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Genetic analysis of parthenogenetic capability and fecundity in *Drosophila albomicans*

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Abstract

Background: The successful rate of parthenogenesis in *Drosophila* harvested from natural population was extremely low, which could be effectively improved under selection pressure. Facultative parthenogenesis in *Drosophila albomicans* may be advantageous for its expansion from sub-tropical to temperate area. Since the understanding of the genetics involved in the capability and fecundity of parthenogenesis is limited, this study aims to preliminarily map the chromosome regions that are preferentially important for parthenogenesis.

Results: Genetic mapping was performed with F₂ individuals that were parthenogenetically produced by F₁ from crosses between a parthenogenetic strain K KU119 and a sexual strain #55.1 of *Drosophila albomicans*. Among 105 F₂, 53.3% of them had parthenogenetic capability which is highly associated with three markers a28, c4081, and c7198 located near or inside *In(2L)B₁D₅*. A sexual strain with high *In(2L)B₁D₅* heterozygosity originating from Wulai, Taiwan in 1970 was able to perform parthenogenesis. However, the fecundity of those F₂ varied in a wide range, forming a continuous distribution as expectation of a quantitative trait and was correlated with the number of homozygous markers for all markers on the second chromosome and neo-X chromosome arm.

Conclusions: We have genetically analyzed the capability and fecundity of parthenogenesis in *Drosophila albomicans*. The former is specifically associated with a limited region in the B₁ to D₅ of 2L arm where inversion *In(2L)B₁D₅* may play certain role for the maintenance of parthenogenesis, whereas the latter is apparently related to several quantitative loci on the second chromosome and neo-X chromosome arm.

Keywords: Facultative parthenogenesis; Genetic mapping; Inversion polymorphism

Background

Hundreds of lineages in the animal kingdom have experienced the evolution from sexual reproduction to parthenogenesis by which females reproduce offspring without mating (Schwander et al. 2010). As compared to sexual reproduction, the advantages of parthenogenesis include theoretically twofold fecundity, given all else is equal (Maynard Smith 1978), and increased potential of colonization in harsh, high latitude and/or altitude, isolated environments or newly invaded marginal habitats, where population size is usually too small for females to find mates (Suomalainen 1950). Alternatively, parthenogenesis may suffer disadvantages for long-term evolution because of the loss of genetic variation and

accumulation of deleterious mutations (Simon et al. 2003). Facultative parthenogenesis, coexistence of parthenogenesis and sexual reproduction, is apparently the better reproductive strategy than parthenogenesis (Hurst and Peck 1996).

Most parthenogenetic *Drosophila* species conduct facultative parthenogenesis (Templeton 1983), and hence, they may escape from the accumulation of deleterious mutations and display higher fitness in the condition of low population density. Although the hatchability of unfertilized eggs of parthenogenetic *Drosophila* harvested from natural population is extremely low, it could be significantly increased under certain selection pressure (Carson 1967; Stalker 1954; Markow 2013). Since productivities were increased with generations under artificial selection, parthenogenesis is considered to be regulated by polygenes. However, if parthenogenesis is controlled by a

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polygenic system, successful parthenogens will be hard to evolve (Fuyama 1986). A study on the parthenogenetic *Drosophila mercatorum* reported that the genotypes for incapability of parthenogenesis were quickly eliminated in the first few generations, and the population size varied among different genotypes after several generations of parthenogenesis (Annett and Templeton 1978). Therefore, we consider that there are only a few essential genes for parthenogenetic capability while there are many genes influencing the fecundity (i.e., the number of parthenogenetically produced offspring). The former phenotype is either capable or incapable to perform parthenogenesis, and the latter indicates the parthenogenetic efficiency of genetically different females. Genetic mapping for parthenogenesis conducted on *Drosophila ananassae* complex with parthenogenetic capability as phenotype indicated a major gene located on its 2L chromosome arm (Matsuda and Tobari 2004). Fuyama (1986) indicated that *Drosophila melanogaster* can perform gynogenetic reproduction (i.e., females can reproduce by mating with sterile males), which was related to recessive factors located between *Tft* locus and *nw* locus on 2R chromosome arm and *Gl* and *Sb* on 3R chromosome arm.

