Identification and Characterization of a gene required for meiosis I mono-orientation of sister chromatids in Drosophila

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Abstract

Prior to meiosis, kinetochores of sister chromatids are replicated, functional and important for orienting chromosome alignment at the metaphase plate. At meiosis I sister kinetochores must attach to the same spindle pole to ensure that sister chromatids are segregated to the same daughter cell. In yeasts, the monopolin complex mediates this co-orientation of sister kinetochores to promote proper chromosome segregation. Monopolin constituents are not well conserved across phyla; Meikin appears to carry out the same function in humans and other organisms. Analogues to monopolin components have not yet been identified in the fruit fly *Drosophila melanogaster*; despite a conserved function of chromosome segregation in meiosis I. Here, I describe an EMS-induced meiotic mutant, CG30383^{Z}, in *Drosophila melanogaster* that is defective in co-orientation of sister kinetochores at meiosis I. In genetic tests of chromosome transmission, mutants in CG30383^{Z} produce both reductional nondisjunction of homologs and equational nondisjunction of sister chromatids. Complementation tests using a deletion allele have revealed that this is the null phenotype. Furthermore, using fluorescence *in situ* hybridization, I observed that mutant CG30383^{Z} sister chromatids had separated and segregated to opposite poles at meiosis I in 69.3% of meiotic cells. All aspects of this phenotype suggest that I have identified the first component of the monopolin complex in flies.

Introduction

Meiosis is a highly conserved process where two cellular divisions occur sequentially without an intervening DNA replication to form haploid gametes. This complex event provides the basis for most sexual reproduction. Unlike mitosis, meiosis I (MI) is characterized by a reductional division where homologous pairs are lined up at the metaphase plate and segregate to opposite poles (McCollum, 2012). Then, in meiosis II (MII), sister chromatids separate from each other as in mitosis. This intricate bi-divisional process is crucial for genetic diversification and is significantly different from mitosis because of how the sister chromatids are pulled to the same side (mono-orientation) of a germ cell during the first division. The kinetochore is a crucial element regulating chromosome segregation in both meiosis and mitosis. The kinetochore apparatus plays a major role in determining which direction chromosomes are pulled: unidirectional or bidirectional. Although each sister chromatid has a functional kinetochore, sister chromatids are always mono-oriented at the metaphase plate in meiosis I and are both pulled to one side of the dividing cell. Certain meiotic mutants, however, can disrupt this process, and these presumably identify genes required for this mono-orientation. Mutations in monopolin-complex genes result in a mitotic-like division in meiosis I in which sister kinetochores attached to opposite poles, leading to the aberrant separation of sister chromatids (Toth *et al.*, 2000). Monopolin is proposed to act as a sister chromatid crosslink that mediates the co-orientation of sister kinetochores (Corbett and Harrison, 2016) to ensure that they segregate like a single chromosome.

The monopolin complex has been most fully characterized in budding yeast, *Saccharomyces cerevisiae,* and includes four proteins Mam1, Csm1, Lrs4, and Hrr25 necessary for sister kinetochore localization and co-orientation (Toth *et al.*, 2000; Rabitsch *et al.*, 2003; Petronczki *et al.,* 2006). S. *cerevisiae* have a single “point” centromere, in which a single microtubule attaches to a sister kinetochore. Monopolin elements fuse sister chromatids together so that they bind to

a single microtubule to achieve co-orientation (Corbett and Harrison, 2016). The analogous human complex has many similarities to the yeast complex and some components seem to be conserved. MEIKIN, a meiosis-specific kinetochore protein in mammals, functions only in meiosis I and has a major influence in mono-orientation of chromosomes (Kim, J. *et al.*, 2015). Assembly of MEIKIN, is mediated by the kinetochore protein CENP-C which further enhances chromosome cohesion; however, the mammalian system has more complex centromeric microtubule attachments than the single "point" attachment yeast has (Kim, J. *et al.*, 2015; Tanaka *et al.*, 2009). Analogous components of a monopolin complex have not yet been discovered in the *Drosophila* system, despite concerted efforts to identify them.

Genetic mutants offer valuable insight into a gene or protein's function and most *Drosophila* meiotic mutants are sex specific due to a variation in male and female genetic processes; however, some affect both sexes. *Drosophila* males and females have developed different genetic mechanisms to accomplish meiosis I, as females undergo crossover and the formation of a synaptonemal complex while the males execute neither. The regulation of sister chromatid cohesion and orientation, however, appears to be shared by both, as mutations in *meiS332* and *ord* (Kerrebrock *et al.* 1995; Goldstein 1980; Mason 1976) cause precocious sister separation in both sexes. Ord is required in meiosis I to ensure cohesion, while meiS332, a Shugoshin homolog, acts to protect centromeric cohesin from degradation until anaphase of meiosis II (Goldstein 1980; Mason 1976). Centromeric cohesins ensure that sisters segregate together at meiosis I. Whether co-orientation of sister kinetochores at meiosis I is controlled the same way in both sexes is not known.

In both sexes, sister kinetochores are fully functional but attach to the same pole, presumably due to a monopolin complex analog. A proteinaceous complex is responsible for orienting sister kinetochores in the same direction to undergo reductional division in meiosis. A reductional division is a reduction in chromosome number from diploid (two sets of chromosomes) to

haploid (one set) and usually occurs in the first division of meiosis. In contrast, equational division results in the same chromosome number after division and occurs during mitosis and meiosis II.

