: 1B chromosome contained total 32 markers with polymorphism of 3.7 % of the total mapped markers and 1.366 cM per marker. There was only two SSR markers "gwm273" according to its published location (Somer et al. 2004; Song et al. 2005) mapped on 1B. All of the DArT markers were in accordance to the chromosomal location provided by Triticart. Four DArT markers were matched with Akbari et al. (2005), and 6 markers were shared by Mantavoni et al. (2008). Order of the two DArT marker pairs wPt-8267 and wPt-2315 and wPt-1403 and wPt-6975 were inverted in our study compared with Akbari et al. (2005). However order of later markers (wPt-1403 and wPt-6975) was in accordance with Mantavoni et al. (2008). Order of 4 out of 6 shared markers with Mantavoni et al. (2008) was in accordance with our order, except two markers wPt-3753 and wPt-8168 showed inversion in Gerek x Arrehane map. One marker, wPt-742929, with reported chromosomal location either on 2B/6B was mapped on 1B in the present map.

2- **Chromosome 1D:** Chromosome 1D presented 1.07cM map length per marker with total of 54 markers (6.24 % polymorphism).This chromosome has 3 linkage groups. Two SSR markers were mapped on 1D. The order of

SSR markers showed good agreement with Somer et al. (2004). Eight DArT markers were matched with Akbari et al. (2005), however several rearrangements for DArT markers were noticed on 1D. Four DArT markers, wPt-7092, wPt-1531, wPt-2897 and wPt-7697, were skipped from their order as described by Akbari et al. (2005). An inversion between markers wPt-1531 & wPt-2897 was noticed. Position of the markers were consistent with Triticart, however two markers, wPt-8637 and wPt-2861, were mapped on 1D instead of 5B and 1B (Triticart company).

3- **Chromosome 2A:** This chromosome represent lose structure with only 16 markers, on two linkage groups, with marker density of 1.40cM per marker. There was no SSR marker on this chromosome. Only one DArT marker was similar with Akbari et al. (2005). One DArT marker, wPt-740658 of unknown chromosomal location, was mapped on 2A. Therefore order of the markers was not compared due to lack of common markers with the published literature (Akbari et al. 2005; Semagn et al. 2006; Mantavoni et al. 2008; Peleg et al. 2008).

4- **Chromosome 2B:** This chromosome was among the most polymorphic chromosomes with total 78 markers and marker density of 0.903cM/marker. 2B chromosome also showed some rearrangements compared with Akbari et al. (2005), Mantavoni et al. (2008) and Semagn et al. (2006). Six and two DArT markers were common with Akbari et al. (2005) and with Mantavoni et al. (2008), respectively. However order of the markers showed some contradiction such as wPt-5738 was mapped after wPt-6311 and wPt-5188, and in addition, later two markers were inverted in Gerek x Arrehane map. wPt-8560& wPt-3132 showed inversion, however order of wPt-8560 and wPt-9350 was in accordance with Mantavoni et al. (2008). Two DArT markers, wPt-7567 (1A) and wPt-9423 (2B/6B), were mapped on 2B in contradiction with chromosomal location provided by Triticart.

5- **Chromosome 2D:** The length of map on the chromosome 2D was 78.45cM containing 32 marker, with average marker density of 2.459cM/marker. Highest numbers of SSR markers (9 SSRs) were mapped on 2D on 5 linkage groups belonging to 2D chromosome. Order and position of SSR markers was compared with consensus map of Somer et al. (2004) and with ITMI map (Song et al. 2004), and best possible order was adjusted for 5 linkage groups belonging to 2D chromosome. Only two DArT markers were common with Akbari et al. (2005) and their order was in accordance. One DArT marker, wPt-665972, was mapped on 2D other than its in the published literature reported chromosomal location of 2A.

6- **Chromosome 3A:** This chromosome harbored 24 markers spanning to 28.99cM with marker density of 1.591cM/marker. No SSR was mapped on 3A chromosome. All of the DART markers mapped on 3A were in accordance to their chromosomal location of Triticart. However the order of the DArT markers could not be compared because only one DArT marker, wPt-5133, was in common with integrated SSR-DArT durum wheat linkage map (Mantavoni et al. 2008).

7- **Chromosome 3B:** Chromosome 3B was well saturated and enjoying the highest polymorphism with 87 markers (10.05% polymorphism), with three linkage groups. This chromosome constituted 2 SSR and 85 DArT markers, with map length 81.367cM (0.935cM per marker). Eight DArT markers were reported by Mantavoni et al. (2008), where 2 and 1 markers were in common with Semagn et al. (2006) and Akbari et al. (2005).Four common DArT markers were placed on the expected order, whereas two marker pairs (wPt-9510-wPt-9432, and wPt-7614-wPt-5072) were inverted according to Mantavoni et al. (2008). Three DArT markers, wPt-732795, wPt-732950 and wPt-5313, were mapped on 3B instead of 3D chromosomes according the location of markers provided by Triticart. One DArT marker, wPt-7486, mapped on 3B in Gerek x Arrehane instead of 6B chromosome reported by Mantavoni et al. (2008).

8- **Chromosome 3D:** On 3D, 60 markers were mapped including 1 SSR markers with map length of 28.416cM. 3D was among most saturated chromosomes having a marker density of 0.47cM/marker, and consisted of only one linkage group. SSR "wmc533" was mapped on 3D chromosome as in previously published maps (Somer et al. 2004; Song et al 2005). 19 DArT markers with unknown chromosomal location were also grouped on 3D. None of the DArT markers were in common with the previously published DArT linkage maps (Mantavoni et al. 2008; Semagn et al. 2006; Akbari et al. 2005).

9- **Chromosome 4A:** 4A consisted of relatively low number of markers (21 markers including one SSR) on 2 linkage groups. SSR marker "wmc233" was on its previously known chromosomal location (Somer et al. 2004; Song et al 2005). Chromosomal locations of all DArT markers were in agreement with their previously known chromosomal locations (Triticart). Three and four DArT markers were in common with Semagn et al. (2006) and Akbari et al. (2005), respectively. This chromosome showed full agreement of DArT order with the above mentioned authors.

10- **Chromosome 4B:** Chromosome 4B was constructed with only 16 markers and two linkage groups. Map length on the 4B chromosome was 26.53cM with a marker density of 1.658cM/marker. Two SSR markers, gwm6 and gwm149, were in agreement with consensus map of bread wheat (Somer et al. 2004). Three DArT markers matched with previous maps (Akbari et al. 2005; Semagn et al. 2006). Two DArT markers (wPt-6209-wPt-6149) showed inversion according to Semagn et al. (2006), however they were in full agreement with Akbari et al. 2005. One DArT marker, wPt-3439, was placed on 4B chromosome in Gerek x Arrehane map, whereas it was mapped on 5B by Akbari et al. (2005). One DArT marker, wPt-6869, previously located on 7B (Akbari et al. 2005; Mantavoni et al. 2008) was mapped on 4B in Gerek x Arrehane map (Figure 4.4).

11- **Chromosome 4D:** 4D chromosome was also among the least polymorphic unsaturated chromosomes with total 7 markers and map length of 3.252cM. All of the seven DArT markers were tightly linked with marker density of 0.465 cM/ marker. Only one DArT marker, wPt-2349, with chromosomal position on 4D was mapped whereas rests of the six markers with unknown chromosomal location were linked with wPt-2349. None of the markers on 4D chromosome were in common with the previously published bread wheat DArT maps (Akbari et al. 2005; Semagn et al. 2006).

12- **Chromosome 5A:** chromosome 5A was constructed with three SSR markers and eight DArT markers and consisted of three linkage groups. SSR markers "cfa2190 and gwm294" were mapped at 14.45cM distance and were separated with two DArT markers. In microsatellite consensus map of bread wheat cfa2190 and gwm294 were flanked with each other with 7cM distance. Order of the three SSR markers in Gerek x Arrehane map was in full agreement with microsatellite consensus map of bread wheat (Somer et al. 2004) and ITMI map (song et al. 2005). There was no common DArT marker with earlier bread and durum wheat DArT linkage maps (Akbari et al. 2005; Semgn et al. 2006; Mantavoni et al. 2008).

13- **Chromosome 5B:** Total 30 markers including 2 SSR markers and 28 DArT markers clustered on 5B chromosome. SSR loci "gwm554 and gwm408 were mapped on 11cM distance in Gerek x Arrehane map in contrast to 31cM in bread wheat consensus map (Somer et al. 2004). Order of the SSR markers was in agreement with previous maps (Somer et al. 2004; Song et al. 2005). Only three DArT markers, wPt-2586, wPt-7101 and wPt-4628, were in common with Akbari et al. (2005). Order of the markers was in agreement except one small inversion was found between wPt-7101 and wPt-4628 in Gerek x Arrehane map (Fig4.5). One DArT marker, wPt-0103, was mapped on 5B instead of 7B repoted by Semagn et al. (2006)

14- **Chromosome 5D:** Chromosome 5D was the linkage group with least marker coverage with total five markers including one SSR marker and total 5.16cM map length having a marker density of 0.576cM/marker. One SSR marker "cfd29" with previously known chromosomal location at 5D (Somer et al. 2004; Song et al. 2005; Semagn et al. 2006) was anchored with 4 DArT markers with previously unknown chromosomal location.

15- **Chromosome 6A:** This chromosome presented 79 DArT markers with no SSR marker on three linkage groups. Chromosome 6A was well saturated with a marker density of 0.406cM/marker and total map length of 32.046cM. The all of the markers on 6A chromosome were according to their previously known chromosomal location (Triticart). There was several translocation-inversion rearrangements (Fig. 4.6) comparing with the DArT map of bread wheat (Akbari et al. 2005). Five DArT markers were in common with Akbari et al. (2005), and seven DArT markers were shared my Mantavoni et al. (2008). The order of the markers in Gerek x Arrehane map was not in full agreement with both these authors. Following the order with one author cause contradiction with the other, therefore best possible combination and order was established according to Mantavoni et al. 2008. Order of common DArT markers was inconsistent with Akbari et al. (2005), however seven DArT markers "wPt-4017, wPt-7330, wPt-3468,wPt-4016, wPt-3091, wPt-3247, wPt-1642, shared by DArT-SSR integrated linkage map of durum wheat,were in agreement with Mantavoni et al. (2008), except one marker wPt-3091.

16- **Chromosome 6B:** The homologous chromosome 6B was well formed with 70 DArT markers on two linkage groups with total length of 54.82cM and marker density of 0.783cM/marker. Location of the DArT markers on 6B were according to their previously known chromosomal location, however five DArT markers, Pt-7427, wPt-6748, wPt-7524, wPt-9445 and wPt-7001, with previously known chromosomal location at 4A and one DArT marker wPt-731250 with previously known chromosomal location on 6A were

mapped on 6B in Gerek x Arrehane linkage map. Eight DArT markers were in common with Mantovani et al. (2008), and their order showed agreement except wPt-7662 and wPt-5333. Five DArT markers were matched with Semagn et al. (2006), with 3 markers show order agreement. Two DArT marker, wPt-2424 and wPt-8183, were mapped on same position but in Gerek x Arrehane map wPt-2424 was mapped after wPt-8183. Only one DArT marker was common with Akbari et al. (2005) and Peleg et al. (2008)

17- **Chromosome 6D:** Chromosome 6D presented 4 SSR markers and 32 DArT markers with 70.744cM length and marker dcensity of 1.965cM/marker, and consisted of two linkage groups. One SSR marker, cfd5, was mapped on 6D chromosome instead of its in the literature reported chromosomal location at 5B (Somer et al. 2004; Song et al. 2005). The order of the rest of 3 SSR markers “cfd49, cfd188 and cfd76 were in full agreement with bread wheat consensus map (Somer et al. 2004). The entire DArT markers were mapped according to their previously known chromosomal location except wPt-663764 mapped on 6D instead of 6B chromosome (Triticart). Eleven DArT markers with unknown chromosomal location were placed on 6D (Figure 4.7). None of the DArT marker in Gerek x Arrehane linkage map was matched with the earlier DArT studies (Akbari et al. 2005; Semgn et al. 2006; Mantavoni et al. 2008; Peleg et al. 2008).

18- **Chromosome 7A:** 7A chromosome comprised with four small linkage groups and was constructed with total 22 markers including 2 SSR markers with total 46.03cM and marker density of 2.092cM/marker. Two SSR markers “wmc593 and wmc603” were mapped at genetic distance of 39cM in bread wheat consensus map (Somer et al. 2004), whereas in Gerek x Arrehane map both SSR markers were mapped on two different linkage groups (Figure 4.7), however their order was in agreement with Somer et al. (2004) and ITMI map (Song et al. 2005). Four DArT markers were common with genetic linkage map (Semagn et al. 2006) and their order was in full agreement. Two matched DArT markers “wPt-4835 &wPt-6447” with Akbari et al (2005) showed little

inversion in Gerek x Arrehane map. Similarly, two DArT markers common with Mantavoni et al. (2008) showed good order accordance.

19- **Chromosome 7B:** Total five linkage groups belonging to 7B chromosome, consisted of forty six markers (5.31% of total polymorphic markers) including 3 SSR markers. 4 out of 5 linkage groups comprised small groups containing 4 markers. Total length of the chromosome was 73.709cM with marker density of 1.602 cM per marker. Order of the three SSR markers "gwm537, wmc476 and wmc517" were in accordance with previously published microsatellite bread and durum wheat linkage maps (Somer et al. 2004; Song et al. 2005; Zhang et al. 2009). Three DArT markers "wPt-7947, wPt2316, andwPt-7763", mapped on 7A by Semgan et al. (2006) placed on 7B in Gerek x Arrehane map. Three DArT markers "wPt-671801, wPt-665112 and wPt-664517 with unknown chromosomal location were mapped on 7B in our linkage map. One (wPt-3190) and two DArTs (wPt-2883, wPt-0504) were in common and showed accordance with linkage maps published by Semagn et al. (2006) and Mantavoni et al. (2008), respectively. There was contradiction with one out of six DArTs order matched with Akbari et al. (2005). DArT marker wPt- 0884 was placed after wPt-6320, whereas Akbari et al. (2005) mapped wPt- 0884 before wPt-7108.

20- **Chromosome 7D:** Chromosome 7D was also well saturated consisting of 65 markers with marker density of 0.462 cM/marker. This chromosome was formed with two linkage groups and anchored by 2 SSRs on each linkage group. The order of two SSR markers, wmc 506 and cfd69 was in accordance with earlier SSR based linkage maps of bread wheat (Somer et al. 2004; Song et al. 2005). Only one DArT "wPt-2592" was matched with Akbari et al. (2005), therefore order of the DArT marker could not be compared with the previous published DArT maps (Akbari et al. 2005; Semagn et al. 2006; Mantavoni et al. 2008; Peleg et al 2008)

There were several rearrangements, inversion and translocation of markers in Gerek x Arrehane linkage map, compared with the previous published DArT genetic linkage maps (Akbari et al. 2005; Semagn et al. 2006; Mantavoni et al. 2008; Peleg et al 2008). Semagn et al. (2006) reported that the differences in marker orders among different genetic maps are not surprising as genetic mapping only gives an indication of relative positions of the markers to each other. Moreover the number and type of the markers is different in different published maps, which also could cause the changes in the position and order of the markers due to linkage between different markers. Mantavoni et al. (2008) also illustrated that the difference in the order of some markers is acceptable and moreover the inconsistency in the map position could explained by the presence of additional loci in the wheat genome.

In Gerek x Arrehane map, we obtained a good and uniform coverage of all A, B and D genomes; however some chromosomes were remained unsaturated with low number of markers. Some homologous groups such as group 5 particularly 5D and 5A contained only 5 and 11 markers respectively. The low density of DArTs markers in group 5 was already reported in hexaploid wheat. Akbari et al. (2006) and Semagn et al (2006) also reported only 3 markers on 5a over a total several hundred successfully mapped DArT markers. The underrepresentation of the polymorphic fragments on the group 5D, 5A and 4D in the present Gerek x Arrehane map t genome might be due to methylation-sensitive restriction enzyme such as ***PstI*** and ***Sse83871*** (Mantavoni et al. 2008)

Genetic map with full genome coverage and confidence in locus order is necessary for detection, mapping and estimation of gene effects on phenotypic traits. The present map based on a cross between Gerek x Arrehane most likely covers the majority of the wheat genome. Furthermore, this map is probably one of the most intensive published maps for hexaploid wheat, with highest number of DarT markers (827 markers). This is the first genetic map of bread wheat from Turkish as well as from gene pool from Fertile Crescent, and, therefore, it could serve as new source to identify new markers (clones) linked with unique alleles from Fertile crescent. This map provides a valuable resource for wheat genetic research, confirming the genetic location DarT markers on their relevant chromosomes, morever, around 77 DarT

markers were also mapped on different chromosomes with their unknown position, which will expand the pool of markers available for wheat research. The constructed map will be used for further QTL detection for agronomic, quality and as well as kernel physical characteristics of bread wheat, and as a tool for marker-assisted selection and map-based breeding for traits of interest. QTL analysis for agronomic and yield related traits based on this map will be described in detail in the next section of this chapter,

4.2. Phenotypic Variations

4.2.1. Frequency distribution of the agronomic traits

One hundred and fourteen recombinant inbred lines (F_6) along with their parents were grown at four locations/years/environments to get phenotypic data on different morphological, agronomic and yield related traits. One line was removed from the phenotypic data evaluation due to damage and as well as due to mixing. Totally 113 RILs were used to check phenotypic distribution of traits as well as for QTL analysis. The maximum, minimum and mean values for each studied trait along with standard deviation values for RILs as well as two parents for all locations/environments are given in the Table 4.7. For many traits, the performances of the parents “Gerek” and “Arrehane” were well within the bounds of population providing evidence of transgressive segregation. For most of the agronomic and yield related traits, Arrehane was superior while both Gerek and Arrehane are the bread wheat cultivars for stress environments.

Frequency distribution in the RILs for segregating phenotypic classes of nine important agronomic and morphological traits such as spike length, number of spikelets per spike, number of grains per spike, grain weight per spike, spike compactness, 1000 grain weight, days to heading, plant height and peduncle length are illustrated separately for each location/environment as well as mean for all environments in the figure 4.14-4.22. All these traits were measured on a quantitative scale and showed continuous variation with normal distribution. The RILs showed

transgressive segregation for all the traits showing the polygenic control. The distribution for each of the agronomic traits is separately described below.

Days to heading

In the three field trials in two years, cultivar Arrehane was 15-20 days earlier than Gerek in days to heading. The average days to heading for Gerek were 118 days whereas in case of Arrehane it was 102 days. The average days to heading for all environments varied from 97 days to 123 days with population mean of 109 days. The days to heading of the RILs displayed a continuous, but double peak line distribution with the population mean falling between the parents in all location (Table 4.7; Figure 4.14). These results suggested that RIL population showed normal distribution for days to heading and there were major loci controlling the days to heading in this population. According to the data averaged over three environments, the RIL population showed transgressive segregation for days to heading (Figure 4.14)

Plant height

Plant height of parents and RILs were evaluated based on an average of twenty plants. The two parents Gerek and Arrehane differed markedly for plant height. Gerek was a tall cultivar with an average plant height of 112 cm whereas Arrehane was a semi dwarf cultivar with an average plant height of 98 cm, with 14 cm average difference between two parents. The average plant heights for all four environments ranged from 90 to 127 cm with a population mean of 109 cm (Table 4.7). More than 70% of the RILs had plant height above or below the parental values showing high degree of transgressive segregation (as shown in Table 4.7 and Fig. 4.15).

Peduncle lenght

From the two parental cultivars, Gerek showed higher peduncle length than Arrehane. The average peduncle length of Gerek was 43.3 cm whereas mean peduncle length of Arrehane was 37.6 cm (Figure 4.16).

The average peduncle lengths of all RILs for all three locations were ranged from 36 cm to 53 cm with a population mean of 43 cm.

Spike lenght

Parental genotypic variations and distribution among RILs for spike length in four different environments as well as their means are shown in the Table 4.7 and Figure 4.17. Arrehane produced longer spikes with grand mean of 10.7 cm and Gerek produced shorter spikes with an average of 7.8 cm, across all environments. In all environments except Adıyaman 2011, the transgressive segregation for spike length occurred in the RIL population.

Number of spikelets per spike

Mean numbers of spikelets per spike among RIL population for each location was higher than the mid parent and lower than the better parent (Table 4.7). The average numbers of spikelets per spike varied between 15.9 and 19.8 for Gerek and from 19.7 to 22.6 for Arrehane. The average numbers of spikelets per spike for environments ranged from 16.6 to 22.5 with a grand population mean of 19.1. RIL population showed transgressive segregation for the number of spikelets per spike in all environments (Figure 4.18).

Spike compactness

Considerable segregation was observed for spike compactness evaluated in Gerek x Arrehane population. There were considerable differences in spike compactness. Arrehane had longer and less dense spike whereas Gerek has shorter spike but more compact spike (with high spikelet density). The average spike density ranged from 2.106 to 2.409 for Gerek and for Arrehane it varied between 1,867 to 2,213. In all four field trials the mean values for spike compactness was within the range of population showing the normal phenotypic distribution of the traits according to Mendelian segregation (as shown in Table 4.7 and Figure 4.19).

Number of grain per spike

Arrehane performed more favorably for number of grains per spike compared to Gerek in all four environments. Number of grains per spike ranged from 21.3 to 38.3 for Gerek whereas it varied 34.9 to 48.7 for Arrehane. The mean numbers of grain per spike values of the RILs ranged from 28.9 (Konya-2011) to 40.9 (Adana-2010), with grand population mean of 33.6. The frequency distributions of RIL population for the trait in different environments were different showing environmental influence on the grain number per spike (Figure 4.20). The wide range of variation of the number of spikelets per spike and normal phenotypic distribution (Figure 4.20) as well as transgressive segregation suggested polygenic inheritance of the trait.

Grain weight per Spike

Grain weight per spike is the product of all spike traits. Grain weight per spike for Arrehane was higher (range-1.797 to 2.586 g) compared to Gerek with ranging from 0.945 to 1.992. Distribution of grain weight per spike among RILs was more or less normal with values in more than 70% of the RILs above or below the mid parent and better parent except in Konya-2011 environment where around 30% of the RILs skewed towards Gerek. The overall mean for four environments for grain weight per spike also skewed towards Gerek as shown in the Figure 4.21.

Thousand grain weight

The mean 1000 grain weight of the two parental genotypes in different environments/years differed significantly, and ranged from 21.8 to 41.2 g for Gerek and from 32.4 to 50.3 for Arrehane. The mean 1000 grain weights of the individual RILs in different environments ranged 19.1 to 48.4 g for Adana 2010, from 28.8 to 50.2 g for Adana 2011, 31.4 to 53.4 g for Konya 2011, from 30.3 to 53.6 g for Adıyaman 2011. The overall grand mean of the RILs population was 38.1 g. The 1000 grain weight values for RILs in each environment gave a good fit to normal distribution and, they showed trangressive segregation in most of the environments tested (Table 4.7 and Figure 4.22).

Table 4.7. Mean values, standard deviation, maximum and minimum values of all traits evaluated in four environments in 2010 and 2011.

Traits	Environments/ Years	RILs (n= 113)				Parental Lines	
		Mean	Min.	Max.	SD	Gerek	Arrehane
Ph	Average	109	87	132	7.04	112	98
	Adana 2010	105	84	130	9.40	108	94
	Adana 2011	115	90	138	9.31	120	103
	Konya 2011	105	85	127	10.15	109	96
	Adıyaman 2011	111	90	133	8.23	114	99
Pl	Average	43	32	56	3.33	44.3	37.6
	Adana 2011	44	33	53	4.42	45.5	39.2
	Konya 2011	43	29	55	5.28	41.2	33.4
	Adıyaman 2011	44	33	60	4.27	46	40.1
Hd	Average	109	97	123	6.22	118	102
	Adana 2010	112	97	132	8.57	115	100
	Adana 2011	98	88	111	5.79	111	95
	Adıyaman 2011	118	104	128	5.61	127	112
TGW	Average	38.1	27.4	51.4	3.77	33.7	42.6
	Adana 2010	30.3	19.1	48.4	5.57	21.8	32.4
	Adana 2011	40.2	28.8	50.2	4.32	38.2	46.9
	Konya 2011	42.8	31.4	53.4	4.19	41.2	50.3
	Adıyaman 2011	39.3	30.3	53.6	4.62	33.5	40.9
SL	Average	9.2	7	11.8	1.14	7.8	10.7
	Adana 2010	10.4	7.9	14.2	1.40	9.4	11.7
	Adana 2011	9.6	7.2	13.7	1.36	8.4	12.1
	Konya 2011	8.3	5.4	11.2	1.19	6.6	8.9
	Adıyaman 2011	8.7	6.9	11.1	1.12	6.9	10.2
NSS	Average	19.1	15.5	22.8	1.33	17.7	21.3
	Adana 2010	20.9	17.7	24.8	1.70	19.8	22.2
	Adana 2011	20.5	16.1	24.8	1.73	19.1	22.6
	Konya 2011	16.6	13.2	19.8	1.24	15.9	19.7
	Adıyaman 2011	18.3	15.0	21.9	1.51	15.9	20.7
NGS	Average	33.6	21.3	49.8	4.76	29.0	40.6
	Adana 2010	40.9	28.2	57.1	6.62	38.3	42.9
	Adana 2011	33	18.6	49.0	6.04	28.9	48.7
	Konya 2011	28.9	17.8	47.3	6.29	21.3	34.9
	Adıyaman 2011	31.6	20.6	45.9	5.39	27.5	35.8
GWS	Average	1.338	0.668	2.079	0.201	1.223	2.169
	Adana 2010	1.330	0.610	2.095	0.325	1.992	2.586
	Adana 2011	1.372	0.702	2.085	0.292	0.992	2.397
	Konya 2011	1.290	0.581	2.127	0.331	0.945	1.797
	Adıyaman 2011	1.362	0.780	2.011	0.281	0.964	1.897
SC	Average	2.076	1.576	2.476	0.155	2.269	1.991
	Adana 2010	2.010	1.479	2.490	0.197	2.106	1.897
	Adana 2011	2.135	1.423	2.490	0.188	2.274	1.867
	Konya 2011	2.001	1.630	2.426	0.187	2.409	2.213
	Adıyaman 2011	2.103	1.774	2.495	0.160	2.304	2.029

Ph: Plant Height (cm); Pl: Peduncle Lenght (cm); Hd: Days to heading; TGW: thousand grain weight (g); SL: spike lenght (cm); NSS: Number of spiklets per spike; NGS: Number of grains per spike; GWS: grain weight per spike; SC: spike compactness; GY: grain yield; SD: standard deviations

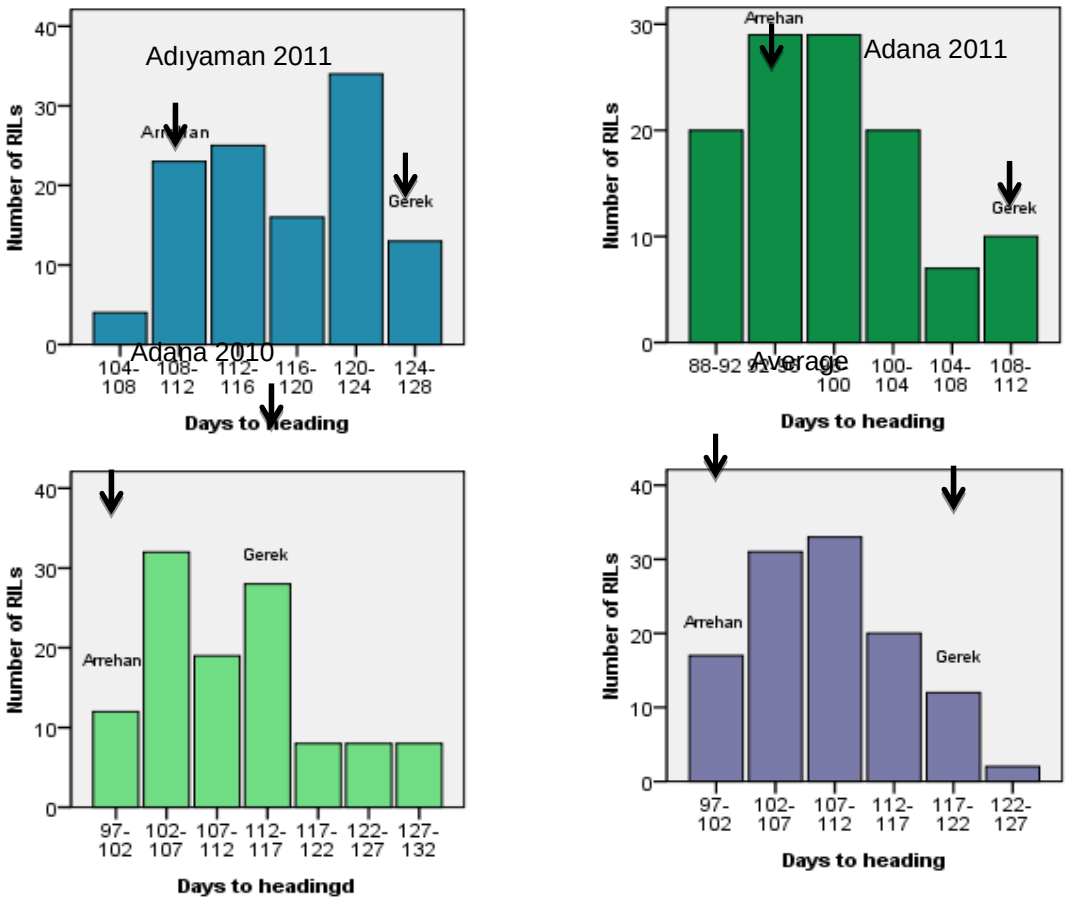

Figure: 4.14 Frequency distribution of days to heading Gerek x Arrehane mapping population evaluated in three environments