In the present study, we used *Drosophila albomicans*, a species invading from sub-tropical areas (i.e., probably Wulai, Taiwan) to the temperate climate zone (Ohsako et al. 1994), as material for genetic mapping. Parthenogenesis of *Drosophila albomicans* was first reported by Ohsako and Fuyama (1995) in Kiikatsuura, Japan which was the boundary of *Drosophila albomicans* distribution. Strong natural selection in favor of parthenogenesis may be operated for an adaptation to a cold climate in which population density is low. Parthenogenetic strain was established with 80% to 90% of them capable of parthenogenesis and about 11 offspring were reproduced per female from 11th to 30th generation after strain establishment (Ohsako and Fuyama 1995). In order to find a proper sexual strain to cross with this parthenogenetic strain, we checked the parthenogenetic capability of strains. We crossed between the sexual strain and parthenogenetic strain and collected F_1 virgin females to produce recombinant F_2 since meiosis was performed during parthenogenesis. Therefore, the F_2 offspring were adopted to map the genetic elements for parthenogenetic capability and do genetic analysis for parthenogenetic fecundity. We also discussed the maintenance of parthenogenesis in *Drosophila albomicans*.

Methods

Fly strains

The parthenogenetic *Drosophila albomicans* strain was provided as a courtesy from Dr. Fuyama. It was established from a virgin female of an isofemale strain (KKU119), which was originally collected from Kiikatsuura, Japan in

1990, and maintained by parthenogenesis ever since (Ohsako and Fuyama 1995). Isofemale sexual strains #55.1 and #56.1 were established in 1970 from Hualien and Wulai, Taiwan, respectively. For the chromosomal inversion arrangements, there is no inversion heterozygote on the neo-sex arm or the 2R arm in all of these three strains but only one inversion type, *In(2L)B₁D₅*, on the 2L arm. The strain #56.1 has about 75% *In(2L)B₁D₅* heterozygosity. The 2L arms of KKU119 and #55.1 are homozygous, but they carry different arrangements. Flies were maintained on standard cornmeal medium at 23°C under 12:12 h LD cycle. Flies used for crosses were sexed within 8 h after eclosion.

Parthenogenetic capability

In order to do proper genetic mapping, we made sure that the chosen sexual strain could not perform parthenogenesis. The parthenogenetic capability of strains #55.1 and #56.1 was examined by transferring virgin females to new vials twice a week until 4 weeks. The vials were checked for offspring reproduced in 4 weeks.

Recombinant individuals

In order to perform the genetic mapping for parthenogenesis through genotype-phenotype association, we generated recombinant individuals with known parthenogenetic phenotypes: capability and fecundity. Recombinant flies were produced through the following cross scheme: KKU119 females were crossed with #55.1 males, and their F_1 virgin females were subjected to reproduce F_2 parthenogenetically. The phenotypes of F_2 , including parthenogenetic capability and fecundity, were determined by culturing each individual F_2 in a vial and transferred to a new vial twice a week for consecutive 4 weeks. The parthenogenetic capability is determined by whether or not the F_2 parthenogenetically reproduces offspring in 4 weeks and the fecundity is the number of F_3 . Approximate 100 F_2 were genotyped using 14 PCR-RFLP markers, 7 on the second chromosome arm and 7 on the neo-X chromosome arm (Table 1). Single-fly genomic DNA was extracted using the Puregene Cell and Tissue DNA Isolation Kit (Gentra Systems, Minneapolis, MN, USA) following the manufacture's protocol.

Statistical analysis

The association between genotypes and parthenogenetic capability was revealed by Fisher's exact test. Linkage disequilibrium D' (Lewontin 1964) values among highly associated markers were calculated to reveal hitchhiking. When the linkage disequilibrium was high, we checked recombinant individuals through Fisher's exact test in order to reveal which of the two markers was more influential. Recombination rates for all markers were also

Table 1 Marker list with location, primer name, primer sequence, PCR annealing temperature, and restriction enzyme