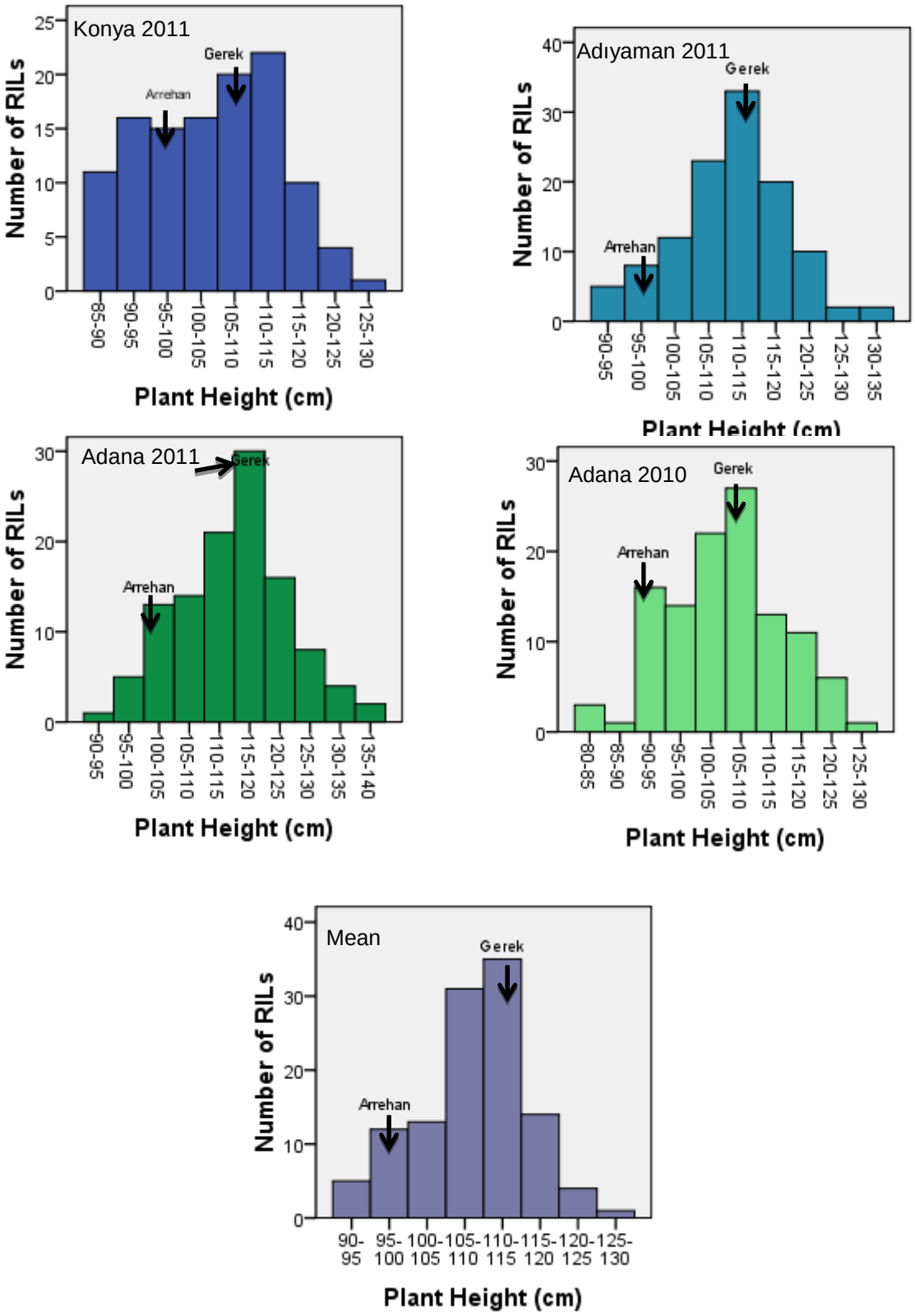

Figure: 4.15 Frequency distribution of plant height Gerek x Arrehane mapping population evaluated in four environments

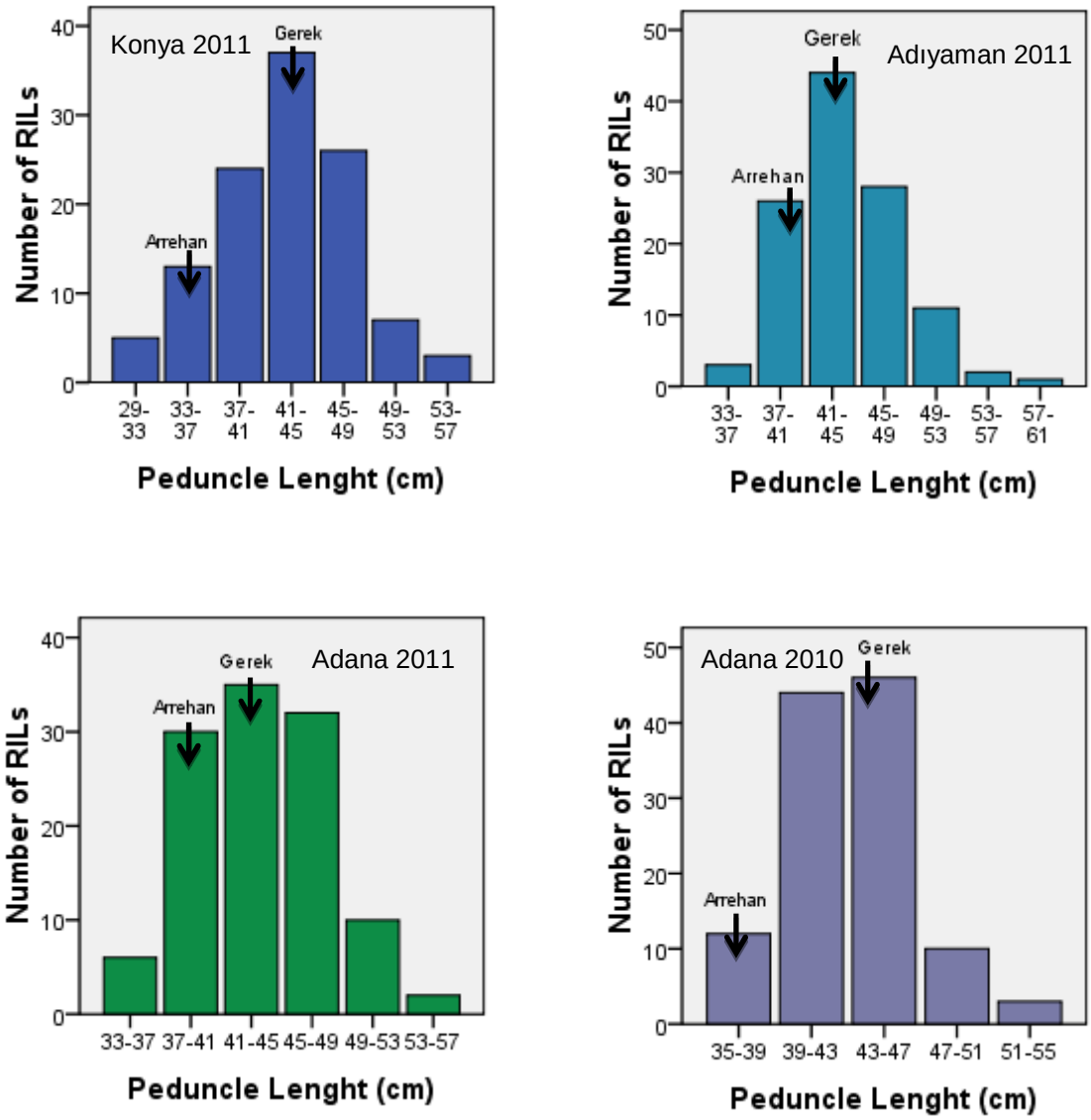

Figure: 4.16. Frequency distribution of peduncle length Gerek x Arrehane population in for environments

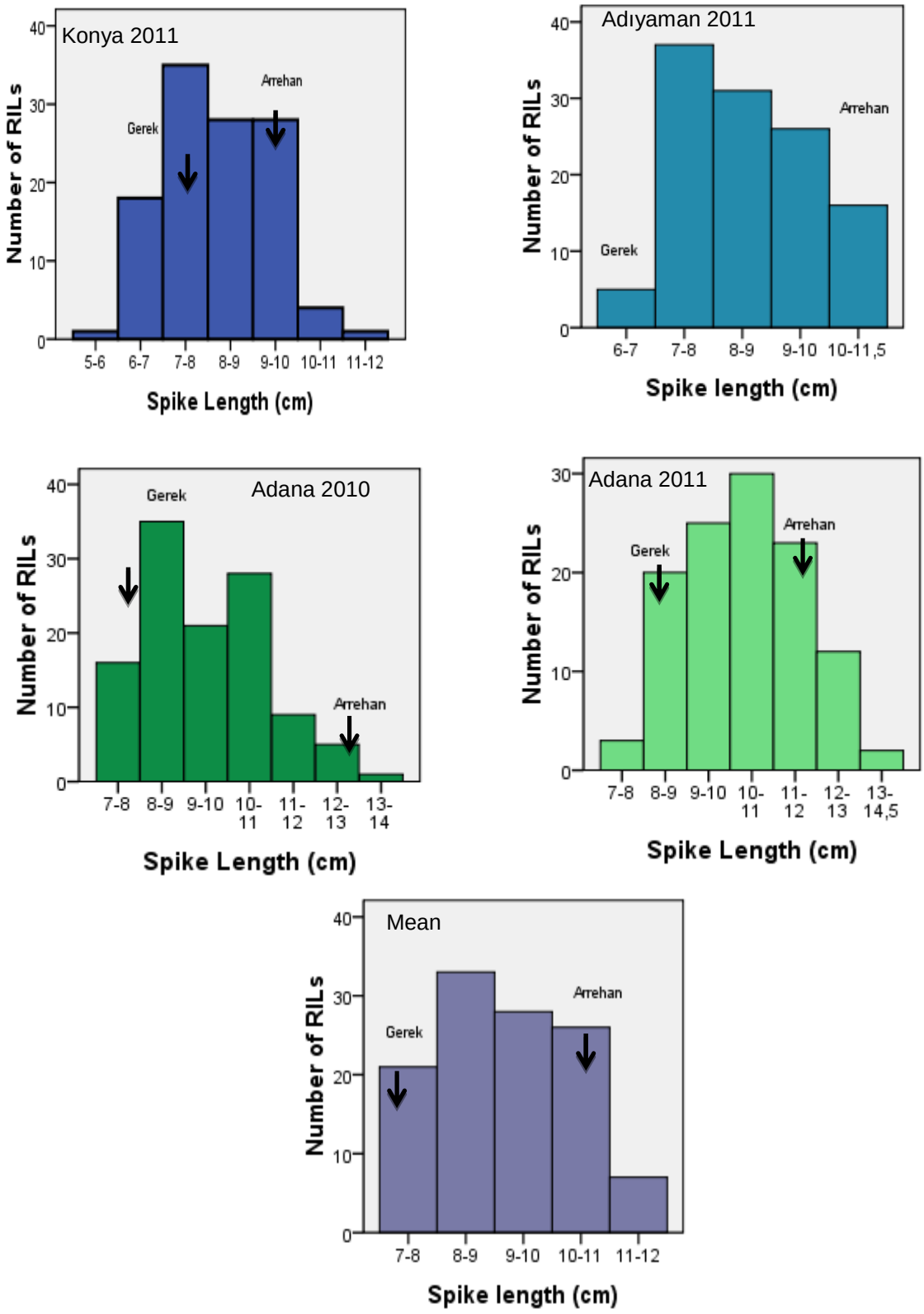

Figure: 4.17. Frequency distribution of spike length of Gerek x Arrehane mapping population evaluated in four environments

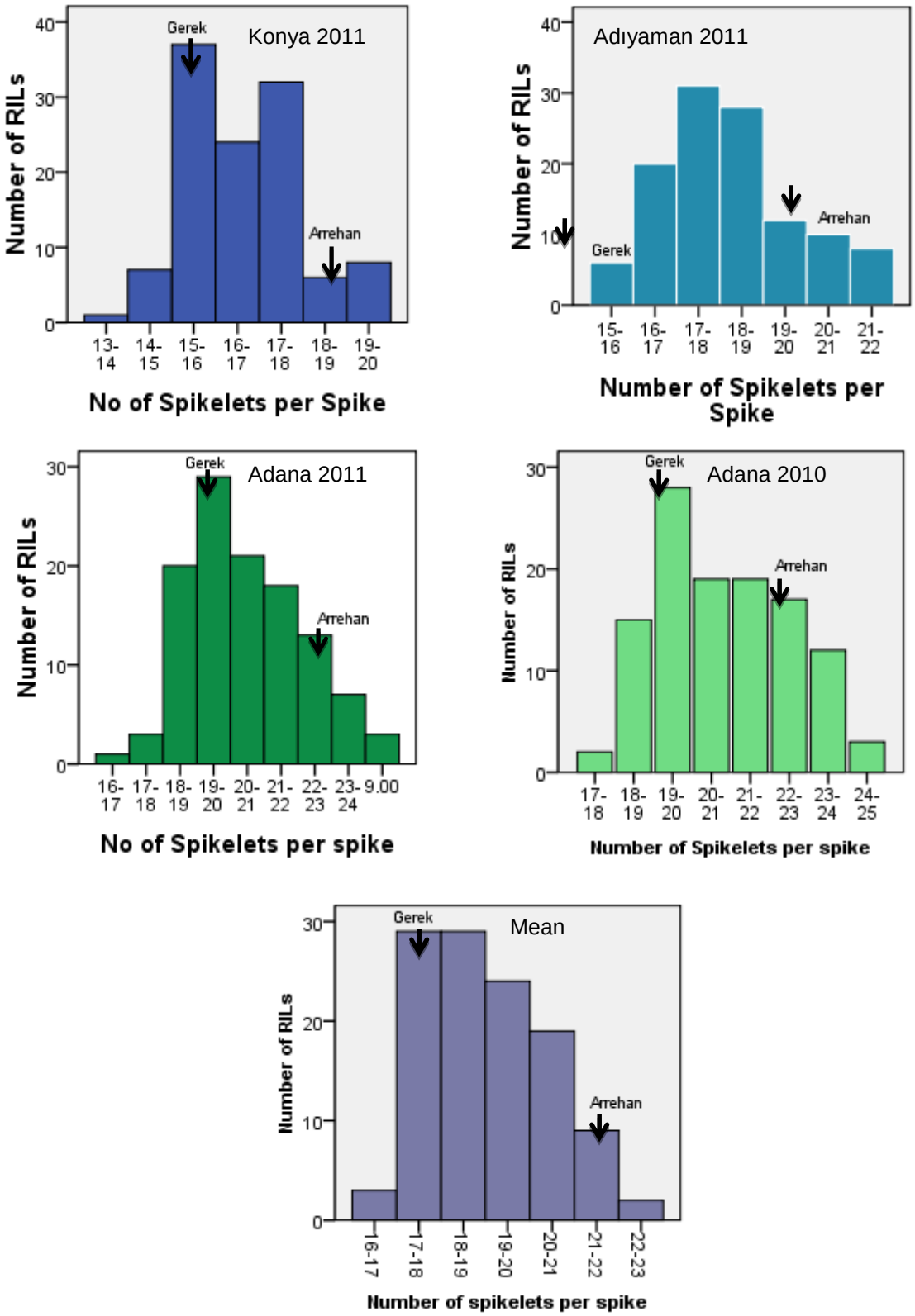

Figure: 4.18 Frequency distribution of number of spikelets per spike of Gerek x Arrehane mapping population evaluated in four environments

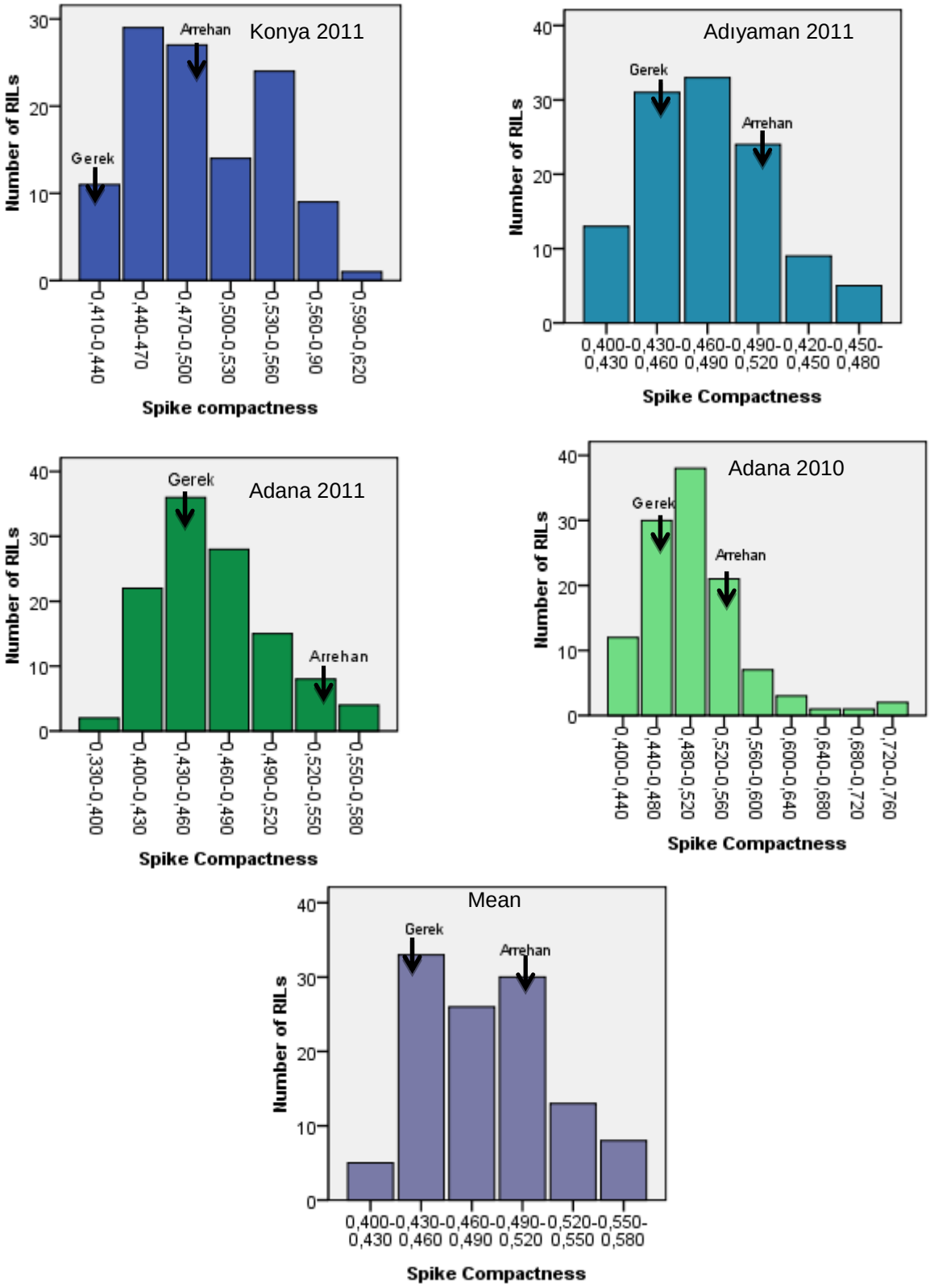

Figure: 4.19. Frequency distribution of spike compactness for Gerek x Arrehane mapping population evaluated in for environments

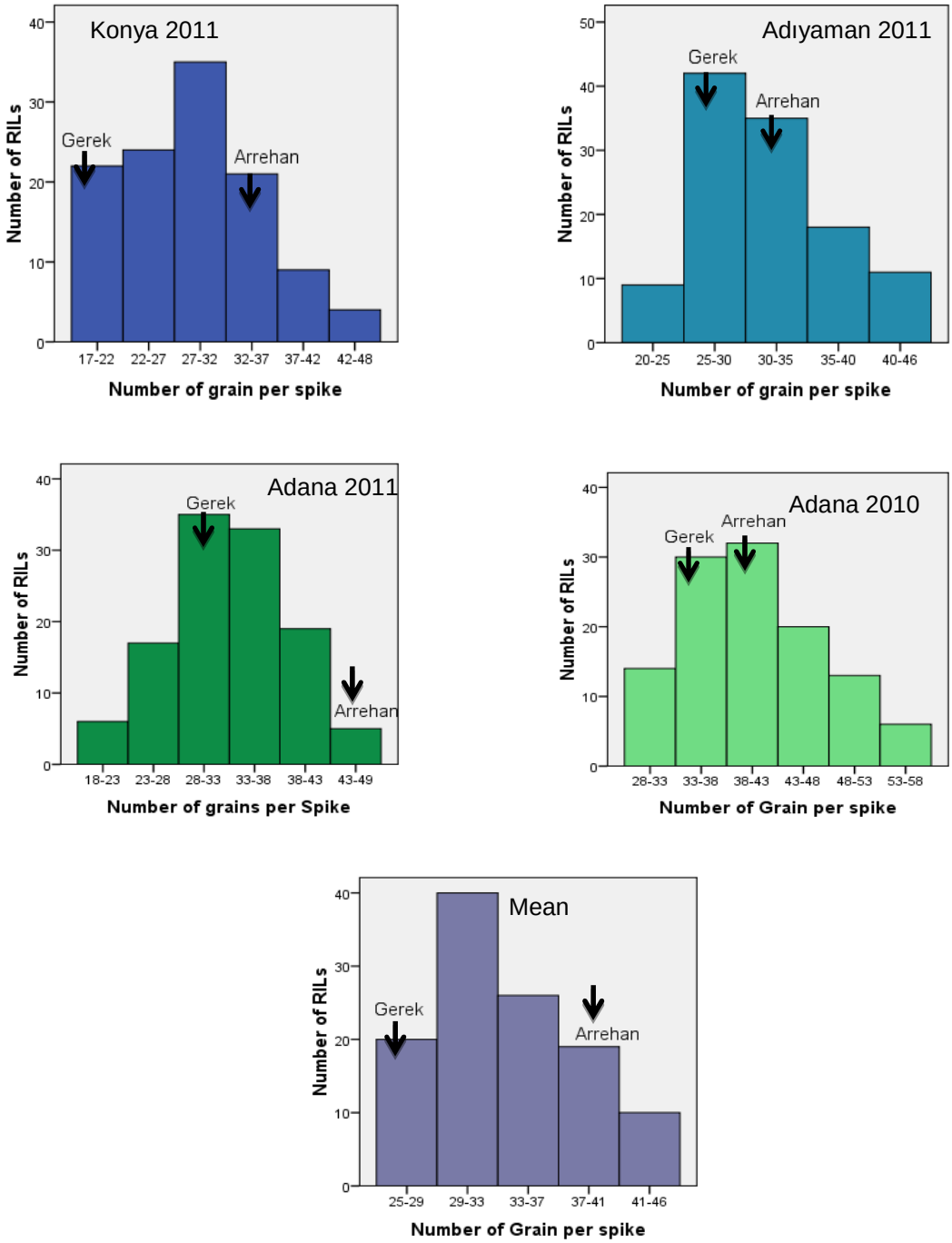

Figure: 4.20 Frequency distribution of number of grains per spike of Gerek x Arrehane mapping population evaluated in four environments

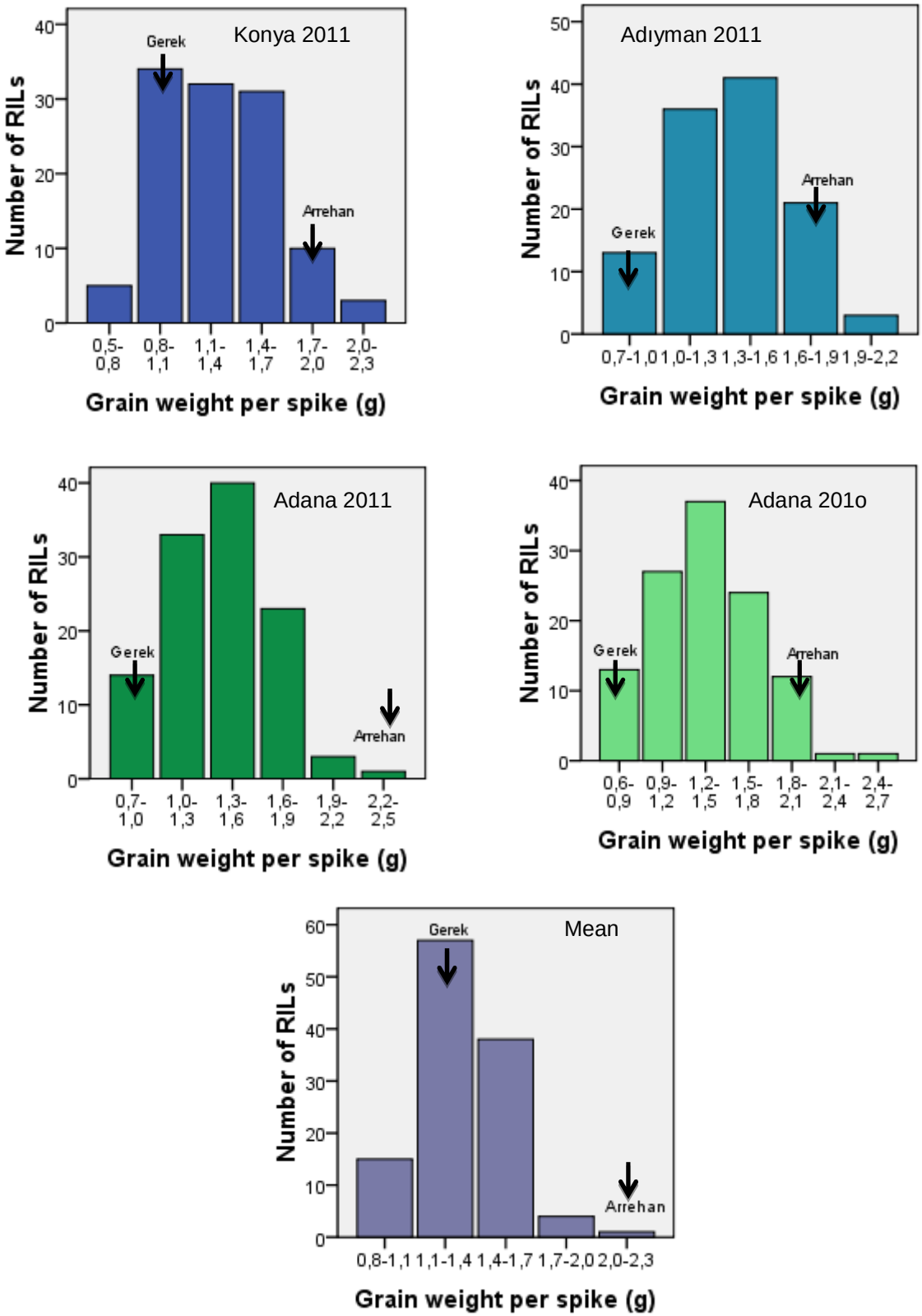

Figure: 4.21. Frequency distribution of grain weight per spike for Gerek x Arrehane mapping population evaluated in for environments

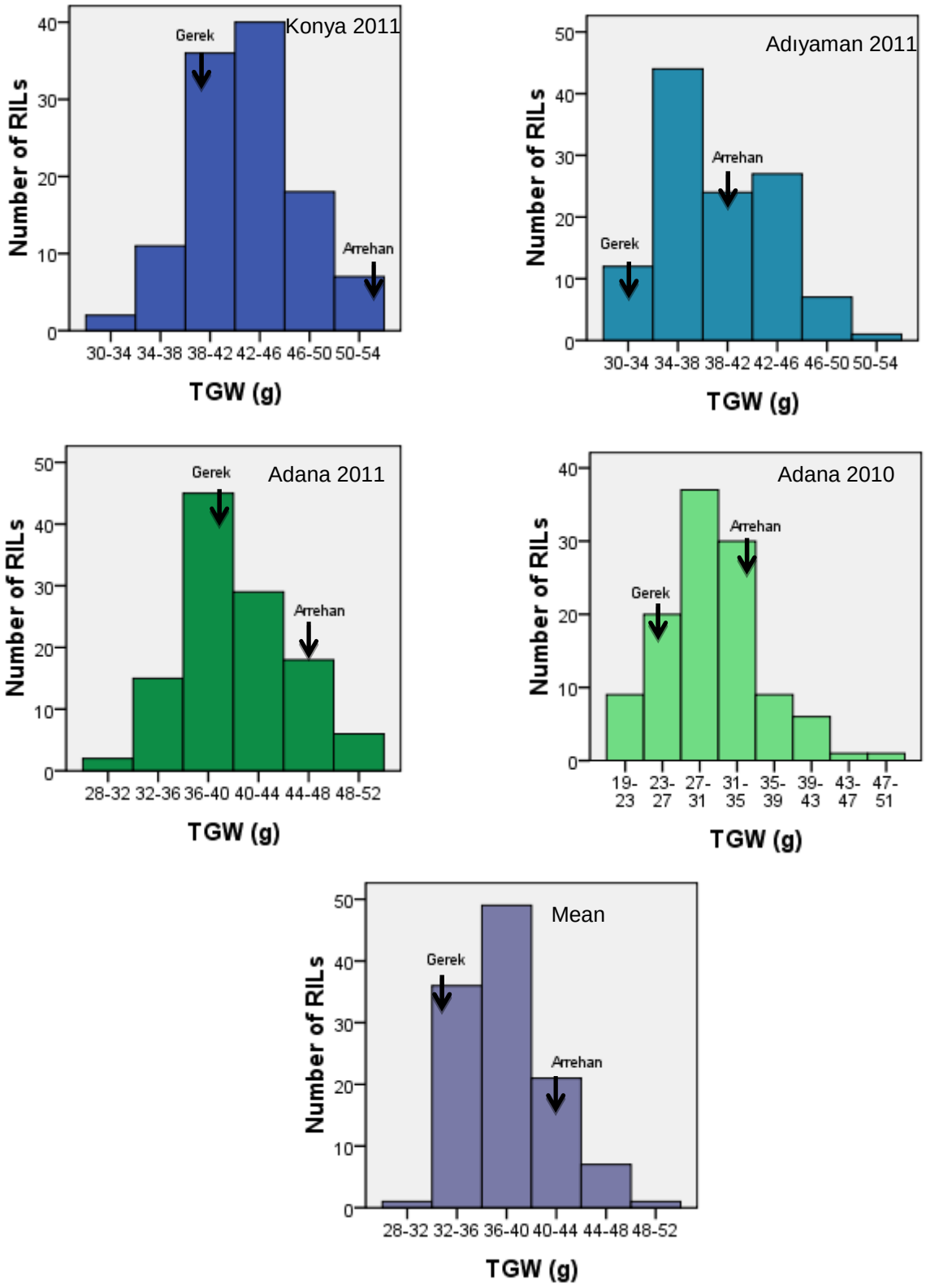

Figure: 4.22 Frequency distribution of 1000 grain weight for Gerek x Arrehane mapping population evaluated in four environments

4.2.2. Associations among traits

The associations among agronomic and yield related traits were determined by correlation analysis. The Pearson correlation coefficients for agronomic and yield related traits were calculated from the mean of all four environments/location for 113 RILs of Gerek x Arrehane mapping population. Table 4.8 shows statistical relationships among all studied characteristics.

Table 4.8. Simple correlation coefficients among agronomic and yield related traits for the Gerek x Arrehane mapping population calculated from the mean of the four environments for 113 RILs

Traits	PH	PeL	SL	NSS	NGS	GWS	SC	TGW	HD
PH	1.000								
PeL	0.596**	1.000							
SL	-0.147	0.094	1.000						
NSS	-0.084	0.073	0.827**	1.000					
NGS	-0.311**	-0.047	0.771**	0.749**	1.000				
GWS	-0.240**	.0.048	0.661**	0.520**	0.753**	1.000			
SC	-0.150	0.085	0.867**	0.491**	0.642**	0.630**	1.000		
TGW	-0.141	-0.061	0.281**	0.051	0.041	0.363**	0.376**	1.000	
HD	0.257**	0.239*	-0.354**	-0.188*	-0.110	-0.213*	0.403**	-.0.694**	1.000

PH:Plant height; PeL:Peduncle Lenght; SL: Spikelenght; NSS: Number of Spkelets per spike: NGS: Number of grain per spike; GWS: grain weight per spike; SC:Spike compactness; TGW: 1000 grain weight;HD: Days to heading

** Correlation is significant at 0.01 level

* Correlation is significant at 0.05 level

Correlations between different traits are generally due to the presence of linked genes and the epistatic effects of different genes. Environment plays an important role in correlation. In some cases, environment affects both the traits simultaneously in the same direction or sometimes in different directions (Yücel et al. 2009).

Correlation coefficients for different physical parameters showed that significant and positive as well as negative correlations existed between most of the agronomic and yield related traits. Plant height was positively significant ($P < 0.01$) correlated with peduncle length ($r = 0.596$) and days to heading ($r = 257$) but it was negatively significant correlated with number of grain per spike ($r = -0.311$) and

grain weight per spike (r = -0.240). Peduncle length was correlated with any of spike and yield related traits however it was only significantly ($P < 0.05$) associated with days to heading (r = 0.239). Spike length is an important character and was positively significant ($P< 0.01$) correlated with number of spikelets per spike (r = 0.827), number of grain per spike (r = 771), grain weight per spike (r = 661), spike compactness (r = 867) and thousand grain weight (r =281). Positive correlation of spike length with other spike traits was also observed by different previous researchers (Yücel et al. 2009; Wang et al. 2011). Number of spikelets per spike was significantly and positively associated with number of grain per spike, grain weight per spike and spike compactness. Similarly number of grain per spike was significant and positively associated with grain weight per spike and spike compactness. Thousand grain weights was positively and significantly ($P < 0.01$) associated with spike length, grain weight per spike and spike compactness but have positive and not significant relationship with number of grain per spike and as well negative but non-significant plant height and peduncle length. MacCartney et al. (2005) found positive significant correlation between days to heading and plant height. Days to heading is one the most important adaptation traits of bread wheat and there was significant negative correlation between days to heading and spike length, number of spikelets per spike, grain weight per spike, spike compactness and thousand grain weight, however it was positively associated with plant height and peduncle length. Thus, selection for the right characteristics will ensure improvement of more than one characteristic simultaneously due to the correlation among different traits.

4.3. QTL analysis and modes of inheritance

Developmental quantitative genetics assume that development of complex morphological structures and other traits of interest occur through the action and interaction of many genes acting differentially during ontogeny and whose expressions are modified by interaction with other genes and also by environments. Many quantitative traits are influenced by the environmental factors which make it necessary to perform phenotypic assessments in replicated trials under different and multiple environments using immortal mapping population consisting of recombinant inbred lines (RILs) or double haploids (DHs). In this study, we used the F6 recombinant inbred line (RILs) population derived from Gerek x Arrehane to identify QTLs determining spike length, number of spikelet per spike, number of grain per spike, grain weight per spike, 1000 grain weight, spike compactness, plant height, peduncle length, days to heading, and to investigate their mode of inheritance and interaction. As expected traits with higher heritability generally had more phenotypic variation explained by QTL detected in different agro climatic condition. The detection of the QTLs was carried out for each of the four environments/locations. The QTLs with their R^2 values (phenotypic variation explained by QTL), chromosomal locations and their additive effects and LOD values at confidence interval are illustrated for each trait and each locations in the Tables 4.9-4.17.

In the literature a LOD of <3 is often considered as a lower value, since the QTL analysis is faced with multiple testing (Lander and Botstein 1989; Börner et al. 2002). However, major and minor QTLs were detected at the same position in different experiments for several traits. Therefore LOD values <3 should also be taken into account in repeated experiments.

4.3.1. QTL for Thousand Grain weight (TGW)

Parent cultivar “Arrehane” have thicker grains with higher 1000 grain weight whereas cultivar “Gerek” have smaller and thinner grains with lower 1000 grain

weight. All putative QTLs for 1000 grain weight are presented in Table 4.9 and locations of the most significant QTLs are shown in the genetic map (Figure 4.23). Twelve chromosomal regions were identified associated with QTL for TGW each on 1A, 1D, 2A, 2D, 3A, 3B, 4A, 5A, 5B, 6B, 6D and 7B. Most of the QTLs were consistent over locations. Of the twelve QTLs detected for TGW, six QTLs were consistent for all four environments; they were located on 1A, 2A, 3A, 4A, 5A and 7B chromosomes, whereas five were found in three of the four environments and two QTLs in two environments. Some of the QTLs detected for TGW were similar with those detected in previous studies and some were different (Groose et al. 2003; Zanet et al. 2001; Patil et al. 2009). Patil et al. (2009) found three QTLs for TGW on chromosomes 2A, 4B and 6B. Maccaferri et al. (2008) discovered QTLs for TGW on chromosomes 2A, 2B, 3A, 4B, 5A and 6A. Houshmand et al. (2008) found 10 QTLs affecting 1000 grain weight locating on chromosomes 1A, 2A, 2B, 3B, 4B, 5B, 6B and 7B. Huang et al. (2004) reported that fourteen QTLs significantly influenced the TGW and mapped on 11 different chromosomes.

Interestingly, most of the QTLs were contributed by Arrehane and increased significantly TGW, whereas only three QTLs were contributed by Gerek. The strongest QTLs with highest LOD were QTgw.cu.3B.e2 and QTgw.cu.7B.e2. QTgw.cu.3B.e2 located on chromosome 3B flanked with markers interval “wPt-10003-wPt-664982” with LOD varying between 5.11 (KON-11) - 7.56 (ADM-11) and explained up to 26.9% of phenotypic variation depending on environments. QTgw.cu.7B.e2. with closest marker “wPt-6276”, with LOD score ranging from 3.38 to 7.38 located on chromosome 7B, contributed up to 26.4% variation of the trait. Another QTL located on 1A between loci wPt-1720- wPt-7951, with LOD 3.61 up to 6.45 consistently observed in all four environments explained 13.9 to 23.5% phenotypic variation of the trait, highest LOD detected in ADM-11 and lowest LOD found in ADA-11. Wang et al. (2011) also detected QTL for TGW on 1A and 7B with LOD up to 4.77 but with small phenotypic contribution. One QTL “QTgw.cu.4A” was detected on 4A in all four environments with LOD 3.91 up to 6.53, and explained phenotypic variation up to 23.7% and contributed significantly to

Table 4.9. QTLs detected in the Gerek x Arrehane mapping population for thousand grain weight in four environments

QTL[a]	Marker interval	LOD	A[c]	S[d]	R^2 (%)[e]
QTgw.cu.1A.e1[b]	wPt-1720-wPt-7951	4.67	-1.92	Ar	17.8
QTgw.cu.1A.e2	wPt-1720-wPt-7951	6.45	-2.40	Ar	23.5
QTgw.cu.1A.e3	wPt-1720-wPt-7951	3.61	-1.70	Ar	13.9
QTgw.cu.1A.e4	wPt-1720-wPt-7951	3.67	-2.25	Ar	14.1
QTgw.cu.1D.e1	wPt-730879	2.37	-1.44	Ar	9.4
QTgw.cu.1D.e2	wPt-730879	4.04	-2.00	Ar	15.4
QTgw.cu.1D.e3	wPt-730879	3.36	-1.71	Ar	13
QTgw.cu.2A.e1	tPt-9405-wPt-664128	2.19	-1.43	Ar	8.8
QTgw.cu.2A.e2	tPt-9406-wPt-664128	3.25	-1.90	Ar	12.6
QTgw.cu.2A.e3	tPt-9407-wPt-664128	4.20	-1.94	Ar	16
QTgw.cu.2A.e4	wPt-1657	3.16	-2.00	Ar	12.3
QTgw.cu.2D.e1	wPt-3757-wPt-665972	3.94	-1.99	Ar	15.2
QTgw.cu.2D.e2	wPt-3757-wPt-665972	2.32	-1.64	Ar	9.2
QTgw.cu.2D.e4	wPt-3757-wPt-665972	2.49	-2.04	Ar	9.8
QTgw.cu.3A.e1	wPt-730156-wPt-7496	2.79	-1.43	Ar	11
QTgw.cu.3A.e2	wPt-730156-wPt-7496	3.07	-1.69	Ar	12
QTgw.cu.3A.e3	wPt-730156-wPt-7496	3.20	-1.65	Ar	12.4
QTgw.cu.3A.e4	wPt-5133-wPt-5886	3.47	-2.31	Ar	13.4
QTgw.cu.3B.e1	wPt-10003-wPt-664981	5.11	2.00	Gr	19.2
QTgw.cu.3B.e2	wPt-10003-wPt-664981	7.56	2.72	Gr	26.9
QTgw.cu.3B.e4	wPt-10003-wPt-664981	5.49	2.85	Gr	20.4
QTgw.cu.4A.e1	wPt-5428-wPt-0817	3.91	1.72	Gr	15.1
QTgw.cu.4A.e2	wPt-5428-wPt-0817	6.53	2.28	Gr	23.7
QTgw.cu.4A.e3	wPt-5428-	3.94	1.67	Gr	15.1
QTgw.cu.5A.e1	cfa2190-wPt-0605	3.08	-1.68	Ar	12.1
QTgw.cu.5A.e2	cfa2190-wPt-0605	5.74	-2.47	Ar	21.2
QTgw.cu.5A.e3	cfa2190-wPt-0605	2.34	-1.52	Ar	9.3
QTgw.cu.5A.e4	wPt-5120-wPt-7029	3.34	-2.20	Ar	12.9
QTgw.cu.5B.e2	wPt-5120-wPt-7029	6.17	-2.17	Ar	22.6
QTgw.cu.5B.e3	wPt-5120-wPt-7029	2.75	-1.40	Ar	10.8
QTgw.cu.5B.e4	wPt-5120-wPt-7029	2.09	-1.59	Ar	8.3
QTgw.cu.6B.e1	wPt-8412-wPt-4312	5.94	-2.24	Ar	22
QTgw.cu.6B.e2	wPt-8412-wPt-4312	3.15	-1.81	Ar	12.2
QTgw.cu.6B.e4	wPt-8412-wPt-4312	4.11	-2.47	Ar	15.7
QTgw.cu.6D.e4	wPt-733284-wPt-6811	5.58	-2.53	Ar	20.7
QTgw.cu.7B.e1	wPt-6276	4.38	1.96	Gr	16.8
QTgw.cu.7B.e2[b]	wPt-6276	7.38	2.59	Gr	26.4
QTgw.cu.7B.e3	wPt-6276	4.61	1.53	Gr	10.7
QTgw.cu.7B.e4	wPt-6276	4.61	2.54	Gr	17.4

a Nomenclature for QTL in wheat: "Q" refers to QTL, followed by trait designator, "cu" for the laboratory, and the chromosome and environment

b E1,E2, E3 and E4 refers to locations: Konya 2011, Ad ıyaman2011, Adana2011 and Adana2010

c refer to additive effect. Positive additive effect indicate increased effect from parent "Gerek" and negative value indicate increase effect parent "Arrehane

d Source of allele from contributing parents

e Percentage of phenotypic variation explained by each QTL.

increase TGW. Another QTL with high LOD value (5.58) and with high phenotypic contribution (20.7%) was detected in only ADA-11 environment.

Four QTLs (QTgw.cu.1D, QTgw.cu.2A. QTgw.cu.2D and QTgw.cu.3A) on chromosomes ID, 2A, 2D and 3A were detected in three environments at average LOD 2.92 and 3.02 with minor effect and explained 11.4 and 11.8% phenotypic variation of the trait. QTgw.cu.2A was flanked with tPt-9407-wPt-664128 in three environments except ADA-10 where closest marker with this QTL was wPt-1657. Similarly QTgw.cu.3A flanked with marker loci wPt-730156-wPt-7498 except ADA-10 location. Saleh (2011) also detected one minor QTL with 2.6 LOD value at chromosome 3A in only one out of eight environments. Campbell et al. (2003) reported a QTL located on chromosome 3A contributing 14.2% phenotypic variation.

In our study, we found many QTLs with significant and positive contributions to the phenotypic trait for TGW, which is much higher and in contrast to other the previous QTL studies (Campbell et al. 2003; Börner et al. 2002; Huang et al. 2004; Narasimhamoorthy et al. 2006; Kumar et al. 2006). Kumar et al (2006) reported that most of the QTLs for grain weight explained only small fraction (6.57-12.80%) of the phenotypic variation. They reported that grain weight is a polygenic trait controlled by few gene/QTL with major effect and many QTLs with minor effect. However in this study, many QTLs with major effect for TGW contributed higher phenotypic variation. We found some stable QTL flanking with same markers with strongest effect on TGW. Interestingly all of the QTLs contributed positively to increase the TGW and were transferred by parent Arrehane. Arrehane cultivar is semi dwarf and early heading cultivar. Therefore it could be hypothesized that the early heading and some favorable weather conditions during grain filling period may indirectly affect the TGW as seen in the correlation analysis (Table 4.8). The comparison of agronomic trait data related to the Gerek and Arrehane provide additional evidence of this relationship.

4.3.2. QTLs for Plant Height (Pht)

Interval mapping revealed eight QTLs controlling plant height (Table 4.10 Fig 4.23). All of the QTLs detected were with minor effect on plant height except one QTL. The strongest QTL for plant height was QPht.cu.4D located on chromosome 4D. This QTL had a LOD score ranging from 3.52 (KON-11) to 5.36 (ADA-11) and R^2 value of 13.6 (KON-11) to 19.9 (ADA-11). The LOD score peak was between the marker interval "wPt-2379-wPt-666459". This QTL was contributed by parent Gerek and resulted in increased plant height. In contrast to our results, McCartney et al. (2005) reported one of the strongest heights reducing QTL QHt.crc-4D on chromosome 4D with LOD 30.6 that contributed to reduce plant height. Chromosome 4D also contained height reducing genes "Rht-D1b (mutant allele for dwarf type)". However in their study the parents used in development of population were RL4452 and AC Domain which contain "*Norin 10* (*source of Rht-B1 and Rht-D1 allele in the world wheat germplasm*) in their pedigree. In our case Gerek is indigenous Turkish cultivar which was most probably selected from the local landraces and is mainly grown in the stress and drought environment of Central Anatolian region. The environments of Adana and Adıyaman used in this study, had higher precipitation levels during the whole field trial periods in 2010-2011 compared with the long years, which also resulted in the increased plant height. Both of the parents "Gerek and Arrehane" used in the population development were tall, with an approximate average 15cm difference between them. The parent Gerek contains Rht-4Da1 (wild allele for longer plant height) (Yediay et.al. 2011). In this study, showing the QTL of QPht.cu.4D on 4D the region controlling the plant height confirmed the results that chromosome 4D contains Rht-4D-a1. Verma et al. (2005) reported that interval mapping and composite interval mapping confirmed QTLs on chromosome 3D, 4B and 4D, the last two relating to RHt-B1 and Rht-D1

Table 4.10. QTLs detected in the Gerek x Arrehane mapping population for plant height in four environments

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QPht.cu.1B.e1	wPt-3475-tPt-0283	2.19	3.08	Gr	8.7
QPht.cu.1B.e2	wPt-3475-tPt-0284	2.83	3.06	Gr	11.1
QPht.cu.1B.e3	wPt-3475-tPt-0285	2.43	3.11	Gr	9.6
QPht.cu.1B.e4	wPt-3475-tPt-0286	4.49	4.57	Gr	17.1
QPht.cu.2B.e2	wPt-1964-tPt-4627	2.33	-2.53	Ar	9.2
QPht.cu.2B.e3	wPt-1964-tPt-4628	2.41	-2.83	Ar	9.5
QPht.cu.2D.e1.1	CFD43-GWM484	2.23	-3.36	Ar	8.8
QPht.cu.2D.e2.1	CFD43-GWM485	2.2	-2.62	Ar	8.7
QPht.cu.2D.e3.1	wPt-9780	2.18	-2.74	Ar	8.7
QPht.cu.2D.e4.1	CFD43-GWM484	3.07	-3.71	Ar	12.1
QPht.cu.2D.e1.2	wPt-2652	2.77	-3.60	Ar	10.9
QPht.cu.3B.e1	wPt-4719	2.32	3.09	Gr	9.2
QPht.cu.3B.e2	wPt-0571	2.11	2.53	Gr	8.4
QPht.cu.3B.e3	wPt-6239-wPt-5432	2.28	2.81	Gr	9
QPht.cu.3B.e4	wPt-6239-wPt-5432	4.01	3.82	Gr	15.5
QPht.cu.4A.e1	wPt-667130	2.16	2.97	Gr	8.6
QPht.cu.4D.e1	wPt-2379-wPt-666459	3.52	4.08	Gr	13.6
QPht.cu.4D.e2	wPt-2379-wPt-666459	4.46	3.70	Gr	16.9
QPht.cu.4D.e3	wPt-2379-wPt-666459	5.36	4.48	Gr	19.9
QPht.cu.4D.e4	wPt-2379-wPt-666460	4.44	4.20	Gr	17
QPht.cu.6B.e1	wPt-741982	2.35	3.56	Gr	9.3
QPht.cu.6B.e2	wPt-4648	2.16	2.83	Gr	8.6
QPht.cu.6B.e3	wPt-4312	3.03	3.70	Gr	11.9

a Nomenclature for QTL in wheat: "Q" refers to QTL, followed by trait designator, "cu" for the laboratory, and the chromosome and environment
b E1,E2, E3 and E4 refers to Konya 2011, Adıyaman 2011, Adana2011 and Adana 2010
d refer to additive effect. Positive additive effect indicate increased effect from parent "Gerek" and negative value indicate increase effect parent "Arrehane
e Source of allele from contributing parents
f Percentage of phenotypic variation explained by each QTL

One QTL "QPht.cu.1B" was detected at chromosome 1B consistent at all four locations with LOD 2.19 up to 4.49 and explained phenotypic variation up to 17.1%. Another minor QTL was detected at chromosome 6B at LOD score 2.16-3.03, explained 8.6-11.9 % phenotypic variation. Zhang et al. (2011) found one QTL at 6B at higher LOD value (10.52) stable at four environments, which could explain that this chromosomal region could be involved another allele controlling plant height.

One minor QTL "QPht.cu.2B." at LOD around 2.41 was detected at chromosome 2B and explained up to 9.5 % of the total phenotypic variation and reduced plant height contributed by Arrehane. This QTL was observed in two environments (ADM11 and ADA11). Several other minor QTLs for plant height were found on different chromosomes such as 2B, 2D, 3B, 4A and 4D. Chromosome 2D contained two QTLs. QPht.cu.2D.1 was detected in three environments whereas QPht.cu.2D.2 was appeared only in Konya environment both were contributed by Arrehane. Wu et al. (2010) also detected a strong QTL on 2D chromosome. Rht8, 'another most important dwarfing gene sourced from Japanese cultivar, "*Akakomugi*" located on short arm of 2D chromosome. But in the present study, QTL at 2D chromosome decreased plant height with minor effect in all environments, which could explain the absence of Rht8 dwarfing gene in Arrehan. Detection of the QTLs for plant height was consistent among environments in the present study. Zhang et al. (2011) also reported 25 QTLs for plant height explaining 2.11% to 9.66% of the phenotypic variation, located on different chromosomes. Similarly, four major QTLs were detected for plant height on chromosome arms 1AS, 2DS, 4AL (detected in two experiments together with minor QTLs detected in three experiments) and 6AS (detected in two experiments together with minor QTLs detected in three experiments) (Börner et al. 2002).

Plant height is one of the most important adaptation trait for bread wheat and one of the most important criteria in world wheat breeding programs. In general, an increase in plant height can result in increased lodging which ultimately negatively affects the final plant output. This was confirmed by many earlier researchers (Huang et al. 2006). Wu et al. (2010) described that genetic structure underlying the plant height development is network of genes with additive and epistatic effects. To date, twenty one Rht genes have been identified on 21 bread wheat chromosomes (McIntosh et al. 1998), of which two GA-insensitive semi dwarfing genes have been successfully used in wheat breeding programs worldwide.

4.3.3. QTL for peduncle length (Pel)

Simple interval mapping for QTL analysis of peduncle length identified three genomic regions controlling the trait in Gerek x Arrehane population. All these three QTLs were with minor effect and highly inconsistent and unstable, each identified in one out of three studied environments. Out of three QTLs, one was contributed by Arrehane and other two were transferred by Gerek (Table 4.11).

Table 4.11. QTL detected in the Gerek x Arrehane mapping population for plant height in three environments

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QPel.cu.1B.e3	wPt-4688	2.15	1.36	Gr	8.5
QPel.cu.3A.e1	wPt-798970	2.21	1.56	Gr	8.8
QPel.cu.6D.e2	wPt-1314	2.53	-1.49	Ar	10

a Nomenclature for QTL in wheat: "Q" refers to QTL, followed by trait designator, "cu" for the laboratory, and the chromosome and environment
b E1,E2 and E3 refers to Konya 2011, Adıyaman 2011 and Adana2011, respectively
d refer to additive effect. Positive additive effect indicate increased effect from parent "Gerek" and negative value indicate increase effect parent "Arrehanee
e Source of allele from contributing parents
f Percentage of phenotypic variation explained by each QTL

One minor QTL on chromosome 1B 'QPel.cu.1B' was appeared in Adana-11 environment. The estimated LOD score of this QTL was 2.15 and flanked with marker 'wPt-4688' and contributed allele from Gerek. This QTL contributed only 8.5% of the phenotypic variation. QTL 'Qpel.cu.3A' was also transferred allele from Gerek and detected on chromosome 3A in environment1 (Konya-11) at LOD score 2.21. This QTL flanked with DArT marker 'wPt-798970' and explained 8.8% of the total phenotypic variation. Another minor QTL 'QPel.cu.6D' also appeared in only one location (ADM-11) at estimated LOD scores 2.53. This QTL was transferred from Arrehane and explained 10% of the phenotypic variation.

All three peduncle length QTLs were inconsistent with environments as well as QTLs with plant height. Heidari et al. (2012) reported that all QTLs for peduncle length were consistent with plant height QTLs. Genetic structure of peduncle length is least studied and had not been focused of QTL studies in the last decade. One few

rare studies investigated the genetics of peduncle length. All the QTLs in this study were different what were reported in Heidari et al. (2012). They reported that among bread wheat chromosomes, 4D, 4B and 2D made strongest contribution to the expression of the peduncle length.

4.3.4. QTLs for Days to Heading (Dh)

Genetic control of heading time or flowering time is very complex phenomena and is control by network of various genes. When two parental lines with diverse genetic backgrounds are used to generate random inbred lines, segregation in the phenotypes of agricultural interest is complicated by the fact that more than one gene contributes to the phenotype. The present mapping population was developed from a cross of winter and spring cultivars. Gerek is a winter wheat cultivar mostly grown in central Anatolian region under hard winter conditions. Arrehane is insensitive to vernalization requirement and is a spring wheat cultivar. Gerek is sensitive to vernalization and exhibited recessive alleles of all four vernalization genes, "*Vrn-A1*, *Vrn-B1, Vrn-D1* and *Vrn-B3*" (Andeden et al. 2011).

The extent of variation observed for duration of ear emergence in this population was large, indicating the possible action of several genes or QTLs resulting in the observed transgressive segregation. It seems that some QTLs with major effect and a number of regions with minor effects independent of these environmental variables control the duration from planting to head emergence in this population. QTL data compilation showed that all chromosome groups were involved in the genetic control of days to heading in bread wheat. Total fifteen QTLs were detected on different chromosomes (Table 4.12).

Only six chromosomes (1D, 3A, 3D, 4D, 5D and 7A) exhibited no QTLs for days to heading in this population. Detected QTLs were highly consistent among environments, and most of them had moderate to high effect with LOD values ranging between 2.3 to 9.09.

Table 4.12. QTLs detected in the Gerek x Arrehane mapping population for days to heading in three environments

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QHd.cu.1A.e1	wPt-1720-wPt-7951	4.15	-2.42	Ar	15.8
QHd.cu.1A.e2	wPt-732970-wPt-664972	4.39	-2.73	Ar	16.6
QHd.cu.1A.e3	wPt-731282-wPt-7784	3.87	-3.77	Ar	14.8
QHd.cu.1B.e1	tPt-0283	3.57	2.11	Gr	13.8
QHd.cu.1B.e2	tPt-0283	4.75	2.47	Gr	17.9
QHd.cu.1B.e3	tPt-0283	6.22	4.12	Gr	22.7
QHd.cu.2A.e1	tPt-1041	3.19	3.51	Gr	12.4
QHd.cu.2A.e2	tPt-1041	3.57	2.50	Gr	13.8
QHd.cu.2A.e3	tPt-1041	3.19	3.51	Gr	12.4
QHd.cu.2B.e1	wPt-0289	3.48	-2.13	Ar	13.4
QHd.cu.2B.e2	wPt-0289	4.04	-2.34	Ar	15.4
QHd.cu.2B.e3	wPt-0289	2.73	-2.89	Ar	10.7
QHd.cu.2D.e1	wPt-665868	3.58	-2.12	Ar	13.8
QHd.cu.2D.e2	wPt-665868	2.3	-1.77	Ar	9.1
QHd.cu.2D.e3	wPt-665972	3.35	-3.13	Ar	13
QHd.cu.3B.e1	wPt-10003-wPt-664981	9.09	3.33	Gr	31.4
QHd.cu.3B.e2	wPt-10003-wPt-664981	8.81	3.66	Gr	30.6
QHd.cu.3B.e3	wPt-10003-wPt-664981	7.55	4.67	Gr	26.9
QHd.cu.4A.e1	wPt-7807-wPt-6900	5.84	2.64	Gr	21.5
QHd.cu.4A.e2	wPt-7807-wPt-6900	5.93	2.76	Gr	21.8
QHd.cu.4A.e3	wPt-7807-wPt-6900	3.76	3.32	Gr	14.5
QHd.cu.4B.e2	GWM6	3.92	2.51	Gr	15
QHd.cu.4B.e3	GWM6	3.26	3.41	Gr	12.7
QHd.cu.5A.e1	cfa2190-wPt-0605	5.1	-2.83	Ar	19.1
QHd.cu.5A.e2	cfa2190-wPt-0605	4.3	-2.59	Ar	16.3
QHd.cu.5A.e3	cfa2190-wPt-0605	3.48	-3.47	Ar	13.4
QHd.cu.5B.e1	tPt-7755-wPt-1409	3.62	-2.13	Ar	13.9
QHd.cu.5B.e2	gwm554-wPt-5604	4.31	-2.71	Ar	16.4
QHd.cu.5B.e3	gwm554-wPt-5604	5.33	-4.41	Ar	19.8
QHd.cu.6A.e1	wPt-730711	3.23	2.07	Gr	12.6
QHd.cu.6A.e2	wPt-7599	4.29	2.36	Gr	16.3
QHd.cu.6A.e3	wPt-7599	4.06	3.40	Gr	15.5
QHd.cu.6B.e1	wPt-6674-wPt-4706	3.32	-2.03	Ar	12.9
QHd.cu.6B.e2	wPt-6674-wPt-4706	3.32	-2.08	Ar	12.9
QHd.cu.6B.e3	wPt-6674-wPt-4706	3.35	-3.18	Ar	13
QHd.cu.6D.e1	wPt-732321-wPt-730912	3.26	2.24	Gr	12.6
QHd.cu.6D.e2	wPt-9068	3.82	2.23	Gr	14.6
QHd.cu.6D.e3	wPt-9068	3.31	3.08	Gr	12.8
QHd.cu.7B.e1	wPt-6276	5.51	2.80	Gr	20.4
QHd.cu.7B.e2	wPt-6276	5.12	2.78	Gr	19.1
QHd.cu.7B.e3	wPt-6276	5.55	4.26	Gr	20.6
QHd.cu.7D.e1	wPt-0827	3.68	2.21	Gr	14.2
QHd.cu.7D.e2	wPt-0827	3.86	2.32	Gr	14.8
QHd.cu.7D.e3	wPt-0827	3.6	3.31	Gr	13.9

a Nomenclature for QTL in wheat: "Q" refers to QTL, followed by trait designator, "cu" for the laboratory, and the chromosome and environment

b E1,E2, E3 refers to Adıyaman2011, Adana2011 and Adana2010

d refer to additive effect. Positive additive effect indicate increased effect from parent "Gerek" and negative value indicate increase effect parent "Arrehane

e Source of allele from contributing parents

f Percentage of phenotypic variation explained by each QTL

Strongest QTL (QHd.cu.3B) was detected on chromosome 3B at marker interval "wPt-10003-wPt-664981" in all three environments. This QTL was observed at highest LOD value between 7.55 (ADA-10) and 9.09 (ADM- 11) with phenotypic variation up to 31.4%. Marza et al. (2006) also detected a QTL on the long arm of 3B chromosome, resulting in delayed flowering time and late maturity. MacCartney et al. (2005) also identified a minor QTL on 3B. However QHd.cu.3B detected in Gerek x Arrehane mapping population was a new quantitative locus and had not previously reported, showing that other genes or group of genes on 3B may be also involved in the control of heading time in bread wheat.