Location	Primer name	Primer sequence	Annealing temperature (°C)	Restriction enzyme	Reference
Second chromosome	a28	F: GGGGCACACTGATTATTAACAA R: TATTTACGCCACAACCTGCAGCAC	57	<i>AluI</i>	Chang et al. (2008)
	a708	F: GAAAAGGGCGAACAGATAGA R: AACAGGAACATAGAAATCAC	55	<i>XmnI</i>	Chang (2011)
	A185D	F: CAAACGCTCTGGAATAATGG R: CTCGGGAGTGTGGGGC	55	<i>RsaI</i>	Chang (2011)
	c4081	F: GCCCTGGAACAAAGTAAAA R: AGTCTGCTGCGTATGGTCAA	52	<i>HaeIII</i>	This study
	c5237	F: TATATGTTCTCTGATTGG R: AAGTTTAAACGCGAACTTTT	55	<i>PstI</i>	Chang (2011)
	c5665	F: TGAAACATTATTACCGCTG R: GATGACGACGACGATTCC	60	<i>HaeIII</i>	This study
	c7198	F: GTGGGAAGCACGTTACAT R: CCATGAACAGTCTGAAGTTT	57	<i>MseI</i>	Chang (2011)
neo-X chromosome arm	a52	F: TATTCATCGCATTCCACAT R: GGCTTCCTCAATCAACTG	53	<i>HaeIII</i>	Chang et al. (2008)
	a386	F: GTTACGATTACGAAGAGTGC R: CTGCCGTGCTTATGTGAT	51	<i>XmnI</i>	Chang et al. (2008)
	a1185	F: ATTCTGTGCTTCGTTTTGA R: GATTTCGGCTTACATTATTG	49	<i>StyI</i>	Chang et al. (2008)
	a1350	F: TACGACCCCGTCAAAGGCTGTG R: GGCTTGTATGCGATTCTGC	52	<i>HpaII</i>	Chang (2011)
	a1953	F: GCCAACAGCGAGCCTTCT R: GCGACCCAAGCACGAATC	56	<i>DdeI</i>	Chang et al. (2008)
	c29	F: CTGGGCAAAGAGTGTAGG R: CAGAAGGAGGGCGAAAA	57	<i>RsaI</i>	Chang et al. (2008)
	c3242	F: TTGAAGCGCAGTTTATGCAC R: TACAACCACGACCTGGACAA	62	<i>SspI</i>	Chang (2011)

calculated in order to estimate the unknown locations of markers. In addition, the correlation between the parthenogenetic fecundity and the number of KKU119 homozygous markers carried by F_2 was done by the analysis of variance (ANOVA) test, and jackknifing was also performed to figure out whether or not there is any key marker for the fecundity.

Results

Association between markers linked to *In(2L)B₇D₅* and parthenogenetic capability

In order to find a proper sexual strain for genetic mapping, we first analyzed the parthenogenetic capability of these two sexual strains #55.1 and #56.1. Among 69 virgin females of strain #56.1, 2 of them (2.9%) were found to produce offspring by parthenogenesis, whereas none

of 72 virgins of strain #55.1 was observed to do so. Therefore, #55.1 was used to perform genetic mapping although they carried different chromosomal inversion arrangements. As to parthenogenetic capability, 56 virgin F_2 could parthenogenetically reproduce offspring in 4 weeks, indicating that it was 53.3% ($n = 105$) of those F_2 . Those F_2 were genotyped using 14 PCR-RFLP markers, and the genotyping result is shown in Additional file 1. The alleles from KKU119 were assigned as *P* and those from #55.1 as *S*. Since heterozygosity (frequency of *PS*) was very low (i.e., in average 1.5%), only homozygotes (i.e., *PP* and *SS*) were included for analysis. Based on haplotypes, recombination rates among seven markers on the second chromosome and those among seven on the neo-X chromosome arm were calculated and summarized, respectively, in Table 2. The locations of two

Table 2 Recombination rate (%) of markers on the second chromosome and neo-X chromosome arm

	Recombination rate (%)					
Second chromosome	a28	a708	A185D	c4081	c5237	c5665
a708	15.4					
A185D	44.6	56.9				
c4081	1.0	16.2	43.1			
c5237	33.7	39.0	45.1	32.4		
c5665	28.8	30.5	40.2	27.6	35.2	
c7198	1.9	17.3	45.5	2.9	32.7	29.8
neo-X chromosome arm	a52	a386	a1185	a1350	a1953	c29
a386	27.3					
a1185	33.0	37.8				
a1350	39.2	45.7	17.3			
a1953	26.8	32.4	44.8	52.0		
c29	46.9	42.6	51.6	46.5	30.6	
c3242	43.8	45.1	20.0	10.8	53.5	50.5