Second largest QTL "QHd.cu.4A" with average LOD value 5.18 was detected on 4A chromosome; explaining phenotypic variation up to 21.8% depending on the environments and this QTL was contributed by Gerek and resulted in delayed flowering time. In contrast, Huang et al. (2004) also detected a QTL on 4A chromosome resulting in early heading. A QTL on the long arm of chromosome 4A was corresponding to heading date in ITMI population (Kluwer et al. 2003) and could be related to photoperiod sensitivity according to Hanocq et al. (2007). Chromosome 5A (QHd.cu.5A) and 5B (QHd.cu.5B) each also contained effective QTLs for ear emergence time. QHd.cu.5A had a overall LOD score of 4.29 and explained up to 19.1% phenotypic variations according to environments. QHd.cu.5B with average LOD value of 4.42, with phenotypic variation up to 19.8 mapped on chromosome 5A in all environments. Chu et al. (2008) also reported two major QTLs on the homologous group 5 (5A and 5B) associated with days to heading. Group 2 chromosomes (2A, 2B and 2D) also contained QTLs with moderate effect on flowering time. Three QTLs accounting for 12.9, 13.2 and 12 % of the phenotypic variation were detected on the 2A, 2B and 2D chromosomes, respectively. Börner et al. (2002) found major loci on chromosome arm 2DS (together with one minor QTL) and on chromosome arm 5DL (detected in three experiments, together with two minor QTLs) for ear emergence time in hexaploid wheat. Cytogenetic studies have shown that groups 2 and 5 contain major genes controlling photoperiod sensitivity (PS) (Ppd series; Welch et al. 1973) and vernalization requirement (VR) (Vrn series; Law et al. 1976). Scarth and Law (1984) reported three genes controlling

photoperiod response in wheat including *Ppd-D1* located on long arm of chromosome 2D, *Ppd-B1* located on short arm of chromosome 2B and *Ppd-A1* located on long arm of 2A. Hanocq et al. (2007) reported that group 2 chromosomes not only involved in the control of PS but also in the control of earliness (IE) (2B and 2D) and VR (2B). Chromosome 5A carries two major genes, Vrn-A1 (Law et al. 1976) and Q locus (Miller and Reader 1982) for VR and ear morphology. Kato et al. (1999) analyzed a population derived from a CS-Cappelle- Desprez 5A substitution line and reported two QTLs on 5A associated with ear emergence time. One QTL was in the vicinity of Vrn-A1 gene and other in the vicinity of Q gene. They also reported that QTLs on 5B may correspond to vernalization gene Vrn-B1. Similar findings were also observed by Hanocq et al. (2004). Therefore, it seems that variation in days to heading in this RIL population might be influenced by allelic variation in vernalization loci, which is also confirmed by the DNA analysis of cultivar Gerek by functional markers of vernalization and photoperiod genes (Andeden et al. 2011).

One QTL "QHd.cu.7B" with moderate effect was detected on chromosome 7B with LOD 5.12 (ADA-11) and 5.51 (ADM-11), and explained up to 20.6% phenotypic variation depending on the location. Lin et al. (2008) reported a major early flowering QTL on short arm of 7B chromosome. QTL governing the heading date on 7B also had been reported by many other previous researchers (Kuchel et al. 2006; Sourdille et al. 2000). Another QTL "QHd.cu.7D" was observed on 7D chromosomes with average LOD of 3.71 and explained up to 14.3% of phenotypic variation depending on the environment.

Group 1 chromosomes also contained effective QTLs. 1A, 1B contained one QTL each. QHd.cu.1A accounted up to 16.6% of phenotypic variation. QHd.cu.1B was detected on chromosome 1B up to LOD score of 6.22 (ADA-10), and explained up to 22.7% of phenotypic variation. Zhang et al. (2009) reported a main effect QTL on chromosome 1B. Law et al. (1998) showed that genes or QTLs for ear emergence were present on each homologous member of group 1 chromosomes. Similarly Kuchel et al. (2006) detected one QTL for heading date on chromosome 1A in a DH population.

Several genomic regions associated with heading date were detected in Gerek x Arrehane RIL population. Some of the new QTLs were detected in this study. However, many QTLs for a single trait have been reported, but one QTL may appear in one population but not in another one, because of various genetic backgrounds of the parental lines used in different experiments (Yan 2009).

4.3.5. QTLs for spike morphology and related traits

Spike related characteristics contribute greatly to the final grain yield of bread wheat (Cui et al. 2012). Three yield components ---productive spikes per unit area, number of kernels per spike, and kernel weight---- together determine the grain yield level of wheat. Indeed these component traits are also under quantitative trait loci control, but they exhibit less sensitivity to environmental conditions and higher heritability than grain yield (Ma et al. 2007). Thus, it is efficient to dissect factors affecting the yield portioning into its components. In addition, variation in spike morphology is one of the most widely used criteria for species determination and is extensively investigated in wheat. So it is of importance to unravel the control mechanism of spike-related traits at QTL level. QTLs for agronomic traits such as heading date, plant height etc. and yield have been studied extensively and some functional markers have been reported and started to be used in marker assisted selection worldwide in last decade. The spike morphology and spike related traits have been also studied, however the QTL analysis for spike related traits is very complex phenomena and still information available for spike related traits is insufficient and need to be studied in detail in different genetic backgrounds, in order to understand the genetic mechanism behind as well as to develop functional markers for future markers assisted selection for these traits

4.3.5.1. Spike Length (Sl)

Interval mapping analysis of the Gerek x Arrehane population for spike length showed that eighteen chromosomal regions were identified in the RIL

population (Table 4.13; Figure 4.23).

Out of these 18 regions 8 chromosomal regions were the major regions affecting the spike length. They were located on chromosome 1A, 1B, 2B, 2D, 3B, 5A, 6B and 7A. Cui et al. (2012) identified four major QTLs and 20 minor additive QTLs in two RIL populations affecting the spike length. Marza et al. (2006) identified ten QTLs for spike length on 1AL, 1B, 1AS, 2BL, 2BS, 3B, 4B, 5B, 7A and 7B. Most of the major or minor QTLs detected in Gerek x Arrehane contributed for longer spike length from parent Arrehan whereas only three QTLs were contributed by parent Gerek. A major QTL "QSl.cu.1A" contributing positively to this trait, which was detected in four environments, was mapped chromosome 1A (Fig. 4.22) at LOD value ranging between 6.88 to 11.36, explained 22.1-37.6% of phenotypic variation. This QTL has not been previously described in the literature. However, in this study, this QTL was strongest QTL resulting in longer spikes.

Other QTLs with major effect and at high LOD were located on 1B, 2B, 2D, 3B, 3D, 6B and 7A. QTL "QSl.cu.2B" on 2B was detected at LOD 4.18-8.35, explained 15.9-29.3 % of phenotypic variation depending on the environment. Kumar et al. (2007) also detected a QTL on chromosome 2B with phenotypic variation up to 18.10%

Two QTLs were detected on chromosome 2D. One major QTL "QSl.cu.2D.2" flanking with marker interval "wPt-3757-wPt-665972" was also detected in all environments on chromosome 2D at LOD 4.4-11.35, explained 16.7-37.5% of phenotypic variation. Other QTL "QSl.cu.2D.1" was with moderate additive effect contributed by Arrehane, detected in three environments. QTL for spike length on chromosome 1A and 2D were verified in the IF2 mapping population (Ma et al. 2007). It was reported that chromosome 2D contained *Rht8* gene (Worland et al. 1998), and has been related with spike length in different genetic backgrounds (Suenaga et al. 2005), and associated with yield, test weight, days to heading (Narasimhamoorthy et al.2006) and thousand grain weight (Groose et al. 2003). Kumar et al. (2007) reported that QTLs for five traits (spike length, spikelets number per spike, yield, and harvest index and grain number per spike) on chromosome 2D are consistent.

Table 4.13. QTLs detected in the Gerek x Arrehane mapping population for spike length in four environments

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QSl.cu.1A.e1	wPt-1720-wPt-7951	6.98	-0.55	Ar	22.1
QSl.cu.1A.e2	wPt-1720-wPt-7952	10.04	-0.70	Ar	34.1
QSl.cu.1A.e3	wPt-1720-wPt-7953	11.36	-0.90	Ar	37.6
QSl.cu.1A.e4	wPt-1720	6.88	-0.69	Ar	24.8
QSl.cu.1B.e1	tPt-0283-wPt-3475	4.45	0.57	Gr	16.8
QSl.cu.1B.e2	tPt-0283-wPt-3476	9.42	0.71	Gr	32.3
QSl.cu.1B.e3	tPt-0283-wPt-3477	5.25	0.67	Gr	19.6
QSl.cu.1B.e4	tPt-0283-wPt-3478	3.23	0.55	Gr	12.6
QSl.cu.1D.e1	wPt-5721-wPt-664829	2.6	-0.45	Ar	10.2
QSl.cu.1D.e2	wPt-5721-wPt-664830	5.01	-0.56	Ar	18.8
QSl.cu.1D.e3	wPt-5721-wPt-664831	4.23	-0.63	Ar	16.1
QSl.cu.1D.e4	wPt-5721-wPt-664832	2.75	-0.54	Ar	10.8
QSl.cu.2B.e1.1	wPt-6158	5.27	-0.54	Ar	19.6
QSl.cu.2B.e2.1	wPt-6120	5.95	-0.52	Ar	21.9
QSl.cu.2B.e3.1	wPt-1813	4.74	-0.60	Ar	17.9
QSl.cu.2B.e4.1	wPt-1813	4.92	-0.62	Ar	18.5
QSl.cu.2B.e1.2	wPt-741382	6.5	-0.58	Ar	23.3
QSl.cu.2B.e2.2	wPt-741383	8.35	-0.66	Ar	29.3
QSl.cu.2B.e3.2	wPt-741382	7.58	-0.72	Ar	27
QSl.cu.2B.e4.2	wPt-741382	4.18	-0.56	Ar	15.9
QSl.cu.2B.e1.3	wPt-665014-wPt-7567	3.16	-0.44	Ar	12.3
QSl.cu.2B.e2.3	wPt-665014-wPt-7567	4.75	-0.56	Ar	17.9
QSl.cu.2B.e3.3	wPt-665014-wPt-7567	4.99	-0.72	Ar	18.7
QSl.cu.2B.e4.3	wPt-665014-wPt-7567	5.13	-0.79	Ar	19.2
QSl.cu.2D.e1.1	cfd36-wPt-3812	2.59	-0.41	Ar	10.2
QSl.cu.2D.e2.1	cfd36-wPt-3812	4.65	-0.51	Ar	17.5
QSl.cu.2D.e3.1	cfd36-wPt-3812	6.7	-0.71	Ar	24.3
QSl.cu.2D.e1.2	wPt-3757-wPt-665972	4.84	-0.51	Ar	18.2
QSl.cu.2D.e2.2	wPt-3757-wPt-665972	11.35	-0.71	Ar	37.5
QSl.cu.2D.e3.2	wPt-3757-wPt-665972	8.39	-0.84	Ar	29.4
QSl.cu.2D.e4.2	wPt-3757-wPt-665972	4.4	-0.64	Ar	16.7
QSl.cu.3B.e1	wPt-732044-wPt-9170	7.91	0.62	Gr	28
QSl.cu.3B.e2	wPt-732044-wPt-9170	6.78	0.55	Gr	24.5
QSl.cu.3B.e3	wPt-732044-wPt-9170	6.71	0.67	Gr	24.3
QSl.cu.3B.e4	wPt-732044-wPt-9170	4.68	0.58	Gr	17.6
QSl.cu.3D.e1	wPt-742488	6.01	-0.55	Ar	22.1
QSl.cu.3D.e2	wPt-742689	5.39	-0.50	Ar	20
QSl.cu.3D.e3	wPt-742488	6.6	-0.66	Ar	23.9
QSl.cu.5A.e1	wPt-4279-gwm293	2.35	-0.40	Ar	9.3
QSl.cu.5A.e2	wPt-4279-gwm294	4.21	-0.50	Ar	16
QSl.cu.5A.e3	wPt-4279-gwm295	7.07	-0.75	Ar	25.4
QSl.cu.5A.e4	wPt-4279-gwm296	3.6	-0.57	Ar	13.9

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QSl.cu.6A.e1.1	wPt-4907	4.18	-0.48	Ar	15.9
QSl.cu.6A.e2.1	wPt-6520	5.42	-0.52	Ar	20.1
QSl.cu.6A.e3.1	wPt-4907	4.17	-0.55	Ar	15.9
QSl.cu.6A.e4.1	wPt-6520	3.27	-0.51	Ar	12.7
QSl.cu.6A.e1.2	wPt-671568	3.14	-0.42	Ar	12.2
QSl.cu.6A.e2.2	wPt-671568	6.25	-0.54	Ar	22.8
QSl.cu.6A.e3.2	wPt-671568	6.01	-0.65	Ar	22.1
QSl.cu.6B.e1.1	wPt-745074	5.4	-0.54	Ar	20.1
QSl.cu.6B.e2.1	wPt-745074	10.18	-0.66	Ar	34.4
QSl.cu.6B.e3.1	wPt-745074	6.79	-0.68	Ar	24.5
QSl.cu.6B.e4.1	wPt-745074	5.03	-0.61	Ar	18.8
QSl.cu.6B.e2.2	wPt-1241	5.96	-0.58	Ar	21.9
QSl.cu.6B.e3.2	wPt-1241	4.84	-0.65	Ar	18.2
QSl.cu.6B.e4.2	wPt-1241	5.57	-0.73	Ar	20.6
QSl.cu.7A.e1.1	wPt-4489-wPt-7122	5.74	-0.56	Ar	21.2
QSl.cu.7A.e2.1	wPt-4489-wPt-7123	7.49	-0.59	Ar	26.7
QSl.cu.7A.e3.1	wPt-0971	6.55	-0.66	Ar	23.8
QSl.cu.7A.e4.1	wPt-3226	3.81	-0.53	Ar	14.6
QSl.cu.7A.e1.2	wPt-3393-wPt-7299	5.9	-0.64	Ar	21.7
QSl.cu.7A.e2.2	wPt-0205	9.62	-0.69	Ar	32.9
QSl.cu.7A.e3.2	wPt-3393-wPt-7299	6.44	-0.75	Ar	23.4
QSl.cu.7A.e4.2	wPt-3393-wPt-7300	4.4	-0.64	Ar	16.7
QSl.cu.7D.e1	wPt-743331	4.62	0.51	Gr	17.5
QSl.cu.7D.e2	wPt-743332	6.42	0.54	Gr	23.4
QSl.cu.7D.e3	wPt-744841	4.89	0.59	Gr	18.4

a Nomenclature for QTL in wheat: “Q” refers to QTL, followed by trait designator, “cu” for the laboratory, and the chromosome and environment

b E1,E2, E3 and E4 refers to Konya2011, Adıyaman2011, Adana2011 and Adana2010

d refer to additive effect. Positive additive effect indicate increased effect from parent “Gerek” and negative value indicate increase effect parent “Arrehane

e Source of allele from contributing parents

f Percentage of phenotypic variation explained by each QTL

QTL on chromosome 3B “QSl.cu.3B” was also one of the major QTL with LOD score 4.68-7.91, explained 17.-28% of phenotypic variation and increased spike length. This locus for spike length determined in this population has not been previously described in the literature. However, QTLs on 3B, 5B, and 2B showed significance in four of five trials with minor or moderate additive effect (Cui et al. 2012). Marza et al. (2005) found a stable and consistent QTL on 3BL in every environment. QSl.cu.5A on chromosome 5A showed significance in all four trials. It explained up to 25.4 % of the phenotypic variation. . This QTL behave differently in

different environments. In KON-11 environment, it explained only 9.3% proportion of the phenotypic variation, whereas it contributed 25.4% of the spike length variation in ADA-11 environment. One locus determining ear length (*Qel.ocs-5A.1*) was described to be mapped on chromosome arm 5AL (Kato et al. 1999), in a region comparable to the map position of the QTLs shown in Fig. 1.

QTL "QSl.cu.3D" on chromosome 3D was detected in three of four environments at LOD score 5.39-6.6, explained 20-23.9 % of phenotypic variation. Chromosome 6A and 6B each harbored two QTLs. QSl.cu.6A.1 (LOD score 3.27-5.42) was reproducible in all four environments, whereas QSl.cu.6A.2 (LOD score 3.14-6.25) was detected in three of four environments. Both of QTLs on 6A were significant with contribution of 12.2-22.8% and 12.7-20.1% in phenotypic traits respectively depending on the environments. Chromosome 6A contained earliness (Eps) genes (Snape et al. 2001). Since Eps genes influence the number of vegetative and floral primordial and usually affect the spike length, the present QTL(s) may correspond to Eps-6AL. One QTL on chromosome 6B "QSl.cu.6B.1" was major QTL with LOD 5.03-10.18 and was highly significant in contributing 18.8-34.4% of phenotypic variation for longer spike length. Other QTL "QSl.cu.6B.2" was with moderate but consisting effect (LOD score 4.84-5.96), detected in three environments, and explained 18.2-21.9% of the phenotypic variation.

Two major QTLs were discovered on chromosomes 7A. QSl.cu.7A.1 explained 14.6-26.7% of phenotypic variation, whereas QTL 'QSl.cu.7A.2' explained 16.7-32.9% of phenotypic variation. QTL on chromosome 7D "QSl.cu.7D" was detected in three environments and was significant with moderate effect on spike length explaining 17.5-23.4% of the phenotypic variation. Ma et al. (2007) mapped a QTL with strongest effect on spike length. Suenga et al. (2005) have reported a QTL for spike length on 7D chromosomes in the large spike cultivar 'Oligoculm' but it explained less than 10% of phenotypic variation.

4.3.5.2. Number of Spikelets per spike (Sns)

Simple interval mapping for QTL analysis detected eighteen QTLs governing the number of spikelets per spike (Table 4.14). These QTLs were covered on chromosomal regions of 1A, 1B, 1D, 2B, 2D, 3B, 3D, 5B, 6A, 6B, 7A and 7D. Most of the QTLs detected in this study for number of spikelets per spike have negative additive values, indicating contribution from Arrehane, whereas only three chromosomal regions were contributed by Gerek. Some chromosomes harbored more than one QTL. Out of these 18 QTLs, 5 QTLs were with major effect and three were with moderate effect and others were minor QTLs, accounting more than 8% of the phenotypic variations. Five QTLs for number of spikelets per spike could be identified reproducibly in all four trials. Chromosome 2B and 2D were found to have three QTLs each, whereas chromosome 6B and 7A each possessed two QTLs. One QTL (QSns.cu.2D.e2.3) on chromosome 2D was detected only in one environment; one QTL (QSns.cu.2B.1) on chromosome 2B was appeared only in two environments. Cui et al. (2012) reported up to 25 putative additive QTLs for number of spikelets per spike, and were covered all the twenty one wheat chromosomes except 1B, 3D and 6B.

One of the major QTL “QSns.cu.5B” was stable across all four environments, appeared at LOD score 4.65-8.46, exhibited 17.6-29.6% phenotypic variation of the trait. QSns.cu.7A.2 explained 16.8-28.4% of phenotypic variation and was verified at LOD score 4.43-8.04 in all four environments. This QTL was detected on same interval and collocated with the QTL for spike length, both of the QTLs increased spike length and spikelets per spike. Another QTL on 7A was also observed in three of four environments at LOD 2.17-4.06 and contributed up to 15.5 % phenotypic variation. The QTLs for number of spikelets per spike on 7A has not been previously reported. However Li et al. (2007) reported a QTL on 7A chromosome controlling the sterile spikelets per spike in only one environment.

One QTL with main effect on chromosome 1B “QSns.cu.1B” was detected in all environments. This QTL was found at LOD score 2.76-8.96. It explained 10.8-31% of the total phenotypic variation. Another QTL (QSns.cu.3B) with main effect

Table 4.14. QTLs detected in the Gerek x Arrehane mapping population for number of spikelets per spike in four environments

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QSns.cu.1A.e2	wPt-1720	4.12	-0.61	Ar	15.7
QSns.cu.1A.e3	wPt-1720	5.91	-0.79	Ar	21.7
QSns.cu.1A.e4	wPt-1720	3.84	-0.64	Ar	14.7
QSns.cu.1B.e1	wPt-0944-	4.33	0.51	Gr	16.4
QSns.cu.1B.e2	tPt-0283-wPt-3475	8.96	0.93	Gr	31
QSns.cu.1B.e3	wPt-2526-	6.44	0.85	Gr	23.4
QSns.cu.1B.e4	tPt-0283-wPt-3475	2.76	0.57	Gr	10.8
QSns.cu.1D.e2		3.47	-0.64	Ar	13.4
QSns.cu.1D.e3		3.36	-0.70	Ar	13
QSns.cu.2B.e2.1	wPt-6120	3.37	-0.54	Ar	13.1
QSns.cu.2B.e3.1	wPt-1813-wPt-0746	3.49	-0.67	Ar	13.5
QSns.cu.2B.e1.2	wPt-741382	2.06	-0.36	Ar	8.2
QSns.cu.2B.e2.2	wPt-741382	3.93	-0.59	Ar	15.1
QSns.cu.2B.e3.2	wPt-741382	4.29	-0.70	Ar	16.3
QSns.cu.2B.e4.2	wPt-741382	2.16	-0.54	Ar	8.6
QSns.cu.2B.e2.3		3.36	-0.65	Ar	13
QSns.cu.2B.e3.3	wPt-665014	2.21	-0.53	Ar	8.8
QSns.cu.2B.e4.3	wPt-743630	2.73	-0.65	Ar	10.7
QSns.cu.2D.e2.1	cfd36-wPt-3812	2.15	-0.38	Ar	8.5
QSns.cu.2D.e3.1	cfd36-wPt-3812	2.89	-0.55	Ar	11.3
QSns.cu.2D.e4.1	cfd36-wPt-3812	3.46	-0.66	Ar	13.4
QSns.cu.2D.e2.2	wPt-3757-wPt-665972	6.3	-0.73	Ar	23
QSns.cu.2D.e3.2	wPt-3757-wPt-665972	2.25	-0.53	Ar	8.9
QSns.cu.2D.e4.2	wPt-2652-wPt-4242	2.57	-0.58	Ar	10.1
QSns.cu.2D.e2.3	wPt-671742	4.45	-0.68	Ar	16.8
QSns.cu.3B.e1	wPt-2766	4.51	0.50	Gr	17.1
QSns.cu.3B.e2	wPt-0086-wPt-7486	5.2	0.70	Gr	19.4
QSns.cu.3B.e3	wPt-664393	6.13	0.81	Gr	22.5
QSns.cu.3D.e1	wPt-742488	2.49	-0.38	Ar	9.8
QSns.cu.3D.e2	wPt-742488	5.2	-0.66	Ar	19.4
QSns.cu.3D.e3	wPt-742488	5.61	-0.77	Ar	20.8
QSns.cu.5B.e1	gwm554-wPt-5604	4.65	-0.59	Ar	17.6
QSns.cu.5B.e2	gwm554-wPt-5604	7.14	-0.87	Ar	25.6
QSns.cu.5B.e3	gwm554-wPt-5604	8.46	-1.05	Ar	29.6
QSns.cu.5B.e4	wPt-6191-gwm408	8.46	-1.05	Ar	29.6
QSns.cu.6A.e1	wPt-671568	2.51	-0.39	Ar	9.9
QSns.cu.6A.e2	wPt-671568	7.77	-0.80	Ar	27.6
QSns.cu.6A.e3	wPt-671568	5.53	-0.78	Ar	20.5
QSns.cu.6B.e1.1	wPt-745074	2.65	-0.40	Ar	10.4

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QSns.cu.6B.e2.1	wPt-745074	6.75	-0.75	Ar	24.4
QSns.cu.6B.e3.1	wPt-745074	6.33	-0.83	Ar	23.1
QSns.cu.6B.e4.1	wPt-745074	3.04	-0.59	Ar	11.9
QSns.cu.6B.e1.2	wPt-8412-wPt-4312	3.94	-0.66	Ar	15.1
QSns.cu.6B.e2.2	wPt-664250	4.98	-0.75	Ar	18.7
QSns.cu.6B.e3.2	wPt-2175	2.96	-0.60	Ar	11.6
QSns.cu.7A.e2.1	wPt-4489-wPt-7122	4.06	-0.62	Ar	15.5
QSns.cu.7A.e3.1	wPt-4489-wPt-7122	2.76	-0.58	Ar	10.8
QSns.cu.7A.e4.1	wPt-5641	2.17	-0.49	Ar	8.6
QSns.cu.7A.e1.2	wPt-3393-wPt-7299	5.1	-0.62	Ar	19.1
QSns.cu.7A.e2.2	wPt-3393-wPt-7399	8.04	-0.92	Ar	28.4
QSns.cu.7A.e3.2	wPt-3393-wPt-7399	6.84	-0.99	Ar	24.7
QSns.cu.7A.e4.2	wPt-3393-wPt-7399	4.43	-0.80	Ar	16.8
QSns.cu.7D.e1	wPt-743331	3.23	0.44	Gr	12.5
QSns.cu.7D.e2	wPt-7076	4.2	0.60	Gr	16
QSns.cu.7D.e3	wPt-7076	4.29	0.69	Gr	16.3

a Nomenclature for QTL in wheat: "Q" refers to QTL, followed by trait designator, "cu" for the laboratory, and the chromosome and environment
b E1,E2, E3 and E4 refers to Konya 2011, Adıyaman2011, Adana2011 and Adana2010
d refer to additive effect. Positive additive effect indicate increased effect from parent "Gerek" and negative value indicate increase effect parent "Arrehane
e Source of allele from contributing parents
f Percentage of phenotypic variation explained by each QTL

was discovered at 3B chromosome in three out of four environments, which explained 17.1-22.5% phenotypic variation. Narasimhamoorthy et al. (2006) detected one QTL on 3B for SNS in hard winter wheat/synthetic wheat population, whereas Huang et al. (2003) located a QTL on 3BS chromosome in Prinz/W7984 population. A QTL with medium effect explaining 9.8-20.8% of phenotypic variation was located on chromosome 3D. Wang et al. (2011) also detected a QTL with main effect on 3BL and another major QTL on 3D chromosomes.

On 6B chromosome, two consistent QTLs "QSns.cu.6B.1 and QSns.cu.6B..2" were observed at LOD score 2.65-6.75 and 2.96-4.98, contributing 10.4-24.4% and 11.6-15.1% of phenotypic variation respectively. Another major QTL "QSns.cu.6A" was detected on chromosome 6A in three environments, exhibited 9.9-27.6% of phenotypic variation. QTL "QSns.cu.1A" was detected in three environments and explained 14.7-21.7% of the phenotypic variation.

One QTL "QSns.cu.7D" was located on 7D chromosome and detected at LOD 3.23-4.29 in three environments and explained 12.5-16.3 of phenotypic variation. This QTL was also collocated with QTL controlling the shorter spike length contributed by Gerek. Ma et al. (2007) detected seven QTLs for spikelet number per spike in three trials. They described that QTL mapped on 7D consistently showed large effects on spikelet number per spike and was collocated with the QTL governing the spike length.

Three QTLs with minor effects were detected on chromosome 2D. Out of these three QTLs, one QTL "QSns.cu.2D.3" was detected only in one environment and explained 16.8% of the total phenotypic variation. Other two QTLs on 2D were located in three environments with small effect on the phenotypic variation. Ma et al. (2007) also described second strongest QTL for spikelets number per spike on chromosome 2D.

4.3.5.3. Number of grains per spike; (Gns)

Twenty genomic regions were associated with number of grains per spike in Gerek x Arrehane mapping population (Table 4.15). These QTLs were located on different chromosomes such as 1A, 1B, 1D, 2B, 2D, 3B, 3D, 4D, 5A, 5B, 6A, 6B, 6D, 7A and 7D. For most of these QTLs, Arrehane allele caused in an increase in number of grains per spike with a phenotypic effect of 8.7-25.7%. Six QTLs were transferred by parent Gerek with decrease in grain number per spike. QTLs on chromosomes 1D, 5A and 6A were detected only in one environment.

One QTL "QGns.cu.1B" with strongest effect was detected on chromosome 1B under three environments with LOD 3.01-7.15, explained 11.7-25.7% phenotypic variation depending on the environments. Marza et al. (2006) also reported a QTL on 1B at LOD 5.1, explaining 12% of the phenotypic variation. Chromosome 7A harbored two QTLs. Both of them have positive effect on grain number. One QTL 'QGns.cu.7A.2' with strong effect was observed in two environments at LOD 4.65-5.26, and explained 17.5-19.6% of total phenotypic variation. Other QTL on 7A 'QGns.cu.7A.1' was with moderate effect contributing 10.2-14.6% phenotypic

Table 4.15. QTLs detected in the Gerek x Arrehane mapping population for number of grains per spike in four environments

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QGns.cu.1A.e1	wPt-1720-wPt-7951	4.68	-2.81	Ar	17.6
QGns.cu.1A.e2	wPt-1720-wPt-7951	3.38	-2.07	Ar	13.1
QGns.cu.1A.e3	wPt-1720-wPt-7951	2.2	-1.82	Ar	8.7
QGns.cu.1A.e4	wPt-9429	3.51	-2.51	Ar	13.5
QGns.cu.1B.e1	tPt-0283-wPt-3475	3.46	2.67	Gr	13.4
QGns.cu.1B.e2	wPt-4688-wPt-742929	7.15	2.82	Gr	25.7
QGns.cu.1B.e3	wPt-5006-rPt-7906	3.01	-2.70	Ar	11.7
QGns.cu.1D.e1.1	wPt-671869-wPt-8854	4.11	-2.75	Ar	15.7
QGns.cu.1D.e2.1	wmc429-wmc216	2.52	-1.98	Ar	9.9
QGns.cu.1D.e4.1	wmc216-wPt-6316	3.23	-2.42	Ar	12.5
QGns.cu.1D.e1.2	wPt-2861-wPt-8866	3.3	-2.19	Ar	12.8
QGns.cu.2B.e1	wPt-1394	4.73	-2.75	Ar	17.8
QGns.cu.2B.e2	wPt-741382-wPt-4072	4.35	-2.36	Ar	16.5
QGns.cu.2B.e3	wPt-8776	3.31	-2.29	Ar	12.8
QGns.cu.2D.e1.1	CFD43	3.3	-2.43	Ar	12.8
QGns.cu.2D.e2.1	CFD43	2.63	-1.85	Ar	10.4
QGns.cu.2D.e3.1	CFD43	3.65	-2.66	Ar	14.1
QGns.cu.2D.e2.2	cfd233-wPt-731941	3.31	-2.24	Ar	12.8
QGns.cu.2D.e3.2	wPt-2652-wPt-4242	2.92	-2.24	Ar	11.4
QGns.cu.2D.e4.2	wmc18-wPt-0936	5.25	-3.19	Ar	19.6
QGns.cu.3B.e1.1	wPt-798970	2.79	2.08	Gr	10.9
QGns.cu.3B.e2.1	wPt-798970	3.04	1.83	Gr	11.9
QGns.cu.3B.e1.2	wPt-732044	5.12	2.75	Gr	19.1
QGns.cu.3B.e2.2	wPt-732044	3.29	1.89	Gr	12.7
QGns.cu.3B.e3.2	wPt-732044	2.37	1.84	Gr	9.4
QGns.cu.3B.e1.3	wPt-6961-wPt-10071	2.8	2.46	Gr	11
QGns.cu.3B.e2.3	wPt-6961-wPt-10071	2.48	1.80	Gr	9.8
QGns.cu.3B.e3.3	wPt-6961-wPt-10071	2.48	2.21	Gr	9.8
QGns.cu.3D.e1	wPt-742488	5	-2.73	Ar	18.7
QGns.cu.3D.e2	wPt-742488	3.29	-1.90	Ar	12.8
QGns.cu.3D.e3	wPt-4476	3.95	-2.35	Ar	15.1
QGns.cu.4D.e1	wPt-664809	4.68	-2.84	Ar	17.6
QGns.cu.4D.e2	wPt-664809	2.36	-1.74	Ar	9.3
QGns.cu.4D.e3	wPt-664809	2.33	-2.14	Ar	9.2
QGns.cu.5A.e1	wPt-800634	3.18	-2.37	Ar	12.4
QGns.cu.5B.e1	wPt-5928-wPt-2707	4.08	-2.61	Ar	15.6
QGns.cu.5B.e2	wPt-5928-wPt-2707	4.37	-2.26	Ar	16.6
QGns.cu.5B.e3	wPt-5928-wPt-2707	3.2	-2.21	Ar	12.4
QGns.cu.5B.e4	wPt-6191-gwm408	5.38	-3.20	Ar	20

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QGns.cu.6A.e1	wPt-671558	2.6	-1.90	Ar	8.8
QGns.cu.6B.e1	wPt-6667	3.63	-2.37	Ar	14
QGns.cu.6B.e2	wPt-6667	5.33	-2.38	Ar	19.8
QGns.cu.6B.e3	wPt-4706	3.21	-2.15	Ar	12.5
QGns.cu.6B.e4	wPt-1756	3.62	-2.48	Ar	13.9
QGns.cu.6D.e1	wPt-729933	3.31	1.90	Gr	12.8
QGns.cu.7A.e1.1	wPt-4489-wPt-7122	3.8	-2.52	Ar	14.6
QGns.cu.7A.e2.1	wPt-4489	3.22	-1.87	Ar	12.5
QGns.cu.7A.e4.1	wPt-4489-wPt-7122	2.59	-2.16	Ar	10.2
QGns.cu.7A.e1.2	wPt-7299-wmc603	4.65	-2.73	Ar	17.5
QGns.cu.7A.e2.2	wPt-3393-wPt-7299	5.26	-2.75	Ar	19.6
QGns.cu.7D.e1	wPt-743331	2.98	2.20	Gr	11.6
QGns.cu.7D.e2	wPt-664320	4.58	2.23	Gr	17.3
QGns.cu.7D.e3	wPt-7076	2.69	1.97	Gr	10.5
QGns.cu.7D.e4	wPt-7076	2.26	1.98	Gr	8.9

a Nomenclature for QTL in wheat: "Q" refers to QTL, followed by trait designator, "cu" for the laboratory, and the chromosome and environment
b E1,E2, E3 and E4 refers to Konya2011, Adıyaman2011, Adana2011 and Adana2010
d refer to additive effect. Positive additive effect indicate increased effect from parent "Gerek" and negative value indicate increase effect parent "Arrehane
e Source of allele from contributing parents
f Percentage of phenotypic variation explained by each QTL.

variation discovered at LOD score. 2.59-3.8. Huang et al. (2004) also detected two QTLs on 7A chromosome in advanced back cross QTL approach from a German winter wheat with synthetic wheat.

On chromosome 5B, one stable and effective QTL at LOD score 3.2-5.38 was detected in all four environments and contributed 12.4-20% of the phenotypic variation explained. Three different QTLs were located on chromosome 3B. One QTL with major effect and other QTLs were minor in their effects. QTL 'QGns.cu.3B.2" explained 9.4-19.1% of the phenotypic variation, detected at LOD score 2.37-5.12 in three environments, whereas other two QTLs were detected at LOD 2.79-3.04 (found in two environments) and 2.48-2.8 (detected in three environments) respectively. QTLs controlling grain number per spike have been reported on 3B by different researchers (Marza et al. 2006; Huang et al. 2004).

One and two QTLs were identified on chromosome 1A and 1D respectively. QTL on 1A was stable across all four environments, identified at LOD 2.2-4.68 and contributed 8.7-17.6% of the phenotypic variation for the trait. One QTL on chromosome 1D was stable and isolated in three environments and other was detected only in one environment. Kumar et al. (2007) reported and identified one genomic region on each of 1A and 1D controlling grain number per spike in wheat.

One QTL with strong effect was located on chromosome 2B and two QTLs with moderate effect was located on chromosome 2D. On chromosome 2B, 'QGns.cu.2B' was found in three environments at LOD score 3.31.4.73. Both QTLs on chromosome 2D were detected in three out of four environments and were moderate in expressing the traits.

One major QTL was detected on chromosome 3D in three environments, with average LOD score 4.08, explained 12.8-18.8% of total phenotypic variation for the trait. QTL 'QGns.cu.4D' with medium effect was also observed in three locations at LOD score 2.33-4.68, flanked with marker 'wPt-6648091 and explained 9.2-17.6% of phenotypic variations. One QTL 'QGns.cu.5A' on chromosome 5A with medium effect and one other QTL 'QGns.cu.6A' on chromosome 6A with minor effect was detected on only in one environment.

One stable and consistent QTL over all four environments with moderate effect was detected on chromosome 7D, at LOD score 2.26-4.58, and explained 8.9-17.3% phenotypic variation for number of grains per spike. Most of the QTLs for grain number per spike detected in the present study have not been reported in previous mapping studies (Li et al. 2007; Wang et al. 2012)

4.3.5.4. QTLs for grain weight per spike (Gws)

Simple interval mapping for QTL analysis identified several genomic regions associated with grain weight per spike. Twenty six QTLs were identified on different chromosomes (Table 4.16). These QTLs explained 8.9-33.7% of the phenotypic variation depending on the QTL and the environment. Again most of these QTLs showed positive effect coming from allele associated with Arrehane except six QTL

located on 1B, 3B, 6D and 7D. However most of the QTLs identified to be associated with grain weight per spike were unstable, detected in one or two environments. Out of these 26 QTLs, four QTLs were detected only in one of the four environments, whereas sixteen QTLs were observed in two of the four environments. Rest of the QTLs (6 QTLs) was discovered in three out of four environments. Highest number of QTLs was only identified in environment2 (ADM-11). No QTL was detected in 'ADA-10' environment.

Out of four QTLs, which were unstable and identified only in one environments, 2 QTLs were located on chromosomes 2D, while one QTL were located on each of chromosomes 4D, 5A, 6B. All of these QTLs were specific to ADA-11 environment. Adıyman and Konya environments are less favorable and stress environment compared with Adana environment. As both of the parents are suitable for stress environments, which could results in expression of the QTL in these environments.

Sixteen QTLs were identified in two of the four environments. Most of these QTLs were identified in KON-11 and ADM-11 environments. These were located on chromosomes 1B, 1D, 2B, 2D, 3B, 3D, 5B, 6B, 6D, 7A and 7B. Five QTLs were consistent in three out of four environments. These were located on 1A, 2B, 2D, 3A, 3B and 7D.

QTL 'QGws.cu.5B.e1.1' with strongest effect was detected on 5B in two environments at LOD score of 5.22-9.92, explained 11.5-33.7% of phenotypic variation for the trait. This chromosome harbored two QTLs. Other QTL with minor effect was also detected in two environments and explained 12.3-17.3% of phenotypic variation.

Table 4.16. QTLs detected in the Gerek x Arrehane mapping population for grain weight per spike in four environments

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QGws.cu.1A.e1	wPt-1720-wPt-7951	5.7	-0.16	Ar	21
QGws.cu.1A.e2	wPt-1720-wPt-7951	8.25	-0.16	Ar	29
QGws.cu.1A.e3	wPt-1720-wPt-7951	3.46	-0.12	Ar	13.5
QGws.cu.1B.e1	tPt-0283-wPt-3475	3.45	0.13	Gr	13.3
QGws.cu.1B.e2	wPt-742929	6.64	0.14	Gr	24.1
QGws.cu.1D.e1.1	wPt-671869-wPt-8854	4.94	-0.16	Ar	18.5
QGws.cu.1D.e2.1	wPt-671869-wPt-8854	2.86	-0.10	Ar	11.2
QGws.cu.1D.e1.2	wPt-5721-wPt-664829	2.44	-0.12	Ar	9.6
QGws.cu.1D.e2.2	wPt-5721-wPt-664829	5.81	-0.15	Ar	21.4
QGws.cu.2B.e1.1	wPt-5759	3.36	-0.13	Ar	13
QGws.cu.2B.e2.1	wPt-6627	4.54	-0.12	Ar	17.2
QGws.cu.2B.e1.2	wPt-741382-wPt-4072	3.82	-0.15	Ar	14.7
QGws.cu.2B.e2.2	wPt-741382-wPt-4072	6.47	-0.15	Ar	23.5
QGws.cu.2B.e3.2	wPt-0694-tPt-9767	3.6	-0.12	Ar	14
QGws.cu.2B.e1.3	wPt-665014	3.05	-0.12	Ar	11.9
QGws.cu.2B.e2.3	wPt-7883-wPt-743630	4.71	-0.15	Ar	17.7
QGws.cu.2D.e2.1	CFD56-wPt-667101	3.88	-0.13	Ar	14.9
QGws.cu.2D.e2.2	cfd36-wPt-3812	3.33	-0.11	Ar	12.9
QGws.cu.2D.e1.3	CFD43	3.64	-0.13	Ar	14
QGws.cu.2D.e2.3	wPt-9780-gwm102	4.32	-0.12	Ar	16.4
QGws.cu.2D.e1.4	wmc18-wPt-0936	3.06	-0.13	Ar	11.9
QGws.cu.2D.e2.4	cfd233-wPt-731941	7.15	-0.17	Ar	25.7
QGws.cu.2D.e3.4	cfd233	2.28	-0.09	Ar	9.1
QGws.cu.3A.e1	wPt-5133-wPt-5886	2.4	-0.12	Ar	9.5
QGws.cu.3A.e2	wPt-7496-wPt-730156	4.15	-0.11	Ar	15.8
QGws.cu.3A.e3	wPt-7496-wPt-730156	3.04	-0.11	Ar	12
QGws.cu.3B.e1.1	wPt-2757	2.94	0.11	Gr	11.5
QGws.cu.3B.e2.1	wPt-2757	3.48	0.10	Gr	13.4
QGws.cu.3B.e1.2	wPt-10196	7.72	0.18	Gr	27.4
QGws.cu.3B.e2.2	wPt-11278	6.76	0.14	Gr	24.5
QGws.cu.3B.e1.3	wPt-7158	3.16	0.13	Gr	12.3
QGws.cu.3B.e2.3	wPt-6961-wPt-10071	3.88	0.12	Gr	14.9
QGws.cu.3B.e3.3	wPt-6961-wPt-10071	2.52	0.11	Gr	10
QGws.cu.3D.e1	wPt-742488	4.63	-0.14	Ar	17.5
QGws.cu.3D.e2	wPt-742488	6.29	-0.13	Ar	23
QGws.cu.4D.e2	wPt-667924	5.91	-0.17	Ar	21.7
QGws.cu.5A.e2	cfa2190-wPt-0605	4.76	-0.13	Ar	17.9
QGws.cu.5B.e1.1	gwm554-wPt-5604	5.22	-0.17	Ar	19.5
QGws.cu.5B.e2.1	wPt-2607	9.92	-0.16	Ar	33.7

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QGws.cu.5B.e1.2	wPt-4907	3.15	-0.12	Ar	12.3
QGws.cu.5B.e2.2	wPt-4907	4.57	-0.12	Ar	17.3
QGws.cu.6B.e1.1	wPt-7954	3.62	-0.13	Ar	13.9
QGws.cu.6B.e2.1	wPt-7954	7.94	-0.15	Ar	28.1
QGws.cu.6B.e2.2	wPt-8412-wPt-4312	4.01	-0.13	Ar	15.3
QGws.cu.6D.e1	wPt-6163-wPt-666593	2.25	0.11	Gr	8.9
QGws.cu.6D.e2	wPt-6163-wPt-666593	5.17	0.14	Gr	19.3
QGws.cu.7A.e1.1	wPt-4489-wPt-7122	3.66	-0.13	Ar	14.1
QGws.cu.7A.e2.1	wPt-4489	6.3	-0.13	Ar	23
QGws.cu.7A.e1.2	wPt-3393-wPt-7299	6.2	-0.15	Ar	22.7
QGws.cu.7A.e2.2	wPt-0205	3.43	-0.12	Ar	13.4
QGws.cu.7B.e1	GWM537-wPt-671801	3.6	-0.11	Ar	13.9
QGws.cu.7B.e2	GWM537-wPt-671801	2.28	-0.10	Ar	9.1
QGws.cu.7D.e1	wPt-7076	4.13	0.13	Gr	15.8
QGws.cu.7D.e2	wPt-7076	7.62	0.15	Gr	27.1
QGws.cu.7D.e3	wPt-743708	3.28	0.11	Gr	12.8

a Nomenclature for QTL in wheat: "Q" refers to QTL, followed by trait designator, "cu" for the laboratory, and the chromosome and environment
b E1,E2, E3 and E4 refers to Konya2011, Adıyaman2011, Adana2011 and Adana2010
d refer to additive effect. Positive additive effect indicate increased effect from parent "Gerek" and negative value indicate increase effect parent "Arrehane
e Source of allele from contributing parents
f Percentage of phenotypic variation explained by each QTL

Chromosome 1A harbored one QTL 'QGws.cu.1A' at LOD score 3.46-8.25 in three environments, with major affect explaining 13.5-29% of phenotypic variation depending upon environments. Chromosome 1B also contained one QTL with major effect (with 13.3-24.1% of phenotypic variation), in two locations.

Two major QTLs were located on chromosome 7A. Both were observed in two environments. QGws.cu.7A.1 and QGws.cu.7A.2 were observed in KON-11 and DM-11 environments, at LOD 3.66-6.3 and 3.43-6.2, and explained 14.1-23% and 13.4-22.7% of phenotypic variation.

Another major QTL 'QGws.cu.3B.2' was found on chromosome 3B in two environments at high LOD score 6.76-7.72. This QTL explained 24.5-27.4 % of phenotypic variation according to tested environment. Other major QTLs were located on chromosome 2B, 2D and 3D. QTL 'QGws.cu.2B.2' was consistent and stable QTL over three environments, with major effect 14-23.5% of phenotypic

varaition. One major and consistent QTL ‘QGws.cu.2D.4’ was observed in three environments and contributed 25.7% phenotypic variation. QTL ‘QGws.cu.3D was also major QTL, identified in two locations at LOD score 4.63-6.29. This QTL explained 17.5-23% of phenotypic variation. Chromosome 6B also harbored a major QTL in two environments, at LOD score 3.62-7.94. Another QTL with major effect detected on chromosome 7D (discovered in three environments) at LOD score 3.28-7.62, and explained 12.8-27.1% of phenotypic variation.

Several other QTLs with minor effect were detected on different chromosomes. One QTL on each of 2B (QGws.cu.2B.3) and 2D (QGws.cu.2D.3) chromosome were identified in two environments and explained 11.9-17.7% and 14-16.4% of phenotypic variation. Another QTL (QGws.cu.3A) with minor effect but stable in three environments was detected on 3A chromosome at LOD score 2.4-4.15, and contributed 9.5-15.8% of phenotypic variations. Other QTL on 3B (QGws.cu.3B.1) was discovered in two environments, and explained 11.5-13.4% of phenotypic variation. Chromosome 3B harbored one minor QTL (QGws.cu.3B.3) in three locations and explained 10-14.9% of phenotypic variation. Other minor QTLs were also defined in chromosome 5B (QGws.cu.5B.2), 6D (QGws.cu.6D), 7B (QGws.cu.7B) (each observed in two locations).

4.3.5.5. Spike compactness (Sc)

Interval mapping revealed twenty genomic regions involved in controlling the density of spikelets on spike (spike compactness) (Table 4.17). Ten genomic regions were with major and direct effect on the spike density, whereas rests of the ten QTLs were with minor to moderate effect. Most of the QTLs for spike compactness were also sourced from allele of Arrehane. QTLs for spike compactness were distributed on 15 chromosomes (1A, 1B, 1D, 2B, 2D, 3B, 3D, 4A, 4B, 5A, 5B, 6A, 6B, 7A and 7D) of bread wheat. Some chromosomal regions harbored more than one QTL such as 2B, 4A and 6B. Most of the QTLs for spike compactness were stable QTLs detected in all four environments and most of them were in the vicinity of the QTLs for other spike related traits.

Table 4.17. QTLs detected in the Gerek x Arrehane mapping population for spike compactness in four environments

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QSc.cu.1A.e1	wPt-1720-wPt-7951	8.99	-0.03	Ar	31.1
QSc.cu.1A.e2	wPt-1720-wPt-7951	8.54	-0.02	Ar	29.8
QSc.cu.1A.e3	wPt-1720-wPt-7951	6.76	-0.02	Ar	24.5
QSc.cu.1A.e4	wPt-1720-wPt-7951	3.89	-0.03	Ar	14.9
QSc.cu.1B.e1	tPt-0283-wPt-3475	3.1	0.02	Gr	12.1
QSc.cu.1B.e2	tPt-0283-wPt-3475	3.32	0.01	Gr	12.9
QSc.cu.1B.e3	wPt-6975	3.01	-0.01	Ar	11.7
QSc.cu.1D.e1	wPt-671869	4.81	-0.02	Ar	18.1
QSc.cu.1D.e2	wPt-671869	2.22	-0.01	Ar	8.8
QSc.cu.1D.e3	wPt-671869	3.57	-0.02	Ar	13.8
QSc.cu.1D.e4	wPt-671869	2.49	-0.02	Ar	9
QSc.cu.2B.e1.1	wPt-1813	7.24	-0.02	Ar	26
QSc.cu.2B.e2.1	wPt-1813	4.5	-0.02	Ar	17
QSc.cu.2B.e3.1	wPt-1813	4.46	-0.02	Ar	16.9
QSc.cu.2B.e4.1	wPt-1813	3.3	-0.02	Ar	12.8
QSc.cu.2B.e1.2	wPt-741382-wPt-4072	8.04	-0.03	Ar	28.3
QSc.cu.2B.e2.2	wPt-741382-wPt-4072	6.83	-0.02	Ar	24.7
QSc.cu.2B.e3.2	wPt-741382	5.76	-0.02	Ar	21.3
QSc.cu.2B.e4.2	wPt-741382-wPt-4072	2.47	-0.02	Ar	9.7
QSc.cu.2B.e1.3	wPt-7883-wPt-743630	3.43	-0.02	Ar	13.2
QSc.cu.2B.e2.3	wPt-7883-wPt-743630	4.82	-0.02	Ar	18.1
QSc.cu.2B.e3.3	wPt-7883-wPt-743630	3.47	-0.02	Ar	13.4
QSc.cu.2B.e4.3	wPt-7883-wPt-743630	3.29	-0.03	Ar	12.8
QSc.cu.2B.e1.4	wPt-2266	3.99	-0.02	Ar	15.2
QSc.cu.2B.e2.4	wPt-2266	4.16	-0.02	Ar	15.9
QSc.cu.2B.e3.4	wPt-2266	4.55	-0.02	Ar	17.2
QSc.cu.2D.e1	wPt-3757-wPt-665972	5.95	-0.02	Ar	21.9
QSc.cu.2D.e2	wPt-4413	8.96	-0.02	Ar	31
QSc.cu.2D.e3	wPt-3757-wPt-665972	7.47	-0.03	Ar	26.7
QSc.cu.2D.e4	wPt-4413	3.31	-0.02	Ar	12.8
QSc.cu.3B.e1	wPt-9170	7.29	0.02	Gr	26.1
QSc.cu.3B.e2	wPt-9170	4.19	0.01	Gr	15.9
QSc.cu.3B.e3	wPt-9170	5.35	0.02	Gr	19.9
QSc.cu.3B.e4	wPt-9170	2.46	0.02	Gr	9.7
QSc.cu.3D.e1	wPt-742488	6.1	-0.02	Ar	22.4
QSc.cu.3D.e2	wPt-742488	3.09	-0.01	Ar	12
QSc.cu.3D.e3	wPt-742488	5.13	-0.02	Ar	19.2
QSc.cu.3D.e4	wPt-741682	2.62	-0.02	Ar	10.3
QSc.cu.4A.e1.1	wPt-7807	2.54	-0.02	Ar	10

QTL[a]	Marker interval	LOD	A[d]	S[e]	R^2 (%)[f]
QSc.cu.4A.e2.1	wPt-7807	4.55	-0.01	Ar	15.9
QSc.cu.4A.e3.1	wPt-7807	3.54	-0.02	Ar	13.7
QSc.cu.4A.e4.1	wPt-7807	2.54	-0.02	Ar	10
QSc.cu.4A.e1.2	wmc262	2.39	-0.02	Ar	9.4
QSc.cu.4A.e2.2	wmc262	6.74	-0.02	Ar	24.4
QSc.cu.4A.e3.2	wmc262	4.94	-0.02	Ar	18.5
QSc.cu.4A.e4.2	wmc262	3.58	-0.02	Ar	13.8
QSc.cu.4B.e2	wPt-6869	4.12	-0.01	Ar	15.7
QSc.cu.4B.e3	wPt-6869	4.81	-0.02	Ar	18.1
QSc.cu.5A.e2	wPt-4279-gwm293	4.13	-0.02	Ar	15.7
QSc.cu.5A.e3	wPt-4279-gwm293	6.21	-0.02	Ar	22.7
QSc.cu.5A.e4	wPt-4279	3.81	-0.03	Ar	14.6
QSc.cu.5B.e1	gwm554-wPt-5604	11.84	-0.03	Ar	38.8
QSc.cu.5B.e2	tPt-7755	11.79	-0.02	Ar	38.7
QSc.cu.5B.e3	gwm554-wPt-5604	9.01	-0.03	Ar	31.2
QSc.cu.5B.e4	tPt-7755-wPt-1409	3.93	-0.03	Ar	15.1
QSc.cu.6A.e1	wPt-729877-wPt-0228	5.2	-0.02	Ar	19.4
QSc.cu.6A.e2	wPt-729877-wPt-0228	5.2	-0.02	Ar	19.4
QSc.cu.6A.e3	wPt-729877-wPt-0228	4.82	-0.02	Ar	18.1
QSc.cu.6A.e4	wPt-729877-wPt-0228	2.59	-0.02	Ar	10.2
QSc.cu.6B.e1.1	wPt-6667	6.23	-0.02	Ar	22.8
QSc.cu.6B.e2.1	wPt-6667	6.83	-0.02	Ar	24.7
QSc.cu.6B.e3.1	wPt-6667	4.44	-0.02	Ar	16.8
QSc.cu.6B.e4.1	wPt-6667	3.12	-0.02	Ar	12.1
QSc.cu.6B.e1.2	wPt-0151	3.14	-0.02	Ar	12.2
QSc.cu.6B.e2.2	wPt-4930-wPt-1241	5.27	-0.02	Ar	19.6
QSc.cu.6B.e3.2	wPt-4930-wPt-1241	3.42	-0.02	Ar	13.2
QSc.cu.7A.e1	wPt-4489-wPt-7122	6.79	-0.02	Ar	24.5
QSc.cu.7A.e2	wPt-4489-wPt-7122	5.57	-0.02	Ar	20.6
QSc.cu.7A.e3	wPt-4489-wPt-7122	4.01	-0.02	Ar	15.3
QSc.cu.7A.e4	wPt-5641	3.05	-0.02	Ar	11.9
QSc.cu.7D.e1	wPt-744841	3.45	0.02	Gr	13.3
QSc.cu.7D.e2	wPt-744841	3.95	0.01	Gr	15.1
QSc.cu.7D.e3	wPt-664320	2.81	0.01	Gr	11

a Nomenclature for QTL in wheat: "Q" refers to QTL, followed by trait designator, "cu" for the laboratory, and the chromosome and environment

b E1,E2, E3 and E4 refers to Konya2011, Adıyaman2011, Adana2011 and Adana2010

d refer to additive effect. Positive additive effect indicate increased effect from parent "Gerek" and negative value indicate increase effect parent "Arrehane

e Source of allele from contributing parents

f Percentage of phenotypic variation explained by each QTL

One of the major QTL with strongest effect ‘QSc.cu.5B’ was detected on chromosome 5B, at highest LOD score up to 11.84, flanked with marker interval ‘gwm554-wPt-5604’ or ‘tPt-7755’ depending on the environments studied. This QTL was stable around all locations and explained 15.1-38.8% of the phenotypic variation, and allele for this QTL sourced from Arrehane and resulted in less dense spikes. Another major QTL ‘QSc.cu.1A’ with strong contribution (14.9-31.1% of phenotypic variation explained) was located on chromosome 1A in Gerek x Arrehane mapping population, appeared at high LOD value 3.89-8.99 in all four environments. Chromosome 2B harbored four QTLs for spike compactness.