(a28 and a708) on the second chromosome and four (a1350, a386, a52, and c29) on the neo-X chromosome arm were previously determined (Chang et al. 2008; Chang 2011). Therefore, together with those markers with known locations and recombination rates (Table 2), a rough map was obtained and shown in Figures 1 and 2. In Figure 1, the marker a28 is located near the breakpoint D_5 of $In(2L)B_1D_5$ inversion (i.e., on the basal of 2L) and a708 is on the basal of 2R (Chang et al. 2008; Chang 2011). The marker A185D is probably located at the distal end of 2R arm due to the equal or over 50-cM distance with all other six markers. The markers c5237 and c5665 are likely located on the distal end of 2L arm, and c5237 may be located more distal than c5665 due to higher recombination rates between them and a708 (i.e., between c5237 and a708 is 39.0; between c5665 and a708 is 30.5) than those between them and a28 (i.e., between

c5237 and a28 is 33.7; between c5665 and a28 is 28.8). The extremely low pair-wise recombination rates among c4081, c7198, and a28 (Table 2) indicate that they should be regarded as one unit which may be located near or inside the inversion. However, their locations cannot be determined due to heterozygous $In(2L)B_1D_5$ in F_1 (Figure 1). Figure 2 shows the markers on the neo-X chromosome arm. There are four markers with known locations including a1350 located near the centromere, a386 and a52 located in the middle of the chromosome, and c29 located on the tip (Chang et al. 2008). Based on the recombination rates (Table 2), we estimated the relative locations for other markers, a1185, a1953, and c3242, as shown in Figure 2. The 14 markers, except 3 related to the $In(2L)B_1D_5$, are scattered on those chromosome arms.

The association between genotypes (i.e., PP and SS) and parthenogenetic capability (i.e., with and without parthenogenetically produced offspring) was proceeded. As shown in Figure 3, six (a28, a708, c4081, c5237, c5665, and c7198) of the seven markers on the second chromosome showed statistically significant association (Fisher's exact test, $p < 0.01$ for c5237 and $p < 0.001$ for other five markers), whereas none of the seven markers on the neo-X chromosome arm showed significant association (Fisher's exact test, $p > 0.05$). High linkage disequilibrium values appeared among all six markers strongly associated with parthenogenetic capability (Table 3). Among these markers, a28 was always related to parthenogenetic capability in those pair-wise comparisons (Table 4). Small amount of recombinants among markers, a708, c5237, and c5665 showed significant difference from a28 (Fisher's exact test, $p < 0.01$, Table 4). However, the amount of recombinants was too small to discriminate the contribution among a28, c4081, and c7198. Due to the crossing-over inhibition caused by the heterozygous inversion, we may infer that loci involved in parthenogenetic capability are associated with $In(2L)B_1D_5$.

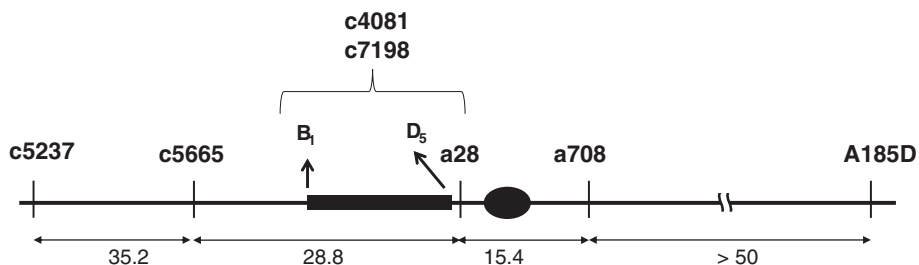


Figure 1 Locations of seven markers on the second chromosome. The genetic distances are shown by percentage of recombination rate. The locations of a28 and a708 were revealed by *in situ* hybridization (Chang et al. 2008; Chang 2011). The recombination rate statistically indistinguishable from 50% was marked as >50 cM. Black horizontal line shows the second chromosome, and a black oval dot represents the centromere. The black bar indicates $In(2L)B_1D_5$ region and D_5 is near the centromere. Two markers c4081 and c7198 may be located near or inside the inversion (see text).

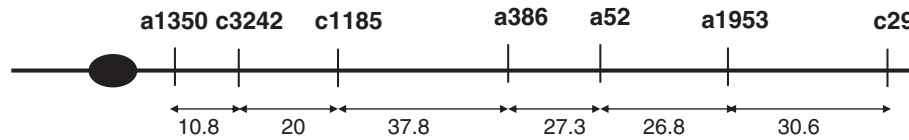


Figure 2 Locations of seven markers on the neo-X chromosome arm. The genetic distances are shown by percentage of recombination rate. The locations of a1350, a386, a52, and c29 were revealed by *in situ* hybridization (Chang et al. 2008). Black horizontal line shows the neo-X chromosome arm, and a black oval dot represents the centromere.

The distribution of quantitative parthenogenetic fecundity loci

The fecundity of F_2 varied from 1 to 59 (in average 16.7 ± 16.2) and showed a continuous distribution as expectation of a quantitative trait (Figure 4). The significant correlation between the fecundity and the number of homozygous markers (ANOVA test, $p < 0.001$) is shown in Figure 5. Through jackknifing, the significant correlation remains no matter which marker was omitted from the re-sampling which indicates no single major gene influencing fecundity.

Discussion

The facultative parthenogenesis, occasional parthenogenesis with primarily sexual reproduction, is not unusual in insect. It is found in at least ten insect orders (Simon et al. 2003). The success of parthenogenesis is in general dependent on parthenogenetic capability as well as fecundity. In the present study, we performed genetic analysis using 14 molecular markers to reveal that parthenogenetic capability is associated with a limited number

of gene loci on 2L chromosome, whereas parthenogenetic fecundity is influenced by numerous quantitative loci distributed on whole genome.

In order to locate the gene for parthenogenetic capability, sexual strains were checked for their parthenogenetic capability. Since females from #55.1 did not perform parthenogenesis, this strain was chosen to cross with KKU119, and their F_2 offspring parthenogenetically produced by F_1 were used for the genetic analysis of parthenogenesis. An advantage of adopting this strain is that their parthenogenetic capability and fecundity must be contributed by KKU119, which has been cultured for 370 generations by parthenogenesis. Their parthenogenetic capability gene(s) should have high or even fixed frequency. Although crossing between strains #55.1 and KKU119 produces heterozygous $In(2L)B_1D_5$ F_1 and invalidates mapping on a portion of the 2L arm, other chromosome regions covered by scattered markers are not influenced. After crossing, all sexually reproduced F_1 have the same genotypes, but their parthenogenetic F_2 may have different

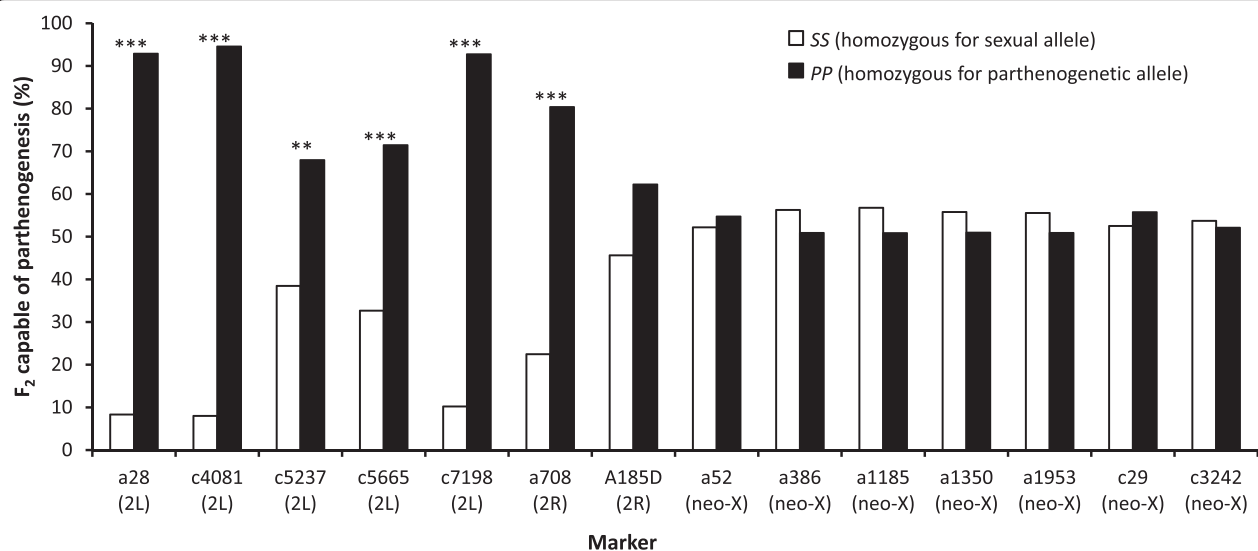


Figure 3 Proportion of F_2 individuals able to perform parthenogenesis with *PP* or *SS* genotypes for each marker. *PP*, homozygous for parthenogenetic allele (shown in black); *SS*, homozygous for sexual allele (shown in white). Sample size is 100 to 105. Markers with significant association between genotypes and parthenogenetic capability through Fisher's exact test were marked $**p < 0.01$ and $***p < 0.001$. Locations of markers are shown in parentheses.