Two QTLs were major effect and other two were moderate effect QTLs. QSc.cu.2B.2 was consistent QTL and identified at LOD score 2.47-8.04 and explained up to 28.3% of phenotypic variation. Next to this QTL was QSc.cu.2B.1 on chromosomes 2B and explained up to 26% of phenotypic variation. Other two moderate effect QTLs ‘QSc.cu.2B.3 and QSc.cu.2B.4’ were discovered at LOD up to 4.82 and 4.55 and explained up to 18.1 and 17.2 % of phenotypic variation respectively.

Three other major QTLs were located chromosome 2D, 3B and 3D. QSc.cu.2D flanked with marker interval ‘wPt-3757-wPt-665972’ in environment 1 and environment 3 and with marker ‘wPt-4413’ in environment 2 & 4. This QTL was consistent in all environments and detected at LOD 3.31-8.96 and explained up to 31 % of the phenotypic variation. QSc.cu.3B. was identified on chromosome 3B at LOD score low as 2.46 in environment 4 and up to 7.29 in environment 1, and contributed up to 26.1% of the phenotypic variation. Other QTL was ‘QSc.cu.3D’ on chromosome 3D, flanked with marker ‘wPt-742488’ at LOD score ranging between 2.62-6.1 and explained 10.3-22.4% of phenotypic variation.

Chromosome 4A, 5A, 6B and 7A each harbored one major QTL on them. QSc.cu.6B.1 was major QTL with closest marker ‘wPt-6667’ at LOD 3.12-6.83 and 12.1-24.7 % phenotypic variation. QSc.cu.5A on chromosome 5A was detected in three out of four environments at LOD up to 6.21. This QTL flanked with marker interval ‘wPt-4279-gwm293’ and explained 14.6-22.7% of the phenotypic variations. QSc.cu.7A. was stable and consistent over locations/environments at LOD score

3.05-6.79, flanked with marker interval 'wPt-4489-wPt-7122', explained 11.9-24.5% of phenotypic variation.

Other medium or moderate effect QTLs were located on 1B, 1D, 4A, 4B and 7D chromosomes. QTL on chromosome 1B and 7D were minor QTLs. Both QTLs contributed maximum 12.9 and 15.1 % of phenotypic variation respectively. The QTLs on the chromosome 1D, 4A, 4B, 6A, 6B and 7D were with medium effect and contributed 8.8-18.1%, 10-15.9, 15.7-18.1%, 10.2-19.4, 12.2-19.6% and 11-15.1% of the phenotypic variation explained by each QTL respectively.

Ma et al. (2007) also reported eight QTL for spike compactness in F_2 and RIL mapping population. They were located on 1A, 2D, 4A, 5A, 5B, 7D at LOD score 2.2-4.3. In the present mapping population, QTLs detected were at higher LOD and explained higher proportion of phenotypic variation than those detected by Ma et al. (2007).

Three major genes such as Q, C and S1 play key role in the gross spike morphology, and they are located on chromosomes 5A, 2D and 3D respectively (Sears 1954; Kato et al. 1999; Sourdille et al. 2000; Paillard et al. 2003; Johnson et al. 2008; Cui et al. 2012). However there is no evidence that allelic variations exist at these loci at the sub-species level of wheat (Sourdille et al. 2000). Spike length, number of spikelets per spike, number of grains per spike, grain weight per spike and spike compactness etc have been subjected to QTL analysis in many other previous reports and some previous QTLs were confirmed in the present study. Differences in spike morphology could be attributed to major and minor genes and almost all the 21 bread wheat chromosomes have been proven to harbor factor affecting spike traits of bread wheat (Sourdille et al. 2000; Kato et al. 2000; Börner et al. 2002; Li et al. 2002; Liu et al. 2006; Marza et al. 2007; Ma et al. 2007; Chu et al. 2008; Deng et al. 2010; Wang et al. 2011; Cui et al. 2012). Due to these three major genes (Q, C and S1), chromosomes 5A, 2D, and 3D should be guaranteed more attention in QTL detection for spike morphological characteristics. It is difficult to adjust relationships among SL, SNS, and SD due to their complex genetic associations, therefore SNS should be considered as prior factor in breeding programs, as the gain of grain number per spike should be ultimate goal in breeding programs. In addition,

increasing spike length without modifying the compactness might be an optimal way to improve ear fertility and grain weight per spike and thus increase the final grain yield of bread wheat. Number of grains per spike is one of the most important yield components. Raising its genetic potential is becoming more critical to increase yield potential under conditions of dense planting, since dense planting would usually result in smaller spike and less number of kernel per spike. Thus in studying the inheritance of yield related traits, much attention should be given to traits related with kernel number per spike such as spike length, spikelet number per spike and spike compactness etc. The finding in this study identified highly stable QTLs on the dense genetic map for spike related traits, and confirmed that traits are under complex genetic control and intra-genic as well as inter-genic interactions play an important role in the performance of spike related traits.

General discussion

Up to now, none of the studies have been published on the quantitative genetic architecture of the agronomic and yield related traits of bread wheat from Anatolian plateau. The present study was one of the first QTL analyses for bread wheat using a Turkish bread wheat cultivar, “Gerek”. In addition, cultivar ‘Arrehane’ used in the development of present mapping population, is widely grown in the Middle Eastern countries particularly in Morocco. Therefore this QTL analysis will be important and representative of genetic structure of bread wheat from Turkey as well as for Middle East and WANA (West Asia and North Africa) region.

Traditionally, breeding high yielding spring bread wheat cultivars has been accomplished by making direct selection for grain yield. Since yield is complex trait with low heritability, early generation selection has generally not been effective and breeders usually maintained large breeding population for a number of generations before selecting for grain yield. Dissecting grain yield into its component and selection based on the QTL coincident with yield components suggesting that effective selection and increase in at least of one of the yield component could efficiently increase the final grain product of the plant. The higher heritability was

also observed for studied yield component traits except grain weight per spike. The results of the present QTL analysis indicated that yield components could be improved through marker assisted selection with the identified QTLs. With future validation work, the identified QTLs for yield components should allow markers assisted breeding strategies to be developed and implemented in wheat breeding program for different production environments.

In the last decades many articles were published on QTL analysis in bread wheat. But most of them were related with adaptation traits such as plant height, ear emergence time, grain weight and related traits and as well as for yield. Due to this intensive work on QTL analysis for these traits, genetic basis of these are now well known and few functional markers are available for marker assisted selection for some genes controlling these characteristics. However, there are very few reports of QTLs for yield components and agronomic traits covering the whole genome of hexaploid wheat. QTLs detected on dense genetic map and shown to be associated with yield and spike related traits across multiple environments were less common in the previous published literature. This genetic map is one of the densest genetic map of bread wheat and QTLs shown here in this mapping population is important for spike morphology and spike related traits and will open a way from QTLs to candidate genes for developing functional markers for marker assisted selection in future for these characteristics.

As the number of published QTLs for various traits increases, the challenge for the plant breeders is to determine how to best utilize this knowledge to increase the efficiency of crop improvement and enhance genetic gain (Wang et al. 2009). QTLs can be detected only if the parents carry different alleles. The favorable alleles may be very specific for one of the parents and cannot be found in other genotypes. Nevertheless, the detected QTLs indicated that an improvement is possible if chromosomal regions with positive effects are combined (Börner et al. 2002). It has been argued that the QTL(s) which are detected in more than one environment or detected by more than one method for QTL analysis prove useful for marker assisted selection (Moncada et al. 2001).

The results of the present study reconfirm that the genetic control of yield components and yield related traits in bread wheat is in very complex in nature, and is controlled by a large number of major and minor QTLs. These QTLs may have main effects and/or may be involved in epistatic interaction (QQ) or environmental (QE or QQE) interactions. The magnitude and directions of the additive effects of individual QTLs may also vary due to genetic background of different genotypes and due to epistatic-by-environment interaction. However, it was intriguing to find that most of the major QTLs, which explained large proportion of the phenotypic variation and/or QTLs which were detected at high LOD score were those, which were not only consistent over environments/locations, but were also pleiotropic and/or coincident with the QTLs for other traits (co-located QTLs Fig 4.23). The selecting QTLs for early heading simultaneously results in the positive increase in spike related traits due to correlation among traits resulted from linkage and/or pleiotropic effect.

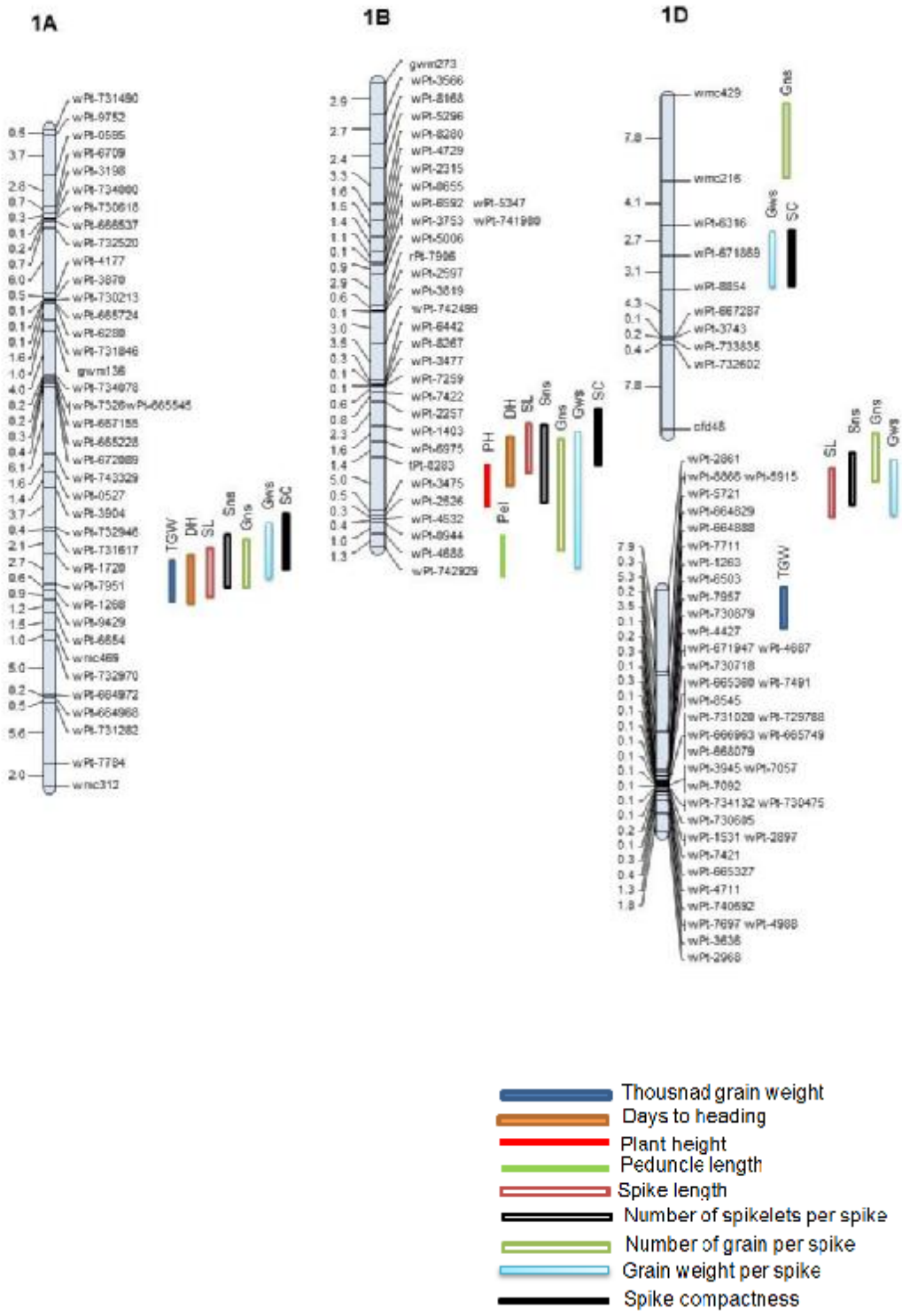

Fig. 4.23. The location of QTLs detected through simple interval mapping for different agronomic and yield related characteristics

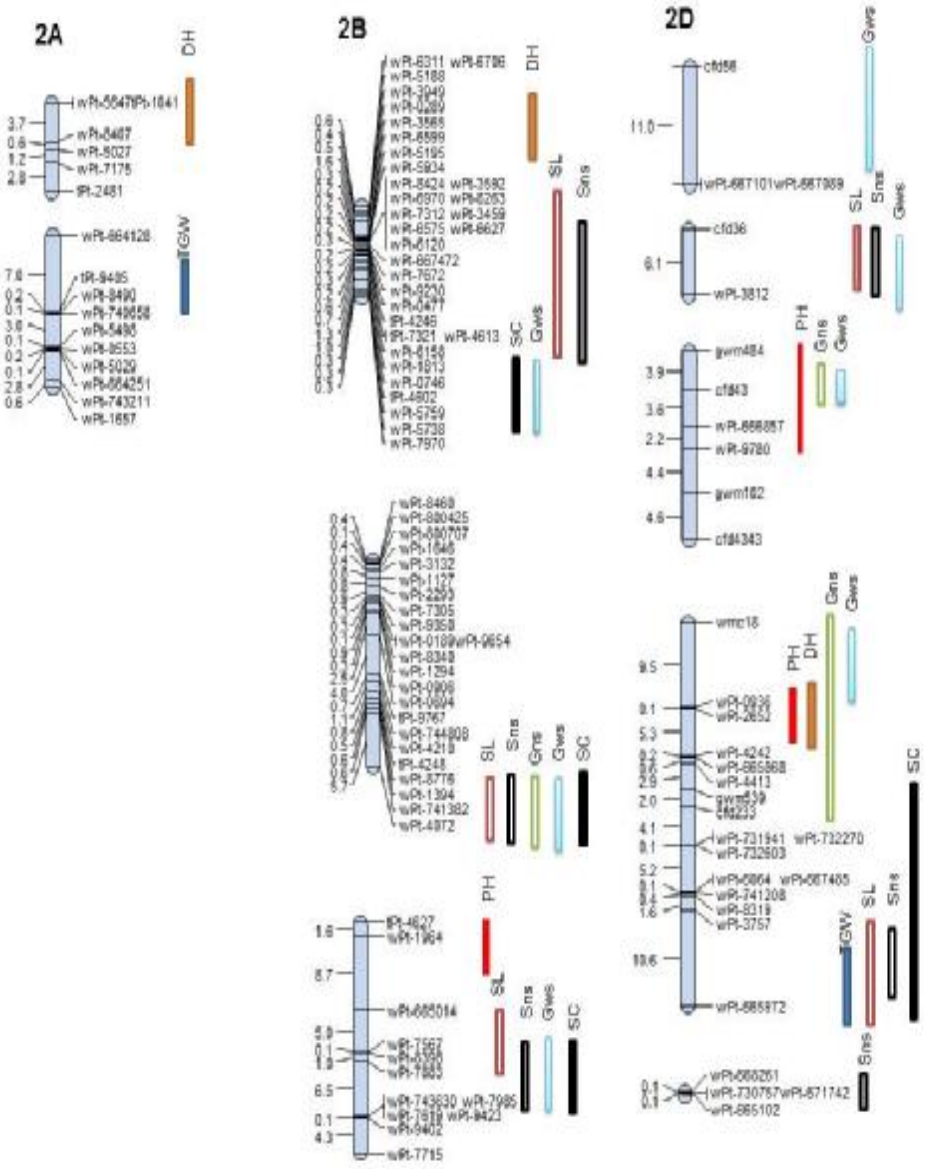

Figure 4.23. Continued

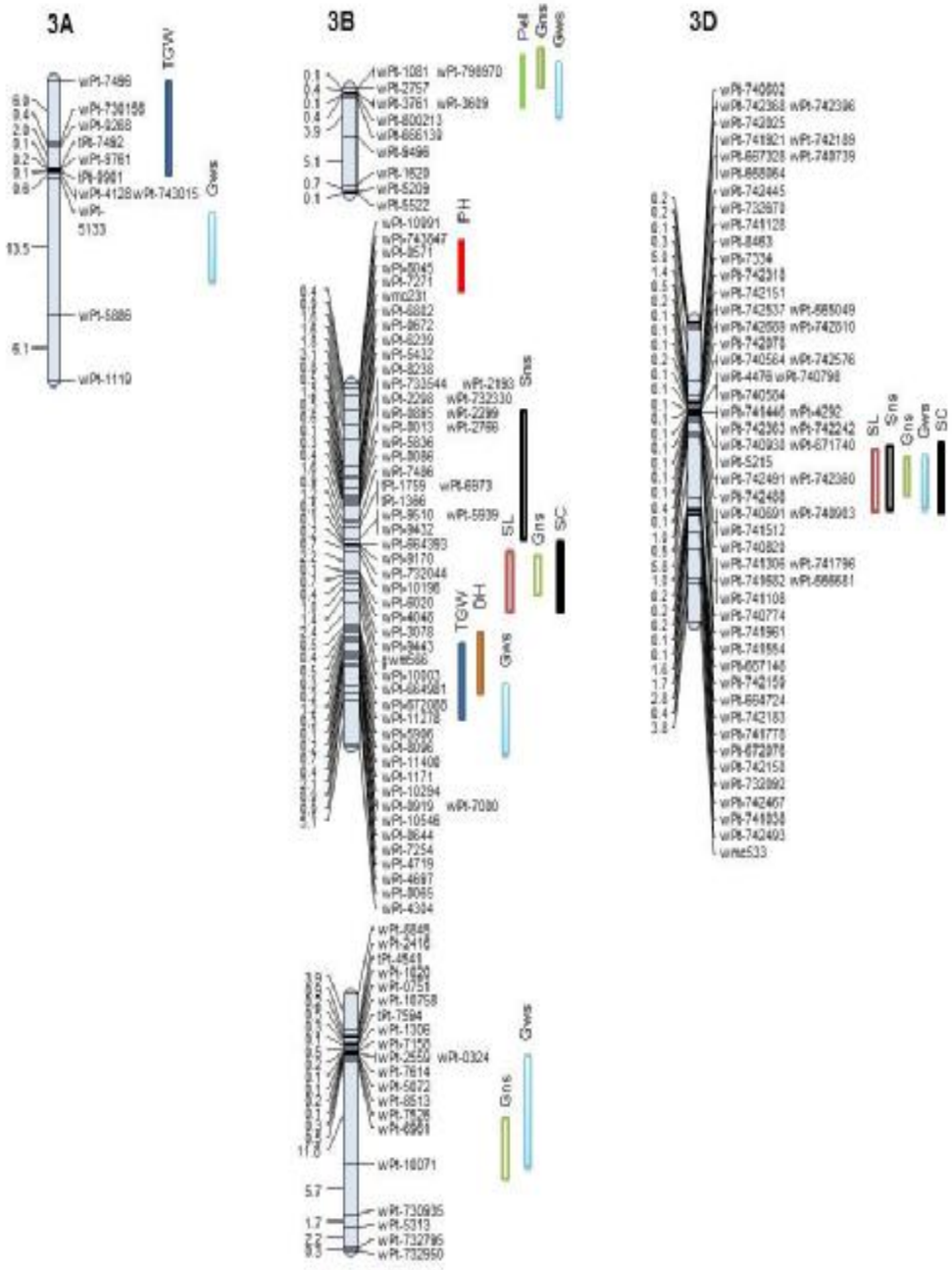

Figure 4.23. Continued

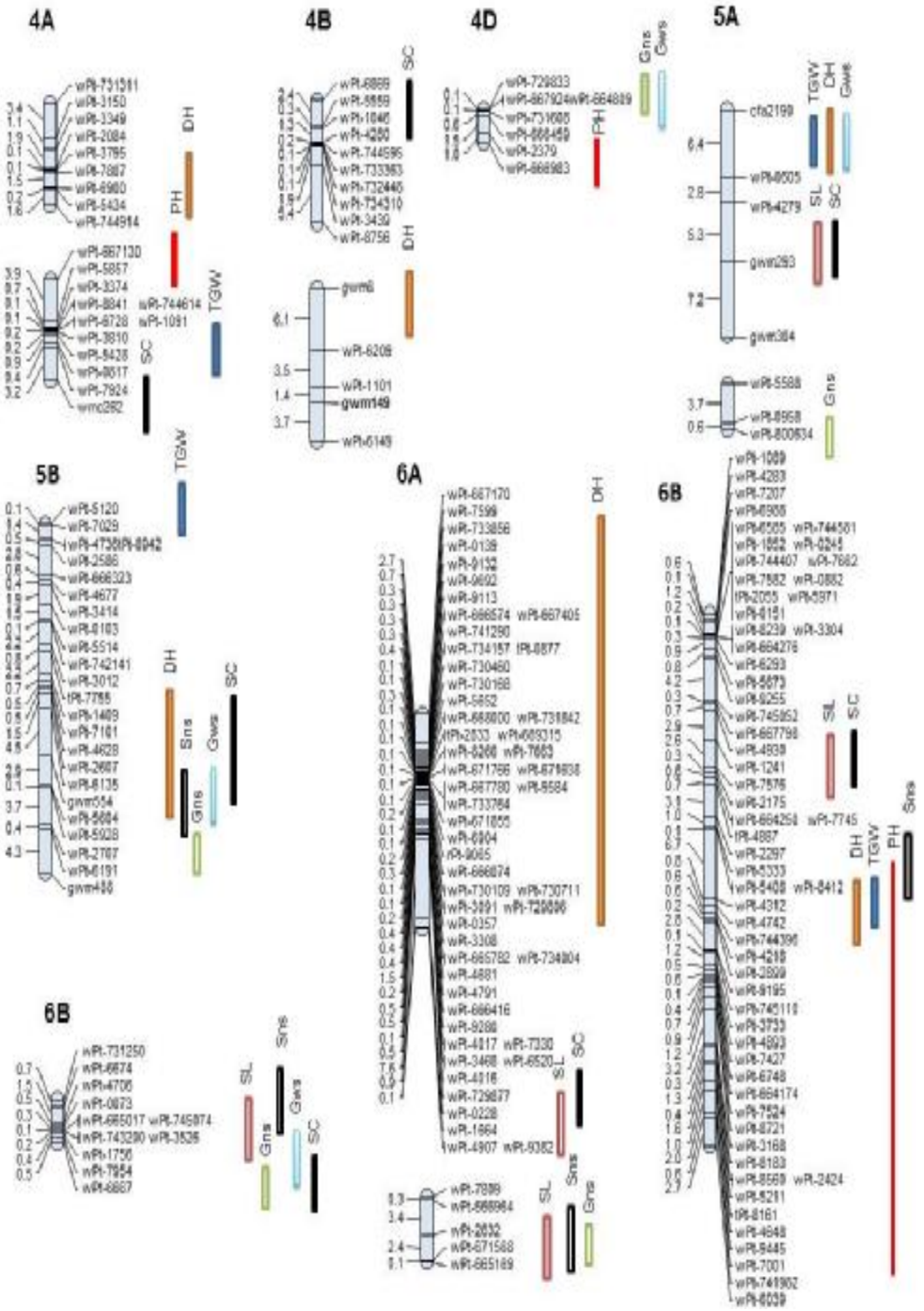

Figure 4.23. Continued

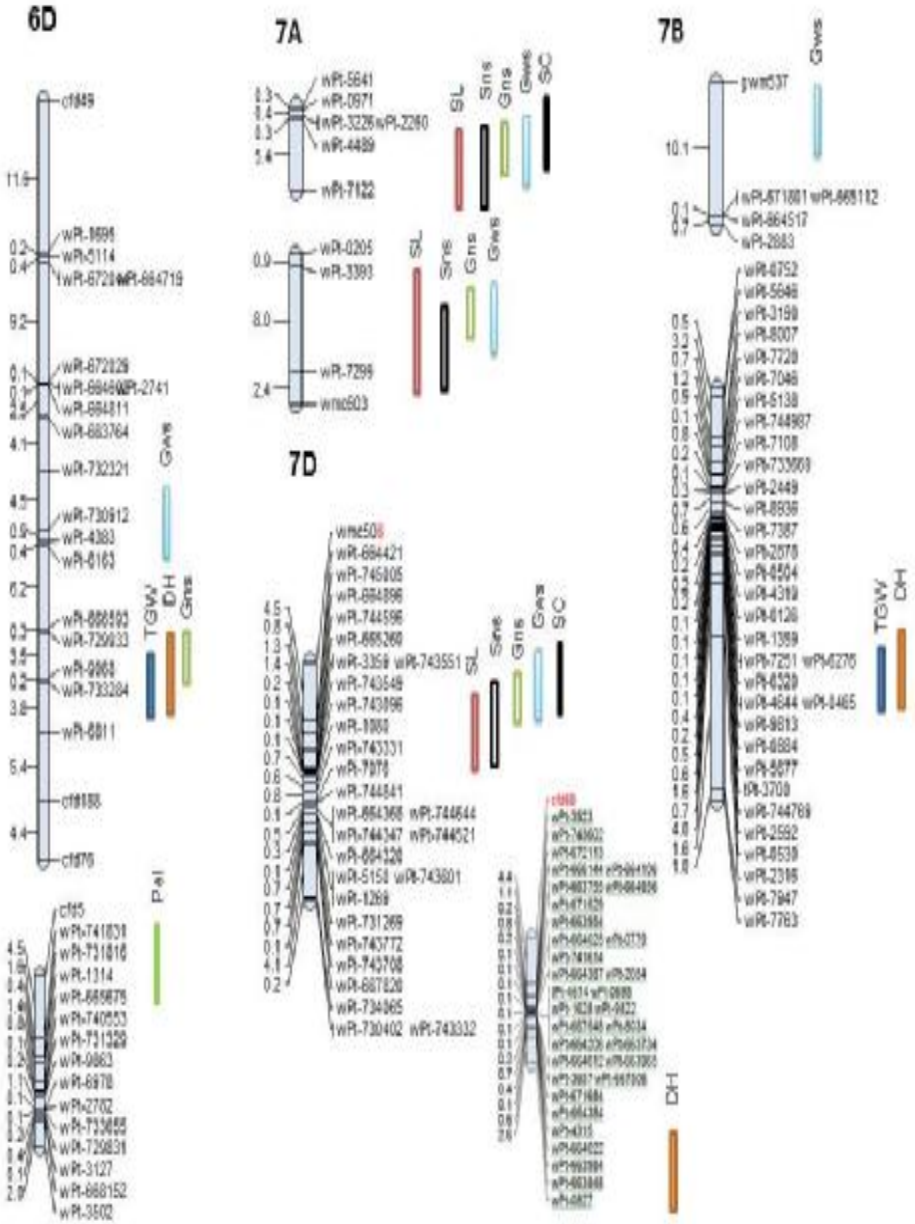

Figure 4.23. Continued

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CURRICULUM VITAE

I was born on 12th April, 1980 in Multan, Pakistan. After being taught in home, ı was send to primary school in 1985. I had completed my middle school in Islamia High school 1993 and passed the Matriculation examination in 1996 from Muhammad Ali Jinnah School, Multan. I attended the Government Science College Multan and cleared F.Sc examination in 1999. In 2004, I had completed B.Sc. (Hons) Agriculture from Bahuddin Zakariya University Multan, Pakistan. I obtained my degree of M.Sc Agronomy in 2006 from the same university. I won Cultural Exchange Scholarship for higher studies in Turkey in 2006. In 2007, I had been awarded PhD scholarship for foreigner citizens from TÜBİTAK. I started my PhD in Department of Field Crops at University of Cukurova, Adana-Turkey. I attended courses related with molecular genetics and QTL analysis at University of Copenhagen and ICARDA in 2011. I also participated in several national and international conferences. I had published several papers in international peer reviewed journals.

SUPPLEMENTARY MATERIAL

SupplementaryTablo 1. SSR markers name, their sequence and polymorphism among parents.

No	Primer Name	Sequence	Amplification in parents
1	gwm136F	TGTAAAACGACGGCCAGTGACAGCACCTTGCCCTTTG	Polymorphic
	gwm136R	CATCGGCAACATGCTCATC	
2	barc83F	TGTAAAACGACGGCCAGTAAGCAAGGAACGAGCAAGAGCAGTAG	Monomorphic
	barc83R	TGGATTTACGACGACGATGAAGATGA	
3	gwm357F	TGTAAAACGACGGCCAGTTATGGTCAAAGTTGGACCTCG	Monomorphic
	gwm357R	AGGCTGCAGCTCTTCTTCAG	
4	barc28F	TGTAAAACGACGGCCAGTCTCCCCGGCTAGTGACCACA	Monomorphic
	barc28R	GCGGCATCTTTCATTAACGAGCTAGT	
5	wmc278F	TGTAAAACGACGGCCAGTAAACGATAGTAAAATTACCTCGGAT	Monomorphic
	wmc278R	TCAAAAAATAGCAACTTGAAGACAT	
6	gwm494F	TGTAAAACGACGGCCAGTATTGAACAGGAAGACATCAGGG	Monomorphic
	gwm494R	TTCCTGGAGCTGTCTGGC	
7	wmc416F	TGTAAAACGACGGCCAGTAGCCCTTTCTACCGTGTTTCTT	Monomorphic
	wmc416R	TATGGTCGATGGACTGTCCCTA	
8	wmc134F	TGTAAAACGACGGCCAGTCCAAGCTGTCTGACTGCCATAG	Monomorphic
	wmc134R	AGTATAGACCTCTGGCTCACGG	
9	wmc44F	TGTAAAACGACGGCCAGTGGTCTTCTGGGCTTTGATCCTG	Polymorphic
	wmc44R	TGTTGCTAGGGACCCGTAGTGG	
10	wmc147F	TGTAAAACGACGGCCAGTAGAACGAAAGAAGCGCGCTGAG	Monomorphic
	wmc147R	ATGTGTTTCTTATCCTGCGGGC	
11	gwm337F	TGTAAAACGACGGCCAGTCCTCTTCCTCCCTCACTTAGC	Monomorphic
	gwm337R	TGCTAACTGGCCTTTGCC	
12	wmc429F	TGTAAAACGACGGCCAGTCGTAAAGATTTTCATTTGGCG	Polymorphic
	wmc429R	AACGGCAGCTTGAAAACATAG	
13	gwm458F	TGTAAAACGACGGCCAGTAATGGCAATTGGAAGACATAGC	No amplification
	gwm458R	TTCGCAATGTTGATTTGGC	
14	gdm126F	TGTAAAACGACGGCCAGTTCCATCATATCCGTAGCACA	Monomorphic
	gdm126R	CGTGGTTGATTTCAGGAGGT	
15	cfd63F	TGTAAAACGACGGCCAGTTCCTGAGGATGTTGAGGACC	Monomorphic
	cfd63R	GAGAGAGGCGAAACATGGAC	
16	gwm232F	TGTAAAACGACGGCCAGTATCTCAACGGCAAGCCG	Monomorphic
	gwm232R	CTGATGCAAGCAATCCACC	
17	gwm512F	TGTAAAACGACGGCCAGTAGCCACCATCAGCAAAAATT	Monomorphic
	gwm512R	GAACATGAGCAGTTTGGCAC	
18	gwm636F	TGTAAAACGACGGCCAGTCGGTAGTTTTTAGCAAAGAG	Monomorphic
	gwm636R	CCTTACAGTTCTTGGCAGAA	
19	wmc407F	TGTAAAACGACGGCCAGTGGTAATTCTAGGCTGACATATGCTC	Monomorphic
	wmc407R	CATATTTCCAAATCCCCAACTC	
20	wmc177F	TGTAAAACGACGGCCAGTAGGGCTCTCTTTAATTCTTGCT	No amplification
	wmc177R	GGTCTATCGTAATCCACCTGTA	
21	wmc522F	TGTAAAACGACGGCCAGTAAAAATCTCACGAGTCGGGC	Monomorphic
	wmc522R	CCCGAGCAGGAGCTACAAAT	
22	gwm95F	TGTAAAACGACGGCCAGTGATCAAACACACACCCCTCC	Monomorphic
	gwm95R	AATGCAAAGTGAAAAACCCG	
23	gwm558F	TGTAAAACGACGGCCAGTGGGATTGCATATGAGACAACG	No amplification
	gwm558R	TGCCATGGTTGTAGTAGCCA	
24	gwm372F	TGTAAAACGACGGCCAGTAATAGAGCCCTGGGACTGGG	Monomorphic
	gwm372R	GAAGGACGACATTCCACCTG	
25	gwm312F	TGTAAAACGACGGCCAGTATCGCATGATGCACGTAGAG	Monomorphic
	gwm312R	ACATGCATGCCTACCTAATGG	
26	wmc154F	TGTAAAACGACGGCCAGTATGCTCGTCAGTGTCATGTTTG	Monomorphic
	wmc154R	AAACGGAACCTACCTCACTCTT	
27	gwm257F	TGTAAAACGACGGCCAGTAGAGTGCATGGTGGGACG	Monomorphic
	gwm257R	CCAAGACGATGCTGAAGTCA	
28	cfa2278F	TGTAAAACGACGGCCAGTGCCTCTGCAAGTCTTTACCG	Monomorphic
	cfa2278R	AAGTCGGCCATCTTCTTCCT	
29	gwm319F	TGTAAAACGACGGCCAGTGGTTGCTGTACAAGTGTTCACG	Monomorphic
	gwm319R	CGGGTGCTGTGTGTAATGAC	
30	wmc477F	TGTAAAACGACGGCCAGTCGTCGAAAACCGTACACTCTCC	Monomorphic
	wmc477R	GCGAAACAGAATAGCCCTGATG	

Supplementary Tablo 1.Continued.

No	Primer Name	Sequence	Amplification in parents
31	wmc361F	TGTAAAACGACGGCCAGTAATGAAGATGCAAATCGACGGC	Monomorphic
	wmc361R	ATTCTCGCACTGAAAACAGGGG	
32	cfd56F	TGTAAAACGACGGCCAGTTTGCATAATTACTTGCCCTCC	Polymorphic
	cfd56R	CTGGTCCAACTTCCATCCAT	
33	cfd51F	TGTAAAACGACGGCCAGTGGAGGCTTCTCTATGGGAGG	Monomorphic
	cfd51R	TGCATCTTATCCTGTGCAGC	
34	wmc503F	TGTAAAACGACGGCCAGTGCAATAGTTCCCGCAAGAAAAG	Monomorphic
	wmc503R	ATCAACTACCTCCAGATCCCGT	
35	gwm261F	TGTAAAACGACGGCCAGTCTCCCTGTACGCCTAAGGC	Monomorphic
	gwm261R	CTCGCGCTACTAGCCATTG	
36	gwm484F	TGTAAAACGACGGCCAGTACATCGCTCTTCACAAACCC	Polymorphic
	gwm484R	AGTTCCGGTCATGGCTAGG	
37	cfd43F	TGTAAAACGACGGCCAGTAACAAAAGTCGGTGCAGTCC	Polymorphic
	cfd43R	CCAAAAACATGGTTAAAGGGG	
38	cfd116F	TGTAAAACGACGGCCAGTTTTGCCCATTACAACAAGCA	Monomorphic
	cfd116F	CAAGCAGCACCTCATGACAG	
39	gwm320F	TGTAAAACGACGGCCAGTCGAGATACTATGGAAGGTGAGG	Monomorphic
	gwm320R	ATCTTTGCAAGGATTGCCC	
40	wmc532F	TGTAAAACGACGGCCAGTGATACATCAAGATCGTGCCAAA	Monomorphic
	wmc532R	GGGAGAAATCATTAACGAAGGG	
41	gwm5F	TGTAAAACGACGGCCAGTGCCAGCTACCTCGATACAACTC	Monomorphic
	gwm5R	AGAAAGGGCCAGGCTAGTAGT	
42	gwm674F	TGTAAAACGACGGCCAGTTCGAGCGATTTTTCCTGC	Monomorphic
	gwm674R	TGACCGAGTTGACCAAAACA	
43	wmc428F	TGTAAAACGACGGCCAGTTTAATCCTAGCCGTCCCTTTTT	No amplification
	wmc428R	CGACCTTCGTTGGTTATTTGTG	
44	cfa2193F	TGTAAAACGACGGCCAGTACATGTGATGTGCGGTCATT	Monomorphic
	cfa2193R	TCCTCAGAACCCCATTCTTG	
45	gwm155F	TGTAAAACGACGGCCAGTCAATCATTTCCCCCTCCC	Monomorphic
	gwm155R	AATCATTGGAAATCCATATGCC	
46	wmc153F	TGTAAAACGACGGCCAGTATGAGGACTCGAAGCTTGGC	No amplification
	wmc153R	CTGAGCTTTTGCGCGTTGAG	
47	wmc169F	TGTAAAACGACGGCCAGTTACCCGAATCTGGAAAATCAAT	Monomorphic
	wmc169R	TGGAAGCTTGCTAACTTTGGAG	
48	gwm389F	TGTAAAACGACGGCCAGTATCATGTCGATCTCCTTGACG	Monomorphic
	gwm389R	TGCCATGCACATTAGCAGAT	
49	gwm493F	TGTAAAACGACGGCCAGTTTCCCATAACTAAAACCGCG	Monomorphic
	gwm493R	GGAACATCATTTCTGGACTTTG	
50	gwm566F	TGTAAAACGACGGCCAGTTCTGTCTACCCATGGGATTTG	Polymorphic
	gwm566R	CTGGCTTCGAGGTAAGCAAC	
51	gwm284F	TGTAAAACGACGGCCAGTAATGAAAAAACACTTGCGTGG	Monomorphic
	gwm284R	GCACATTTTTCACTTTCGGG	
52	barc73F	TGTAAAACGACGGCCAGTGCGTGTCGTGCTTGTTCTCGGTTCTCAG	No amplification
	barc73R	CGCTATTTGCCGCCACCTCCATCA	
53	gwm285F	TGTAAAACGACGGCCAGTATGACCCTTCTGCCAAACAC	Monomorphic
	gwm285R	ATCGACCGGGATCTAGCC	
54	wmc366F	TGTAAAACGACGGCCAGTTACCTCTCTACGATGAAGCC	Monomorphic
	wmc366R	TGGAGTCTTAGTGTGGTGTT	
55	gwm108F	TGTAAAACGACGGCCAGTCGACAATGGGGTCTTAGCAT	Monomorphic
	gwm108R	TGCACACTTAAATTACATCCGC	
56	gwm299F	TGTAAAACGACGGCCAGTACTACTTAGGCCTCCCGCC	Polymorphic
	gwm299R	TGACCCACTTGCAATTCATC	
57	cfd152F	TGTAAAACGACGGCCAGTTGGAAGTCTGGAACCACTCC	Polymorphic
	cfd152R	GCAACCAGACCACACTCTCA	
58	cfd55F	TGTAAAACGACGGCCAGTCCAGTAGCCGGCCCTACTAT	Monomorphic
	cfd55R	GCACGAGATACGGACAATCA	
59	gwm383F	TGTAAAACGACGGCCAGTACGCCAGTTGATCCGTAAAC	Monomorphic
	gwm383R	GACATCAATAACCGTGGATGG	
60	cfd223F	TGTAAAACGACGGCCAGTAAGAGCTACAATGACCAGCAGA	Monomorphic
	cfd223R	GCAGTGTATGTCAGGAGAAGCA	

Supplementary Tablo 1.Continued.

No	Primer Name	Sequence	Amplification in parents
61	gwm341F	TGTAAAACGACGGCCAGTTTCAGTGGTAGCGGTCGAG	No amplification
	gwm341R	CCGACATCTCATGGATCCAC	
62	wmc656F	TGTAAAACGACGGCCAGTAAGTAGGCGAGCGTTGT	No amplification
	wmc656R	TTTCCCTGGCGAGATG	
63	gwm645F	TGTAAAACGACGGCCAGTTGACCGGAAAAGGGCAGA	Monomorphic
	gwm645R	GCCCCTGCAGGAGTTTAAGT	
64	cfd9F	TGTAAAACGACGGCCAGTTTGCACGCACCTAAACTCTG	Monomorphic
	cfd9R	CAAGTGTGAGCGTCGG	
65	wmc420F	TGTAAAACGACGGCCAGTATCGTCAACAAAATCTGAAGTG	No amplification
	wmc420R	TTACTTTTGCTGAGAAAACCCT	
66	gwm601F	TGTAAAACGACGGCCAGTATCGAGGACGACATGAAGGT	Monomorphic
	gwm601R	TTAAGTTGCTGCCAATGTTCC	
67	cfa2256F	TGTAAAACGACGGCCAGTGGTAATATTCAGGTTACCGCACA	Monomorphic
	cfa2256R	GGTAAAGTTATAAATTGTTGTGGGC	
68	gwm610F	TGTAAAACGACGGCCAGTCTGCCTTCTCCATGGTTTGT	Monomorphic
	gwm610R	AATGGCCAAAGGTTATGAAGG	
69	wmc468F	TGTAAAACGACGGCCAGTAGCTGGGTTAATAACAGAGGAT	Polymorphic
	wmc468R	CACATAACTGTCCACTCCTTTC	
70	cfd257F	TGTAAAACGACGGCCAGTTCTCAACTTGCAACTGCCAC	Monomorphic
	cfd257R	CCCTCCATGGATTCTTGCTA	
71	wmc262F	TGTAAAACGACGGCCAGTGCTTTAACAAAGATCCAAGTGGCAT	Polymorphic
	wmc262R	GTAAACATCCAAACAAAGTCGAACG	
72	wmc232F	TGTAAAACGACGGCCAGTGAGATTTGTTCATTTCATCTTCGCA	No amplification
	wmc232R	TATATTAAAGGTTAGAGGTAGTCAG	
73	wmc219F	TGTAAAACGACGGCCAGTTGCTAGTTTGTCATCCGGGCGA	Monomorphic
	wmc219R	CAATCCCGTTCTACAAGTCCA	
74	gwm6F	TGTAAAACGACGGCCAGTCGTATCACCTCCTAGCTAAACTAG	Polymorphic
	gwm6R	AGCCTTATCATGACCCTACCTT	
75	wmc413F	TGTAAAACGACGGCCAGTCACTGGAAACATCTCTTCAACT	No amplification
	wmc413R	ACAGGAAAGGATGATGTTCTCT	
76	gwm251F	TGTAAAACGACGGCCAGTCAACTGGTTGCTACACAAGCA	Monomorphic
	gwm251R	GGGATGTCTGTTCCATCTTAG	
77	gwm149F	TGTAAAACGACGGCCAGTCATTGTTTTCTGCCTCTAGCC	Polymorphic
	gwm149R	CTAGCATCGAACCTGAACAAG	
78	gwm495F	TGTAAAACGACGGCCAGTGAGAGCCTCGCGAAATATAGG	No amplification
	gwm495R	TGCTTCTGGTGTTCCTTCG	
79	wmc710F	TGTAAAACGACGGCCAGTGTAAGAAGGCAGCACGTATGAA	No amplification
	wmc710R	TAAGCATTCCCAATCACTCTCA	
80	wmc285F	TGTAAAACGACGGCCAGTTGTGGTTGTATTTGCGGTATGG	Monomorphic
	wmc285R	TTGTGGTGCTGAGTTAGCTTGT	
81	wmc331F	TGTAAAACGACGGCCAGTCCTGTTGCATACTTGACCTTTTT	Monomorphic
	wmc331R	GGAGTTCAATCTTTCATCACCAT	
82	cfd84F	TGTAAAACGACGGCCAGTGTTGCCTCGGTGTCGTTTAT	Polymorphic
	cfd84R	TCCTCGAGGTCCAAAACATC	
83	gwm194F	TGTAAAACGACGGCCAGTGATCTGCTCTACTCTCCTCC	Monomorphic
	gwm194R	CGACGCAGAACTTAAACAAG	
84	cfa2250F	TGTAAAACGACGGCCAGTAGCCATAGATGGCCCTACCT	Monomorphic
	cfa2250R	CACTCAATGGCAGGTCCTTT	
85	gwm186F	TGTAAAACGACGGCCAGTGCAGAGCCTGGTTCAAAAAG	No amplification
	gwm186R	CGCCTCTAGCGAGAGCTATG	
86	cfa2155F	TGTAAAACGACGGCCAGTTTTGTTACAACCCAGGGGG	Monomorphic
	cfa2155R	TTGTGTGGCGAAAGAAACAG	
87	gwm595F	TGTAAAACGACGGCCAGTGCATAGCATCGCATATGCAT	Monomorphic
	gwm595R	GCCACGCTTGGACAAGATAT	
88	wmc524F	TGTAAAACGACGGCCAGTTAGTCCACCGGACGGAAAGTAT	Monomorphic
	wmc524R	GTACCACCGATTGATGCTTGAG	
89	gwm291F	TGTAAAACGACGGCCAGTCATCCCTACGCCACTCTGC	Monomorphic
	gwm291R	AATGGTATCTATTCCGACCCG	
90	gwm540F	TGTAAAACGACGGCCAGTTCTCGCTGTGAAATCCTATTTC	Monomorphic
	gwm540R	AGGCATGGATAGAGGGGC	

Supplementary Tablo 1.Continued.

No	Primer Name	Sequence	Amplification in parents
91	wmc73F	TGTAAAACGACGGCCAGTTTGTGCACCGCACTTACGTCTC	Monomorphic
	wmc73R	ACACCCGGTCTCCGATCCTTAG	
92	gwm371F	TGTAAAACGACGGCCAGTGACCAAGATATTCAAACTGGCC	Monomorphic
	gwm371R	AGCTCAGCTTGCTTGGTACC	
93	gwm499F	TGTAAAACGACGGCCAGTACTTGTATGCTCCATTGATTGG	Polymorphic
	gwm499R	GGGGAGTGGAAACTGCATAA	
94	wmc537F	TGTAAAACGACGGCCAGTTCTTCTGTACATTGAACAACGA	Monomorphic
	wmc537R	ATGCAGAACCGTGATAGGAT	
95	wmc75F	TGTAAAACGACGGCCAGTGTCCGCCGCACACATCTTACTA	Monomorphic
	wmc75R	GTTTGATCCTGCGACTCCTTG	
96	gwm408F	TGTAAAACGACGGCCAGTTCGATTTATTTGGGCCACTG	Polymorphic
	gwm408R	GTATAATTCGTTCACAGCACGC	
97	cfd18F	TGTAAAACGACGGCCAGTCATCCAACAGCACCAAGAGA	Monomorphic
	cfd18R	GCTACTACTATTTCATTGCGACCA	
98	cfd40F	TGTAAAACGACGGCCAGTGCGACAAGTAATTCAGAACGG	Monomorphic
	cfd40R	CGCTTCGGTAAAGTTTTTGC	
99	cfd12F	TGTAAAACGACGGCCAGTGTTACCCAAACCTGCCCTTT	Monomorphic
	cfd12R	CTACGAGTCGGGATCAGCAT	
100	cfd57F	TGTAAAACGACGGCCAGTATCGCCGTTAACATAGGCAG	Polymorphic
	cfd57R	TCACTGCTGTATTTGCTCCG	
101	gwm174F	TGTAAAACGACGGCCAGTGGGTTCCTATCTGGTAAATCCC	Monomorphic
	gwm174R	GACACACATGTTCCTGCCAC	
102	gwm292F	TGTAAAACGACGGCCAGTTCACCGTGGTCACCGAC	Monomorphic
	gwm292R	CCACCGAGCCGATAATGTAC	
103	gwm212F	TGTAAAACGACGGCCAGTAAGCAACATTTGCTGCAATG	Monomorphic
	gwm212R	TGCAGTTAACTTGTTGAAAGGA	
104	cfd29F	TGTAAAACGACGGCCAGTGGTTGTCAGGCAGGATATTTG	Polymorphic
	cfd29R	TATTGATAGATCAGGGCGCA	
105	gwm272F	TGTAAAACGACGGCCAGTTGCTCTTTGGCGAATATATGG	No amplification
	gwm272R	GTTCAAAACAAATTAAAAGGCCC	
106	wmc256F	TGTAAAACGACGGCCAGTCCAAATCTTCGAACAAGAACCC	Monomorphic
	wmc256R	ACCGATCGATGGTGTATACTGA	
107	wmc201F	TGTAAAACGACGGCCAGTCATGCTCTTTCACTTGGGTTCG	Monomorphic
	wmc201R	GCGCTTGCAGGAATTCAACACT	
108	gwm570F	TGTAAAACGACGGCCAGTTCGCCTTTTACAGTCGGC	Monomorphic
	gwm570R	ATGGGTAGCTGAGAGCCAAA	
109	wmc494F	TGTAAAACGACGGCCAGTGGATCGAGTCTCAAGTCTACAA	Monomorphic
	wmc494R	AGAAGGAACAAGCAACATCATA	
110	gwm361F	TGTAAAACGACGGCCAGTGTAACTTGTTGCCAAAGGGG	Monomorphic
	gwm361R	ACAAAGTGGCAAAAGGAGACA	
111	wmc397F	TGTAAAACGACGGCCAGTAGTCGTGCACCTCCATTTTG	Monomorphic
	wmc397R	CATTGGACATCGGAGACCTG	
112	gwm88F	TGTAAAACGACGGCCAGTCACTACAACTATGCGCTCGC	Monomorphic
	gwm88R	TCCATTGGCTTCTCTCTCAA	
113	gwm626F	TGTAAAACGACGGCCAGTGATCTAAAATGTTATTTTCTCTC	No amplification
	gwm626R	TGACTATCAGCTAAACGTGT	
114	gwm219F	TGTAAAACGACGGCCAGTGATGAGCGACACCTAGCCTC	Polymorphic
	gwm219R	GGGGTCCGAGTCCACAAC	
115	cfd75F	TGTAAAACGACGGCCAGTGCATAAACTTGGACCCTGGA	Monomorphic
	cfd75R	GCTAAGCCACGCTACCACTC	
116	gdm132F	TGTAAAACGACGGCCAGTACCGCTCGGAGAAAATCC	Polymorphic
	gdm132R	AGGGGGGCAGAGGTAGG	
117	cfd132F	TGTAAAACGACGGCCAGTCAAATGCTAATCCCCGCC	Monomorphic
	cfd132R	TGTAAACAAGGTCGCAGGTG	
118	gwm325F	TGTAAAACGACGGCCAGTTTTCTTCTGTCGTTCTCTTCCC	Monomorphic
	gwm325R	TTTTTACGCGTCAACGACG	
119	cfd188F	TGTAAAACGACGGCCAGTAATGGCTTCACTGTTTGCCT	Polymorphic
	cfd188R	AAATGGTCCCAGCATTCAAG	
120	cfd76F	TGTAAAACGACGGCCAGTGCAATTTCACACGCGACTTA	Polymorphic
	cfd76R	CGCTCGACAACATGACACTT	

Supplementary Tablo 1.Continued.

No	Primer Name	Sequence	Amplification in parents
121	cfa2049F	TGTAAAACGACGGCCAGTTAATTTGATTGGGTCGGAGC	Monomorphic
	cfa2049R	CGTGTCGATGGTCTCCTTG	
122	cfa2028F	TGTAAAACGACGGCCAGTTGGGTATGAAAGGCTGAAGG	Monomorphic
	cfa2028R	ATCGCGACTATTCAACGCTT	
123	wmc83F	TGTAAAACGACGGCCAGTTGGAGGAAACACAATGGATGCC	Monomorphic
	wmc83R	GAGTATCGCCGACGAAAGGGAA	
124	cfa2257F	TGTAAAACGACGGCCAGTGATACAATAGGTGCCTCCGC	No amplification
	cfa2257R	CCATTATGTAAATGCTTCTGTTTGA	
125	gwm332F	TGTAAAACGACGGCCAGTAGCCAGCAAGTCACCAAAAC	Monomorphic
	gwm332R	AGTGCTGGAAAGAGTAGTGAAGC	
126	gwm537F	TGTAAAACGACGGCCAGTACATAATGCTTCCTGTGCACC	Polymorphic
	gwm537R	GCCACTTTTGTGTCGTTCCT	
127	wmc426F	TGTAAAACGACGGCCAGTGACGATCGTTTCTCCTACTTTA	Monomorphic
	wmc426R	ACTACACAAATGACTGCTGCTA	
128	gwm46F	TGTAAAACGACGGCCAGTGCACGTGAATGGATTGGAC	Monomorphic
	gwm46R	TGACCCAATAGTGGTGGTCA	
129	wmc335F	TGTAAAACGACGGCCAGTTGCGGAGTAGTTCTTCCCCC	Monomorphic
	wmc335R	ACATCTTGGTGAGATGCCCT	
130	gwm297F	TGTAAAACGACGGCCAGTATCGTCACGTATTTTGCAATG	Monomorphic
	gwm297R	TGCGTAAGTCTAGCATTTTCTG	
131	wmc476F	TGTAAAACGACGGCCAGTTACCAACCACACCTGCGAGT	Polymorphic
	wmc476R	CTAGATGAACCTTCGTGCGG	
132	wmc396F	TGTAAAACGACGGCCAGTTGCACTGTTTTACCTTCACGGA	Monomorphic
	wmc396R	CAAAGCAAGAACCAGAGCCACT	
133	wmc517F	TGTAAAACGACGGCCAGTATCCTGACGTTACACGCACC	Polymorphic
	wmc517R	ACCTGGAACACCACGACAAA	
134	wmc311F	TGTAAAACGACGGCCAGTGGGCCTGCATTTCTCCTTTCTT	Monomorphic
	wmc311R	CTGAACTTGCTAGACGTTCCGA	
135	wmc276F	TGTAAAACGACGGCCAGTGACATGTGCACCAGAATAGC	No amplification
	wmc276R	AGAAGAACTATTCGACTCCT	
136	cfd66F	TGTAAAACGACGGCCAGTAGGTCTTGGTGGTTTTGGTG	Polymorphic
	cfd66R	TTTTCACATGCCCACAGTTG	
137	wmc463F	TGTAAAACGACGGCCAGTGATTGTATAGTCGGTTACCCCT	Polymorphic
	wmc463R	ATTAGTGCCCTCCATAATTGTG	
138	wmc121F	TGTAAAACGACGGCCAGTGGCTGTGGTCTCCCGATCATTC	Monomorphic
	wmc121R	ACTGGACTTGAGGAGGCTGGCA	
139	cfd14F	TGTAAAACGACGGCCAGTCCACCGGCCAGAGTAGTATT	Monomorphic
	cfd14R	TCCTGGTCTAACAACGAGAAGA	
140	wmc671F	TGTAAAACGACGGCCAGTGTACGTCAAAGAAAGAGAATTACCTC	Monomorphic
	wmc671R	CTCAGAGATATATCTTCGTTGTCAGT	
141	gwm428F	TGTAAAACGACGGCCAGTCGAGGCAGCGAGGATTT	Polymorphic
	gwm428R	TTCTCCACTAGCCCCGC	
142	cfd69F	TGTAAAACGACGGCCAGTAAATACCTTGAATTGTGAGCTGC	Polymorphic
	cfd69R	TCTGTTCATCCCCAAAGTCC	
143	gwm533F	TGTAAAACGACGGCCAGTAAGGCGAATCAAACGGAATA	Monomorphic
	gwm533R	GTTGCTTTAGGGGAAAAGCC	
144	barc133F	TGTAAAACGACGGCCAGTAGCGCTCGAAAAGTCAG	Monomorphic
	barc133R	GGCAGGTCCAACTCCAG	
145	cfa2123F	TGTAAAACGACGGCCAGTCGGTCTTTGTTTGCTCTAAACC	No amplification
	cfa2123R	ACCGGCCATCTATGATGAAG	
146	cfa2019F	TGTAAAACGACGGCCAGTGACGAGCTAACTGCAGACCC	Monomorphic
	cfa2019R	CTCAATCCTGATGCGGAGAT	
147	barc121F	TGTAAAACGACGGCCAGTACTGATCAGCAATGTCAACTGAA	Monomorphic
	barc121R	CCGGTGTCTTTCCTAACGCTATG	
148	barc71F	TGTAAAACGACGGCCAGTGCGCTTGTTCCTCACCTGCTCATA	Monomorphic
	barc71R	GCGTATATTCTCTCGTCTTCTTGTTGGTT	
149	gwm271F	TGTAAAACGACGGCCAGTCAAGATCGTGGAGCCAGC	Monomorphic
	gwm271R	AGCTGCTAGCTTTTGGGACA	
150	gwm344F	TGTAAAACGACGGCCAGTCAAGGAAATAGGCGGTAACT	Monomorphic
	gwm344R	ATTTGAGTCTGAAGTTTGCA	

Supplementary Tablo 1.Continued.