Table 3 Linkage disequilibrium (D') for markers shown to be highly associated with parthenogenetic capability

	a708	c4081	c5237	c5665	c7198
a28	0.93	1.00	0.59	0.71	1.00
a708		0.92	0.42	0.68	0.91
c4081			0.63	0.74	1.00
c5237				0.54	0.61
c5665					0.69

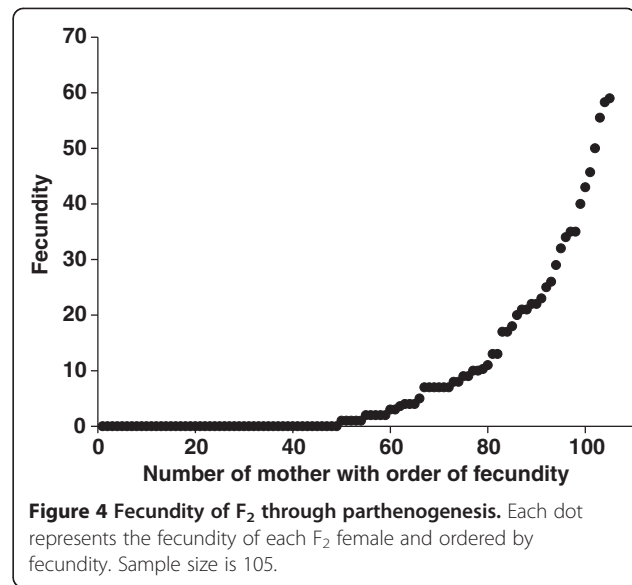
genotypes via recombination. Therefore, those F_2 were subjected to investigate the association between genotypes and parthenogenetic capability.

About half of recombinant homozygous F_2 were unable to do parthenogenesis, which corresponds to the about half which did not acquire the capability allele from the parthenogenetic strain (i.e., ranging from 39% to 56% for 14 markers). On the contrary, the other half of them able to do parthenogenesis may carry the parthenogenetic allele. These results implicate that there is only one genetic element responsible for parthenogenetic capability. Among all markers, the three markers, a28, c4081, and c7198, were most strongly associated with parthenogenetic capability. However, due to crossing-over inhibition of the heterozygous $In(2L)B_1D_5$, they were treated as one genetic element contributing to parthenogenesis. In other words, the capability gene may be located very close to $In(2L)B_1D_5$. The only marker on the second chromosome, A185D, which did not associate with parthenogenesis is located over 50 cM apart from the other six markers therefore assorted independently just like markers on neo-X chromosome arm. To sum up, we infer that the parthenogenetic capability gene or gene cluster is located on 2L arm and is strongly associated with $In(2L)B_1D_5$. However, due to suppressed recombination rates on

Table 4 Number of recombinant F_2 categorized into genotypes and whether or not produced F_3 offspring parthenogenetically

Pair of markers with recombination	Genotypes	Number of F_2		P value ^a
		Without F_3	With F_3	
a28, c5237	SS, PP	15	1	<0.001
	PP, SS	2	17	
a28, c5665	SS, PP	15	0	<0.001
	PP, SS	3	12	
a28, a708	SS, PP	8	0	<0.01
	PP, SS	1	7	
a28, c7198	SS, PP	1	0	NA
	PP, SS	0	1	
a28, c4081	SS, PP	0	0	NA
	PP, SS	1	0	

^aBased on Fisher's exact test. NA, not applicable.



2L arm in heterozygous F_1 , the precise location of the major gene(s) was difficult to be mapped by current genetic analysis. To that purpose, it is essential to find another sexual strain which is unable to perform parthenogenesis while carries the same chromosome arrangement of 2L as that of the parthenogenetic strain for detailed mapping of the major gene(s).

Since the major gene or gene cluster is highly linked to $In(2L)B_1D_5$ inversion, the high $In(2L)B_1D_5$ heterozygosity may be one reason for the preservation of parthenogenetic capability in *Drosophila albomicans*. It was reported that *Drosophila mercatorum* from natural populations which carried high heterozygosity are more capable of parthenogenesis than those from sexual inbreeding which carried high homozygosity (Templeton et al. 1976a), so it may suggest that the high heterozygosity plays an important role in the maintenance of parthenogenesis. A *Drosophila albomicans* sexual strain #56.1 originating from Wulai population, which has the closest phylogenetic relationship to Japan population (Ohsako et al. 1994), was able to do parthenogenesis in spite of strong genetic drift and lacking selection for parthenogenesis with 40-year long-term sexual reproduction culture. The high $In(2L)B_1D_5$ heterozygosity in #56.1 probably preserved parthenogenetic capability gene in the laboratory strain. In natural populations, high $In(2L)B_1D_5$ heterozygosity was observed in winter in Wulai populations (Chang et al. 1987; Yang et al. 2002) and in the Japan population (Ohsako et al. 1994). Inversion heterosis may be responsible for the maintenance of parthenogenetic capability alleles in a population; however, inversion may not have direct relationship with the gene.

Parthenogenetic fecundity may be contributed by many loci widely distributed on whole genome. The parthenogenetic fecundity of F_2 was shown as a quantitative trait,

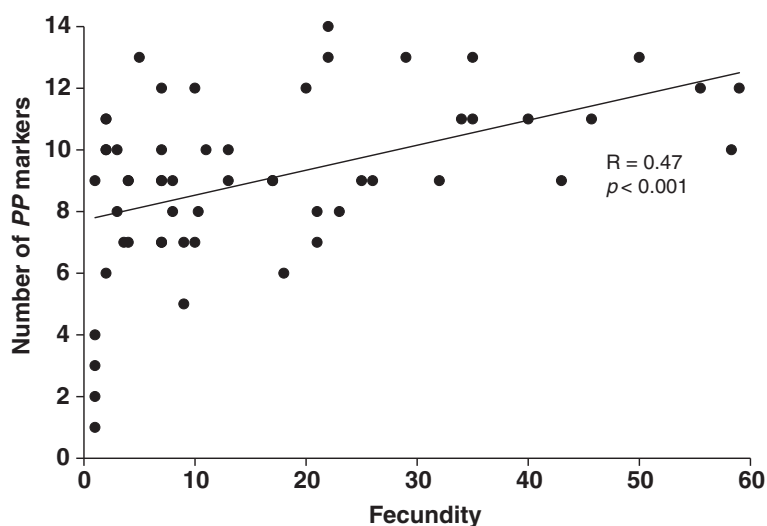


Figure 5 The correlation between the fecundity and the number of *PP* markers. The x-axis is the fecundity of each F_2 , and the y-axis is the number of *PP* (homozygous for parthenogenetic alleles) markers for each F_2 . Sample size is 56. ANOVA test, $p < 0.001$.

suggesting that there may have many loci with additive effect influencing fecundity. The significant correlation between the fecundity and the number of homozygous markers (ANOVA test, $p < 0.001$) suggests that the individuals with more *PP* markers may tend to have higher fecundity. That is, there are many loci with additive effect influencing parthenogenetic fecundity. The consistent significant correlation through jackknifing supports the quantitative trait model (i.e., many loci with small effect) and numerous alleles regulate the parthenogenetic fecundity. Identification of genes involved in parthenogenetic fecundity warrants our further investigation. In *Drosophila mercatorum*, the 'coadapted genome' for parthenogenesis was suggested through the decreasing fecundity with higher perturbation level, through recombinant lines generated by crossing different parthenogenetic strains (Templeton et al. 1976b), and the coadaptation was formed effectively under the selection for parthenogenesis (Templeton 1979). Therefore, the additive effect for parthenogenetic fecundity is likely to respond to artificial selection for parthenogenesis (Ohsako and Fuyama 1995), and it might be coadapted with the gene for parthenogenetic capability.

Conclusions

In *Drosophila albomicans*, parthenogenetic capability is regulated by one genetic element located on 2L arm, and the high *In(2L)B₁D₅* heterozygosity may play a role in preserving parthenogenetic capability since the genetic element responsible for parthenogenetic capability is closely associated with *In(2L)B₁D₅*. Moreover, parthenogenetic fecundity shown as a quantitative trait by its continuous distribution is regulated by several additive loci,

and through jackknifing, there is no highly influential one among the 14 markers. Therefore, we may infer that parthenogenetic fecundity is related to many genes in the genome including 2R, 2L, and neo-X chromosome arms.