No	Primer Name	Sequence	Amplification in parents
151	wmc661F	TGTAAAACGACGGCCAGTCCACCATGGTGCTAATAGTGTC	Polymorphic
	wmc661R	AGCTCGTAACGTAATGCAACTG	
152	wmc474F	TGTAAAACGACGGCCAGTATGCTATTAAACTAGCATGTGTCG	Monomorphic
	wmc474R	AGTGGAAACATCATTCCTGGTA	
153	cfa2226F	TGTAAAACGACGGCCAGTGGAGAAAAGCAAACAGCGAC	Monomorphic
	cfa2226R	CAGTAGCATCTTCCATGGCG	
154	cfd15F	TGTAAAACGACGGCCAGTCTCCCGTATTGAGCAGGAAG	Monomorphic
	cfd15R	GGCAGGTGTGGTGATGATCT	
155	wmc336F	TGTAAAACGACGGCCAGTGTCTTACCCCGCGATCTGC	Monomorphic
	wmc336R	GCGGCCTGAGCTTCTTGAG	
156	gwm164F	TGTAAAACGACGGCCAGTACATTTCTCCCCCATCGTC	No amplification
	gwm164R	TTGTAAACAAATCGCATGCG	
157	cfd59F	TGTAAAACGACGGCCAGTTCACCTGGAAAATGGTCACA	Monomorphic
	cfd59R	AAGAAGGCTAGGGTTCAGGC	
158	gwm135F	TGTAAAACGACGGCCAGTTGTCAACATCGTTTTGAAAAGG	Monomorphic
	gwm135R	ACACTGTCAACCTGGCAATG	
159	wmc469F	TGTAAAACGACGGCCAGTAGGTGGCTGCCAACG	Polymorphic
	wmc469R	CAATTTTATCAGATGCCCGA	
160	wmc312F	TGTAAAACGACGGCCAGTTGTGCCCGCTGGTGCGAAG	Polymorphic
	wmc312R	CCGACGCAGGTGAGCGAAG	
161	cfa2129F	TGTAAAACGACGGCCAGTGTTGCACGACCTACAAAGCA	Monomorphic
	cfa2129R	ATCGCTCACTCACTATCGGG	
162	wmc59F	TGTAAAACGACGGCCAGTTCATTCGTTGCAGATACACCAC	Polymorphic
	wmc59R	TCAATGCCCTTGTTTCTGACCT	
163	cfa2219F	TGTAAAACGACGGCCAGTTCTGCCGAGTCACTTCATTG	Polymorphic
	cfa2219R	GACAAGGCCAGTCCAAAAGA	
164	gwm264F	TGTAAAACGACGGCCAGTGAGAAACATGCCGAACAACA	Monomorphic
	gwm264R	GCATGCATGAGAATAGGAACTG	
165	gwm413F	TGTAAAACGACGGCCAGTTGCTTGTCTAGATTGCTTGGG	Monomorphic
	gwm413R	GATCGTCTCGTCCTTGGCA	
166	wmc128F	TGTAAAACGACGGCCAGTCGGACAGCTACTGCTCTCCTTA	Monomorphic
	wmc128R	CTGTTGCTTGCTCTGCACCCTT	
167	gwm498F	TGTAAAACGACGGCCAGTGGTGGTATGGACTATGGACACT	No amplification
	gwm498R	TTTGCATGGAGGCACATACT	
168	wmc419F	TGTAAAACGACGGCCAGTGTTTCGGATAAAACCGGAGTGC	No amplification
	wmc419R	ACTACTTGTGGGTTATCACCAGCC	
169	gwm273F	TGTAAAACGACGGCCAGTATTGGACGGACAGATGCTTT	Polymorphic
	gwm273R	AGCAGTGAGGAAGGGGATC	
170	cfd65F	TGTAAAACGACGGCCAGTAGACGATGAGAAGGAAGCCA	Monomorphic
	cfd65R	CCTCCCTTGTTTTTGGGATT	
171	gwm11F	TGTAAAACGACGGCCAGTGGATAGTCAGACAATTCTTGTG	Monomorphic
	gwm11R	GTGAATTGTGTCTTGTATGCTTCC	
172	wmc626F	TGTAAAACGACGGCCAGTAGCCCATAAACATCCAACACGG	Monomorphic
	wmc626R	AGGTGGGCTTGGTTACGCTCTC	
173	wmc216F	TGTAAAACGACGGCCAGTACGTATCCAGACACTGTGGTAA	Polymorphic
	wmc216R	TAATGGTGGATCCATGATAGCC	
174	barc181F	TGTAAAACGACGGCCAGTCGCTGGAGGGGGTAAGTCATCAC	Monomorphic
	barc181F	CGCAAATCAAGAACACGGGAGAAAGAA	
175	cfd48F	TGTAAAACGACGGCCAGTATGGTTGATGGTGGGTGTTT	Polymorphic
	cfd48R	ATGTATCGATGAAGGGCCAA	
176	gwm274F	TGTAAAACGACGGCCAGTAACTTGCAAAACTGTTCTGA	No amplification
	gwm274R	TATTTGAAGCGGTTTGATTT	
177	gwm124F	TGTAAAACGACGGCCAGTGCCATGGCTATCACCCAG	Monomorphic
	gwm124R	ACTGTTCGGTGCAATTTGAG	
178	wmc367F	TGTAAAACGACGGCCAGTCTGACGTTGATGGGCCACTATT	Monomorphic
	wmc367R	GTGGTGGAAGAGGAAGGAGAGG	
179	cfd61F	TGTAAAACGACGGCCAGTATTCAAATGCAACGCAAACA	No amplification
	cfd61R	GTTAGCCAAGGACCCCTTTC	
180	cfd21F	TGTAAAACGACGGCCAGTCCTCCATGTAGGCGGAAATA	Monomorphic
	cfd21R	TGTGTCCCATTCACTAACCG	

Supplementary Tablo 1.Continued.

No	Primer Name	Sequence	Amplification in parents
181	cfd92F	TGTAAAACGACGGCCAGTCTTGTTGATCTCCTTCCCCA	Polymorphic
	cfd92R	TTCTCTCATGACGGCAACAC	
182	cfd72F	TGTAAAACGACGGCCAGTCTCCTTGGAATCTCACCGAA	Monomorphic
	cfd72R	TCCTTGGGAATATGCCTCCT	
183	wmc339F	TGTAAAACGACGGCCAGTCCGCTCGCCTTCTTCCAG	Monomorphic
	wmc339R	TCCGGAACATGCCGATAC	
184	gwm642F	TGTAAAACGACGGCCAGTACGGCGAGAAGGTGCTC	Monomorphic
	gwm642R	CATGAAAGGCAAGTTCGTCA	
185	cfa2147F	TGTAAAACGACGGCCAGTTCATCCCCTACATAACCCGA	Monomorphic
	cfa2147R	ATCGTGCACCAAGCAATACA	
186	wmc667F	TGTAAAACGACGGCCAGTGAGGAGAGGAAAAGGCAGGCTA	Monomorphic
	wmc667R	AACTCTTGCGTGTCTCAAACCG	
187	wmc382F	TGTAAAACGACGGCCAGTCATGAATGGAGGCACTGAAACA	Monomorphic
	wmc382R	CCTTCCGGTCGACGCAAC	
188	wmc296F	TGTAAAACGACGGCCAGTGAATCTCATCTTCCCTTGCCAC	Monomorphic
	wmc296R	ATGGAGGGGTATAAAGACAGCG	
189	gwm294F	TGTAAAACGACGGCCAGTGGATTGGAGTTAAGAGAGAACCG	Monomorphic
	gwm294R	GCAGAGTGATCAATGCCAGA	
190	cfd168F	TGTAAAACGACGGCCAGTCTTCGCAAATCGAGGATGAT	Monomorphic
	cfd168R	TTCACGCCCAGTATTAAGGC	
191	wmc181F	TGTAAAACGACGGCCAGTTCCTTGACCCCTTGCACTAACT	No amplification
	wmc181R	ATGGTTGGGAGCACTAGCTTGG	
192	gwm356F	TGTAAAACGACGGCCAGTAGCGTTCTTGGGAATTAGAGA	Monomorphic
	gwm356R	CCAATCAGCCTGCAACAAC	
193	wmc658F	TGTAAAACGACGGCCAGTCTCATCGTCCTCCTCCACTTTG	Monomorphic
	wmc658R	GCCATCCGTTGACTTGAGGTTA	
194	wmc25F	TGTAAAACGACGGCCAGTTCTGGCCAGGATCAATATTACT	No amplification
	wmc25R	TAAGATACATAGATCCAACACC	
195	gwm148F	TGTAAAACGACGGCCAGTGTGAGGCAGCAAGAGAGAAA	Monomorphic
	gwm148R	CAAAGCTTGACTCAGACCAAA	
196	gwm630F	TGTAAAACGACGGCCAGTGTGCCTGTGCCATCGTC	Monomorphic
	gwm630R	CGAAAGTAACAGCGCAGTGA	
197	gwm388F	TGTAAAACGACGGCCAGTCTACAATTCGAAGGAGAGGGG	Monomorphic
	gwm388R	CACCGCGTCAACTACTTAAGC	
198	cfd73F	TGTAAAACGACGGCCAGTGATAGATCAATGTGGGCCGT	Monomorphic
	cfd73R	AACTGTTCTGCCATCTGAGC	
199	wmc332F	TGTAAAACGACGGCCAGTCATTTACAAAGCGCATGAAGCC	No amplification
	wmc332R	GAAAACTTTGGGAACAAGAGCA	
200	wmc149F	TGTAAAACGACGGCCAGTACAGACTTGGTTGGTGCCGAGC	Monomorphic
	wmc149R	ATGGGCGGGGGTGTAGAGTTTG	
201	cfd36F	TGTAAAACGACGGCCAGTGCAAAGTGTAGCCGAGGAAG	Polymorphic
	cfd36R	TTAGAGTTTTGCAGCGCCTT	
202	wmc112F	TGTAAAACGACGGCCAGTTGAGTTGTGGGGTCTTGTTTGG	Monomorphic
	wmc112R	TGAAGGAGGGCACATATCGTTG	
203	cfd43F	TGTAAAACGACGGCCAGTAACAAAAGTCGGTGCAGTCC	Polymorphic
	cfd43R	CCAAAAACATGGTTAAAGGGG	
204	gwm102F	TGTAAAACGACGGCCAGTTCTCCCATCCAACGCCTC	Polymorphic
	gwm102R	TGTTGGTGGCTTGACTATTG	
205	cfd2F	TGTAAAACGACGGCCAGTGGTTGCAGTTTCCACCTTGT	Monomorphic
	cfd2R	CATCTATTGCCAAAATCGCA	
206	wmc18F	TGTAAAACGACGGCCAGTCTGGGGCTTGGATCACGTCATT	Polymorphic
	wmc18R	AGCCATGGACATGGTGTCCTTC	
207	wmc144F	TGTAAAACGACGGCCAGTGGACACCAATCCAACATGAACA	Monomorphic
	wmc144R	AAGGATAGTTGGGTGGTGCTGA	
208	wmc601F	TGTAAAACGACGGCCAGTACAGAGGCATATGCAAAGGAGG	Monomorphic
	wmc601R	CTTGTCTCTTTATCGAGGGTGG	
209	gwm157F	TGTAAAACGACGGCCAGTGTCGTCGCGGTAAGCTTG	Monomorphic
	gwm157R	GAGTGAACACACGAGGCTTG	
210	cfd233F	TGTAAAACGACGGCCAGTGAATTTTTGGTGGCCTGTGT	Polymorphic
	cfd233R	ATCACTGCACCGACTTTTGG	

Supplementary Tablo 1.Continued.

No	Primer Name	Sequence	Amplification in parents
211	gwm539F	TGTAAAACGACGGCCAGTCTGCTCTAAGATTCATGCAACC	Polymorphic
	gwm539R	GAGGCTTGTGCCCTCTGTAG	
212	wmc167F	TGTAAAACGACGGCCAGTAGTGGTAATGAGGTGAAAGAAG	Monomorphic
	wmc167R	TCGGTCGTATATGCATGTAAAG	
213	gwm301F	TGTAAAACGACGGCCAGTGAGGAGTAAGACACATGCCC	Monomorphic
	gwm301R	GTGGCTGGAGATTCAGGTTC	
214	wmc11F	TGTAAAACGACGGCCAGTTTGTGATCCTGGTTGTGTTGTGA	Monomorphic
	wmc11R	CACCCAGCCGTTATATATGTTGA	
215	gwm369F	TGTAAAACGACGGCCAGTCTGCAGGCCATGATGATG	Monomorphic
	gwm369R	ACCGTGGGTGTTGTGAGC	
216	wmc264F	TGTAAAACGACGGCCAGTCTCCATCTATTGAGCGAAGGTT	Polymorphic
	wmc264R	CAAGATGAAGCTCATGCAAGTG	
217	cfa2262F	TGTAAAACGACGGCCAGTACAATGTGGAGATGGCACAA	Monomorphic
	cfa2262R	TACCAGCTGCACTTCCATTG	
218	cfa2076F	TGTAAAACGACGGCCAGTCGAAAAACCATGATCGACAG	Monomorphic
	cfa2076R	ACCTGTCCAGCTAGCCTCCA	
219	wmc231F	TGTAAAACGACGGCCAGTCATGGCGAGGAGCTCGGTGGTC	Polymorphic
	wmc231R	GTGGAGCACAGGCGGAGCAAGG	
220	cfd6F	TGTAAAACGACGGCCAGTACTCTCCCCCTCGTTGCTAT	Monomorphic
	cfd6R	ATTTAAGGGAGACATCGGGC	
221	wmc625F	TGTAAAACGACGGCCAGTCACAGACCTCAACCTCTTCTT	Monomorphic
	wmc625R	AGTACTGTTCACAGCAGACGA	
222	wmc307F	TGTAAAACGACGGCCAGTGTTTGAAGACCAAGCTCCTCCT	Monomorphic
	wmc307R	ACCATAACCTCTCAAGAACCCA	
223	wmc471F	TGTAAAACGACGGCCAGTGGCAATAATAGTGCAAGGAATG	Monomorphic
	wmc471R	GCCGATAATGGGCAATATAAGT	
224	wmc418F	TGTAAAACGACGGCCAGTAGAGCAGCAAGTTGTGTAGCCA	Polymorphic
	wmc418R	TGAAGCTATTGCCAGCACGAG	
225	wmc632F	TGTAAAACGACGGCCAGTGTTTGATTGGTCGTTCCTGGTC	Monomorphic
	wmc632R	AACAGCGAATGGAGGGCTTTAG	
226	gwm340F	TGTAAAACGACGGCCAGTGCAATCTTTTTTCTGACCACG	Monomorphic
	gwm340R	ACGAGGCAAGAACACACATG	
227	cfd35F	TGTAAAACGACGGCCAGTGGGATGACACATAACGGACA	No amplification
	cfd35R	ATCAGCGGCGCTATAGTACG	
228	gwm161F	TGTAAAACGACGGCCAGTGATCGAGTGATGGCAGATGG	Monomorphic
	gwm161R	TGTGAATTACTTGGACGTGG	
229	wmc43F	TGTAAAACGACGGCCAGTTAGCTCAACCACCACCCTACTG	Monomorphic
	wmc43R	ACTTCAACATCCAAACTGACCG	
230	wmc492F	TGTAAAACGACGGCCAGTAGGATCAGAATAGTGCTACCC	Monomorphic
	wmc492R	ATCCCGTGATCAGAATAGTGT	
231	gwm456F	TGTAAAACGACGGCCAGTTCTGAACATTACACAACCCTGA	Monomorphic
	gwm456R	TGCTCTCTCTGAACCTGAAGC	
232	wmc533F	TGTAAAACGACGGCCAGTAATTGGATCGGCAGTTGGAG	Polymorphic
	wmc533R	AGCAAGCAGAGCATTGCGTT	
233	wmc631F	TGTAAAACGACGGCCAGTTTGCTCGCCCACCTTCTACC	Polymorphic
	wmc631R	GGAAACCATGCGCTTCACAC	
234	wmc491F	TGTAAAACGACGGCCAGTGGTAAAACTTCGTGTCCCTTGC	Polymorphic
	wmc491R	TAGTTGCGAGTCGGTAGTCTGC	
235	cfd71F	TGTAAAACGACGGCCAGTCAATAAGTAGGCCGGGACAA	Polymorphic
	cfd71R	TGTGCCAGTTGAGTTTGCTC	
236	wmc96F	TGTAAAACGACGGCCAGTTAGCAGCCATGCTTAGCATCAA	Monomorphic
	wmc96R	GTTTCAGTCTTTCACGAACACG	
237	wmc707F	TGTAAAACGACGGCCAGTGCTAGCTGACACTTTTCCTTTG	Monomorphic
	wmc707R	TCAGTTTCCCACTCACTTCTTT	
238	gwm494F	TGTAAAACGACGGCCAGTATTGAACAGGAAGACATCAGGG	Monomorphic
	gwm494R	TTCCTGGAGCTGTCTGGC	
239	wmc283F	TGTAAAACGACGGCCAGTCGTTGGCTGGGTTATATCATCT	Polymorphic
	wmc283R	GACCCGCGTGTAAGTGATAGGA	
240	gwm160F	TGTAAAACGACGGCCAGTTTCAATTCAGTCTTGGCTTGG	No amplification
	gwm160R	CTGCAGGAAAAAAAGTACACCC	

Supplementary Tablo 1.Continued.

No	Primer Name	Sequence	Amplification in parents
241	wmc313F	TGTAAAACGACGGCCAGTGCAGTCTAATTATCTGCTGGCG	Monomorphic
	wmc313R	GGGTCCTTGTCTACTCATGTCT	
242	wmc125F	TGTAAAACGACGGCCAGTATACCACCATGCATGTGGAAGT	Monomorphic
	wmc125R	ACCGCTTGTCATTTCCTTCTGT	
243	wmc47F	TGTAAAACGACGGCCAGTGAAACAGGGTTAACCATGCCAA	Monomorphic
	wmc47R	ATGGTGCTGCCAACAACATACA	
244	wmc349F	TGTAAAACGACGGCCAGTACACACACTCGATCGCAC	Monomorphic
	wmc349R	GCAGTTGATCATCAAAACACA	
245	gwm192F	TGTAAAACGACGGCCAGTGGTTTTCTTTCAGATTGCGC	No amplification
	gwm192R	CGTTGTCTAATCTTGCCTTGC	
246	wmc238F	TGTAAAACGACGGCCAGTTCTTCCTGCTTACCCAAACACA	Monomorphic
	wmc238R	TACTGGGGGATCGTGGATGACA	
247	wmc89F	TGTAAAACGACGGCCAGTATGTCCACGTGCTAGGGAGGTA	No amplification
	wmc89R	TTGCCTCCCAAGACGAAATAAC	
248	gwm368F	TGTAAAACGACGGCCAGTCCATTTCACCTAATGCCTGC	Monomorphic
	gwm368R	AATAAAACCATGAGCTCACTTGC	
249	wmc48F	TGTAAAACGACGGCCAGTGAGGGTTCTGAAATGTTTTGCC	Monomorphic
	wmc48R	ACGTGCTAGGGAGGTATCTTGC	
250	wmc457F	TGTAAAACGACGGCCAGTCTTCCATGAATCAAAGCAGCAC	Monomorphic
	wmc457R	CATCCATGGCAGAAACAATAGC	
251	wmc622F	TGTAAAACGACGGCCAGTCAGGAAGAAGAGCTCCGAGAAA	Monomorphic
	wmc622R	CTTGCTAACCCGCGCC	
252	gwm443F	TGTAAAACGACGGCCAGTGGGTCTTCATCCGGAACTCT	Monomorphic
	gwm443R	CCATGATTTATAAATTCCACC	
253	cfa2104F	TGTAAAACGACGGCCAGTCCTGGCAGAGAAAGTGAAGG	Monomorphic
	cfa2104R	AGTCGCCGTTGTATAGTGCC	
254	cfa2190F	TGTAAAACGACGGCCAGTCAGTCTGCAATCCACTTTGC	Polymorphic
	cfa2190R	AAAAGGAAACTAAAGCGATGGA	
255	gwm293F	TGTAAAACGACGGCCAGTTACTGGTTCACATTGGTGCG	Polymorphic
	gwm293R	TCGCCATCACTCGTTCAAG	
256	gwm304F	TGTAAAACGACGGCCAGTAGGAAACAGAAATATCGCGG	Polymorphic
	gwm304R	AGGACTGTGGGGAATGAATG	
257	gwm156F	TGTAAAACGACGGCCAGTCCAACCGTGCTATTAGTCATTC	Polymorphic
	gwm156R	CAATGCAGGCCCTCCTAAC	
258	gwm617F	TGTAAAACGACGGCCAGTGATCTTGGCGCTGAGAGAGA	Monomorphic
	gwm617R	CTCCGATGGATTACTCGCAC	
259	wmc415F	TGTAAAACGACGGCCAGTAATTCGATACCTCTCACTCACG	No amplification
	wmc415R	TCAACTGCTACAACCTAGACCC	
260	wmc445F	TGTAAAACGACGGCCAGTAGAATAGGTTCTTGGGCCAGTC	Monomorphic
	wmc445R	GAGATGATCTCCTCCATCAGCA	
261	cfa2163F	TGTAAAACGACGGCCAGTTTGATCCTTGATGGGAGGAG	Monomorphic
	cfa2163R	CATCATTGTGTTTACGTTCTTTCA	
262	cfa2141F	TGTAAAACGACGGCCAGTGAATGGAAGGCGGACATAGA	Monomorphic
	cfa2141R	GCCTCCACAACAGCCATAAT	
263	barc232F	TGTAAAACGACGGCCAGTCGCATCCAACCATCCCCACCCAACA	No amplification
	barc232R	CGCAGTAGATCCACCACCCCGCCAGA	
264	cfa2185F	TGTAAAACGACGGCCAGTTTCTTCAGTTGTTTTGGGGG	No amplification
	cfa2185R	TTTGGTCGACAAGCAAATCA	
265	wmc110F	TGTAAAACGACGGCCAGTGCAGATGAGTTGAGTTGGATTG	Monomorphic
	wmc110R	GTACTTGGAAACTGTGTTTGGG	
266	gwm126F	TGTAAAACGACGGCCAGTCACACGCTCCACCATGAC	Monomorphic
	gwm126R	GTTGAGTTGATGCGGGAGG	
267	cfd5F	TGTAAAACGACGGCCAGTTGCCCTGTCCACAGTGAAG	Polymorphic
	cfd5R	TTGCCAGTTCCAAGGAGAAT	
268	cfd60F	TGTAAAACGACGGCCAGTTGACCGGCATTCAGTATCAA	Monomorphic
	cfd60R	TGGTCACTTTGATGAGCAGG	
269	cfd20F	TGTAAAACGACGGCCAGTTGATGGGAAGGTAATGGGAG	Monomorphic
	cfd20R	ATCCAGTTCTCGTCCAAAGC	
270	wmc376F	TGTAAAACGACGGCCAGTTCTCAACCACCGACTTGTAA	No amplification
	wmc376R	ACATGTAATTGGGGACACTG	

Supplementary Tablo 1.Continued.

No	Primer Name	Sequence	Amplification in parents
271	gwm335F	TGTAAAACGACGGCCAGTCGTACTCCACTCCACACGG	Polymorphic
	gwm335R	CGGTCCAAGTGCTACCTTTC	
272	gwm554F	TGTAAAACGACGGCCAGTTGCCCACAACGGAACTTG	Polymorphic
	gwm554R	GCAACCACCAAGCACAAAGT	
273	cfd7F	TGTAAAACGACGGCCAGTAGCTACCAGCCTAGCAGCAG	Monomorphic
	cfd7R	TCAGACACGTCTCCTGACAAA	
274	wmc289F	TGTAAAACGACGGCCAGTCATATGCATGCTATGCTGGCTA	Monomorphic
	wmc289R	AGCCTTTCAAATCCATCCACTG	
275	wmc160F	TGTAAAACGACGGCCAGTCATGGCTCCAAGATACAAAAAG	No amplification
	wmc160R	AGGCCTGGATTCATGATAGATA	
276	cfd86F	TGTAAAACGACGGCCAGTTTAATGAGCGTCAGTACTCCC	No amplification
	cfd86R	GCAACCATGTTTAAGCCGAT	
277	wmc508F	TGTAAAACGACGGCCAGTAGCCCTTGAGTTGGTCTCATTT	No amplification
	wmc508R	GAGCAGAGCTCCACTCACATTT	
278	gwm497F	TGTAAAACGACGGCCAGTGTAGTGAAGACAAGGGCATT	No amplification
	gwm497R	CCGAAAGTTGGGTGATATAC	
279	wmc233F	TGTAAAACGACGGCCAGTGACGTCAAGAATCTTCGTCGGA	Monomorphic
	wmc233R	ATCTGCTGAGCAGATCGTGGTT	
280	gwm190F	TGTAAAACGACGGCCAGTGTGCTTGCTGAGCTATGAGTC	Polymorphic
	gwm190R	GTGCCACGTGGTACCTTTG	
281	cfd189F	TGTAAAACGACGGCCAGTGCTAAAGCCACATAGGACGG	Monomorphic
	cfd189R	GCACAAGATTTTGCAAGGCT	
282	cfd8F	TGTAAAACGACGGCCAGTACCACCGTCATGTCACTGAG	Monomorphic
	cfd8R	GTGAAGACGACAAGACGCAA	
283	gwm182F	TGTAAAACGACGGCCAGTTGATGTAGTGAGCCCATAGGC	Monomorphic
	gwm182R	TTGCACACAGCCAAATAAGG	
284	cfd102F	TGTAAAACGACGGCCAGTTTGTGGAAGGGTTTGATGAAG	Monomorphic
	cfd102R	TGCAGGACCAAACATAGCTG	
285	cfd183F	TGTAAAACGACGGCCAGTACTTGCACTTGCTATACTTACGAA	Monomorphic
	cfd183R	GTGTGTCGGTGTGTGGAAAG	
286	wmc357F	TGTAAAACGACGGCCAGTTAGTGGGTGACCGGTCAAGA	Monomorphic
	wmc357R	TGGACGGATTTGGTCATTTC	
287	cfd10F	TGTAAAACGACGGCCAGTCGTTCTATGACGTGTCATGCT	Monomorphic
	cfd10R	TCCATTTTCAAAAACACCCTG	
288	gwm334F	TGTAAAACGACGGCCAGTAATTTCAAAAAGGAGAGAGA	No amplification
	gwm334R	AACATGTGTTTTTAGCTATC	
289	wmc487F	TGTAAAACGACGGCCAGTCAAATTTGGCCACCATTTTACA	No amplification
	wmc487R	CGGTTCAATCCTTGGATTTACA	
290	gwm705F	TGTAAAACGACGGCCAGTTCTCCCTCATTAGAGTTGTCCA	Monomorphic
	gwm705R	ATGCAAGTTTAGAGCAACACCA	
291	cfd13F	TGTAAAACGACGGCCAGTCCACTAACCAAGCTGCCATT	Monomorphic
	cfd13R	TTTTTGGCATTGATCTGCTG	
292	gwm518F	TGTAAAACGACGGCCAGTAATCACAACAAGGCGTGACA	No amplification
	gwm518R	CAGGGTGGTGCATGCAT	
293	gwm193F	TGTAAAACGACGGCCAGTCTTTGTGCACCTCTCTCTCC	Monomorphic
	gwm193R	AATTGTGTTGATGATTTGGGG	
294	gwm311F	TGTAAAACGACGGCCAGTTCACGTGGAAGACGCTCC	Monomorphic
	gwm311R	CTACGTGCACCACCATTTTG	
295	cfd49F	TGTAAAACGACGGCCAGTTGAGTTCTTCTGGTGAGGCA	Polymorphic
	cfd49R	GAATCGGTTCACAAGGGAAA	
296	barc54F	TGTAAAACGACGGCCAGTGCGAACAGGAGGACAGAGGGCACGAGAG	Polymorphic
	barc54R	GCGCTTTCCCACGTTCCATGTTTCT	
297	cfd190F	TGTAAAACGACGGCCAGTCAATCAGAAGCGCCATTGTT	Monomorphic
	cfd190R	CCCTGATGTTTTCTTTTTCTCC	
298	gwm666F	TGTAAAACGACGGCCAGTGCACCCACATCTTCGACC	Monomorphic
	gwm666R	TGCTGCTGGTCTCTGTGC	
299	gwm635F	TGTAAAACGACGGCCAGTTTCCTCACTGTAAGGGCGTT	Monomorphic
	gwm635R	CAGCCTTAGCCTTGGCG	
300	gwm471F	TGTAAAACGACGGCCAGTCGGCCCTATCATGGCTG	No amplification
	gwm471R	GCTTGCAAGTTCCATTTTGC	

Supplementary Tablo 1.Continued.

No	Primer Name	Sequence	Amplification in parents
301	wmc593F	TGTAAAACGACGGCCAGTGGGGAGAAGCAGCAGGG	Polymorphic
	wmc593R	CGCGCGGTTGCCGGTGG	
302	wmc603F	TGTAAAACGACGGCCAGTACAAACGGTGACAATGCAAGGA	Polymorphic
	wmc603R	CGCCTCTCTCGTAAGCCTCAAC	
303	gwm400F	TGTAAAACGACGGCCAGTGTGCTGCCACCACTTGC	Monomorphic
	gwm400R	TGTAGGCACTGCTTGGGAG	
304	wmc475F	TGTAAAACGACGGCCAGTAACACATTTTCTGTCTTTCGCC	No amplification
	wmc475R	TGTAGTTATGCCCAACCTTTCC	
305	gwm333F	TGTAAAACGACGGCCAGTGCCCGGTCATGTAAAACG	Monomorphic
	gwm333R	TTTCAGTTTGCGTTAAGCTTTG	
306	wmc76F	TGTAAAACGACGGCCAGTCTTCAGAGCCTCTTTCTCTACA	Monomorphic
	wmc76R	CTGCTTCACTTGCTGATCTTTG	
307	cfa2040F	TGTAAAACGACGGCCAGTTCAAATGATTTCAGGTAACCACTA	Monomorphic
	cfa2040R	TTCCTGATCCCACCAAACAT	
308	wmc10F	TGTAAAACGACGGCCAGTGATCCGTTCTGAGGTGAGTT	Monomorphic
	wmc10R	GGCAGCACCCTCTATTGTCT	
309	gwm146F	TGTAAAACGACGGCCAGTCCAAAAAAACTGCCTGCATG	Monomorphic
	gwm146R	CTCTGGCATTGCTCCTTGG	
310	wmc506F	TGTAAAACGACGGCCAGTCACTTCCTCAACATGCCAGA	Polymorphic
	wmc506R	CTTTCAATGTGGAAGGCGAC	
311	cfd31F	TGTAAAACGACGGCCAGTGCACCAACCTTGATAGGGAA	Monomorphic
	cfd31R	GTGCCTGATGATTTTACCCG	
312	wmc606F	TGTAAAACGACGGCCAGTCCGATGAACAGACTCGACAAGG	Monomorphic
	wmc606R	GGCTTCGGCCAGTAGTACAGGA	
313	gwm44F	TGTAAAACGACGGCCAGTGTTGAGCTTTTCAGTTCGGC	Monomorphic
	gwm44R	ACTGGCATCCACTGAGCTG	
314	wmc438F	TGTAAAACGACGGCCAGTGACCGTTGGGCTGTATAGCATT	Monomorphic
	wmc438R	CTCTGACAGTGGTGGAGCTTGA	
315	gwm111F	TGTAAAACGACGGCCAGTTCTGTAGGCTCTCTCCGACTG	Monomorphic
	gwm111R	ACCTGCTCAGATCCCACTCG	
316	wmc634F	TGTAAAACGACGGCCAGTAGCGAGGAGGATGCATCTTATT	Monomorphic
	wmc634R	GACATACACATGATGGACACGG	
317	cfd175F	TGTAAAACGACGGCCAGTTGTCGGGGACACTCTCTCTT	No amplification
	cfd175R	ACCAATGGGATGCTTCTTTG	