Additional file

Additional file 1: The fecundity and 14 molecular marker

genotypes of 105 F_2 . The additional file contains the fecundity which is the number of offspring parthenogenetically reproduced by F_2 and the genotypes of 14 molecular markers. Each row represents one F_2 individual with their own number. *P* represents the allele from the parthenogenetic strain, KKU119, and *S* represents the allele from the sexual strain, #55.1. NA means the data was not obtained successfully.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

CC and HC designed the study and wrote the manuscript. CC carried out the laboratory work and analyzed the data. Both authors read and approved the final manuscript.

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References

- Annett JL, Templeton AR (1978) Genetic recombination and clonal selection in *Drosophila mercatorum*. *Genetics* 89:193–210
- Carson HL (1967) Selection for parthenogenesis in *Drosophila mercatorum*. *Genetics* 55:157–171

- Chang CH (2011) Early-stage evolution of the neo-Y chromosomes in *Drosophila albomicans*, Master thesis, Graduate Institute of Ecology and Evolutionary Biology, National Taiwan University, Taipei, Taiwan
- Chang H, Chang SH, Lin FJ (1987) Effects of climatic factors on the heterozygosity of *In(2L)B₁D₅* in *Drosophila albomicans*. *B I Zool Acad Sinica* 26:39–45
- Chang TP, Tsai TH, Chang H (2008) Fusions of Muller's elements during chromosome evolution of *Drosophila albomicans*. *Zool Stud* 47:574–584
- Fuyama Y (1986) Genetics of parthenogenesis in *Drosophila melanogaster*. II. Characterization of a gynogenetically reproducing strain. *Genetics* 114:495–509
- Hurst LD, Peck JR (1996) Recent advances in understanding of the evolution and maintenance of sex. *Trends Ecol Evol* 11:46–52
- Lewontin RC (1964) The interaction of selection and linkage. I. General considerations; heterotic models. *Genetics* 49:49–67
- Markow TA (2013) Parents without partners: *Drosophila* as a model for understanding the mechanisms and evolution of parthenogenesis. *G3-Genes Genom Genet* 3:757–762
- Matsuda M, Tobar YN (2004) Genetic analyses of several *Drosophila ananassae*-complex species show a low-frequency major gene for parthenogenesis that maps to chromosome 2. *Genet Res* 83:83–89
- Maynard Smith J (1978) *The evolution of sex*. Cambridge University Press, New York
- Ohsako T, Fuyama Y (1995) *Drosophila albomicans*, a new member of parthenogenetic *Drosophila*. *Dros Inf Serv* 76:150
- Ohsako T, Aotsuka T, Kitagawa O (1994) The origins of the Japanese mainland population of *Drosophila albomicans*. *Jpn J Genet* 69:183–194
- Schwander T, Vuilleumier S, Dubman J, Crespi BJ (2010) Positive feedback in the transition from sexual reproduction to parthenogenesis. *Proc Biol Sci* 277:1435–1442
- Simon JC, Delmotte F, Risper C, Crease T (2003) Phylogenetic relationships between parthenogens and their sexual relatives: the possible routes to parthenogenesis in animals. *Biol J Linn Soc* 79:151–163
- Stalker HD (1954) Parthenogenesis in *Drosophila*. *Genetics* 39:4–34
- Suomalainen E (1950) Parthenogenesis in animals. *Adv Genet* 3:193–253
- Templeton AR (1979) The unit of selection in *Drosophila mercatorum*. II. Genetic revolution and the origin of coadapted genomes in parthenogenetic strains. *Genetics* 92:1265–1282
- Templeton AR (1983) Nature and experimental parthenogenesis. In: *The genetics and biology of Drosophila*, vol 3c. Academic, New York, pp 343–398
- Templeton AR, Carson HL, Sing CF (1976a) The population genetics of parthenogenetic strains of *Drosophila mercatorum*. II The capacity for parthenogenesis in a natural, bisexual population. *Genetics* 82:527–542
- Templeton AR, Sing CF, Brokaw B (1976b) The unit of selection in *Drosophila mercatorum*. I. The interaction of selection and meiosis in parthenogenetic strains. *Genetics* 82:349–376
- Yang YY, Lin FJ, Chang H (2002) Comparison of recessive lethal accumulation in inversion-bearing and inversion-free chromosomes in *Drosophila*. *Zool Stud* 41:271–282

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