The monopolin complex has still not been discovered in many eukaryotic phyla including the fruit fly *Drosophila melanogaster*, despite the conserved function of sister chromatid mono-orientation in meiosis I. This suggests that divergent genes have developed similar functions in other species. Here, I describe a new mutation, CG30383^{Z}, that affects sister chromatid orientation at meiosis I. I present both genetic and cytogenetic evidence that CG30383^{Z} mutants exhibit exceptional effects on meiotic chromosome segregation in both males and females, suggesting the gene's importance for both sexes. Because mutants in CG30383^{Z} display a similar phenotype to monopolin-complex mutants, I suggest that the CG30383^{Z} expression facilitates an analogous monopolin complex in D. *melanogaster*.

Materials and Methods

All stocks were obtained from the Bloomington Stock center except for CG30383^{Z}. This mutation was identified in a screen that perturbed transmission of the fourth chromosomes in male meiosis (Wakimoto *et al.*, 2004). The deficiency stock used was *w^{1118}; Df(2R)BSC265/CyO*. The CG30383^{Z} allele with a P-element insertion stock used was *w^{1118}; PBac{PB}CG30383^{c00440}* .

To test for meiotic nondisjunction (NDJ)*, y/y+Y; spa* males were crossed to *y w sn; C(4)ciey/0* females. Normal progeny resulting from euploid gametes are y+ males and y w sn females. Exceptional progeny resulting from diplo-XY sperm are y+ females, and those resulting from nullo- XY sperm are y w sn males. Fourth chromosome nondisjunction was observed as ci ey progeny (from nullo-4 sperm) and spa progeny (from diplo-44 sperm). The frequency of meiosis I nondisjunction was calculated as (NDJ progeny)/ total progeny.

To analyze whether mutation of CG30383^{Z} affects meiosis II behavior in males, I crossed *yw/y^{+}Y;* CG30383^{Z} *; spa^{pol} males to C(1)RM y v /0 females*. Because the *C(1)RM y v /0 females* possess a compound X chromosome, I can distinguish which progeny are products of abnormal chromosome behavior in MII depending on how the sister chromatids disjoin. Normal progeny resulting from euploid gametes are y males. Exceptional progeny resulting from diplo-XY sperm (MI NDJ) are y+ males. Exceptional progeny resulting from nullo-XY sperm are y v females (either MI or MII NDJ). Lastly, exceptional progeny resulting specifically from MII NDJ will receive both paternal X sister chromatids and will be recognized as y w females. Fourth chromosome NDJ will appear as either spa or ci and ey and will arise from either MI or MII NDJ.

To examine whether mutation in CG30383^{Z} influenced females I crossed *yw; mcI ; spa*^{pol} females to *y w sn/Yy+ ; C(4)RM ci ey /0* males *.* Maternal sex chromosome NDJ will result in XX ova and give rise to y+ w females and y w sn males. Fourth chromosome NDJ would produce either spa or ci ey offspring. These crosses did not allow distinction between MI and MII NDJ.

To understand the phenotypic effects of mutation in CG30383^{Z}, I followed the protocol for fluorescent in situ hybridization, or FISH, as described by Hylton *et al. (*2019) to visualize male meiotic chromosomes. A green alexa fluor-486 probe was hybridized to a Y chromosome repeat (AATAC) and a red alexa fluor-586 probe was hybridized to a unique segment of X euchromatin. These red and green probes were used to label both the sex chromosomes so that meiotic chromosome segregation could be observed.

Results

Various genetic crosses were performed to find the frequencies of nondisjunction and phenotypic effects of genetic mutants for CG30383^{Z}**.** When the CG30383^{Z} gene was mutated nondisjunction occurred for all tested allele combinations. Among progeny of males

homozygous for CG30383^{Z}, I observed around 50% non-disjunction of both sex and fourth chromosomes. This suggests that in the absence of functional CG30383 gene product, both sex and fourth chromosomes segregate at random at meiosis I. When this allele was tested *in trans* to a deficiency (*w^{1118}; Df(2R)BSC265/CyO*) there were similar frequencies of nondisjunction. This indicates that this phenotype reflects the null phenotype that results when the gene function is completely removed. By complementation testing, I identified a third allele resulting from a P-element insertion in CG30383^{Z} (*w^{1118}; PBac{PB}CG30383^{c00440}*). Frequencies of NDJ were lower for both sex and fourth chromosomes involving this allele, suggesting that this was a hypomorphic allele. Consistent with this conclusion, frequencies of NDJ among progeny of P-allele/P-allele were lower than frequencies among progeny of *CG30383^{c00440}*/CG30383^{Z}, and *CG30383^{c00440}*/Df males were all less than CG30383/Df males (Table 1).

Table 1a. Sex and fourth chromosome disjunction data from crosses of *y/y^{+}Y; mcl ; spa^{pol}* males to *y w sn ; C(4)RM ci ey /0* females

Recovered male gametes:			*Y;4*	*X;4*	*0;4*	*X/Y;4*	*X;0*	*Y;0*	*X;4/4*	*Y;4/4*	*0;0*	*0;4/4*	*X/Y;0*	*X/Y;4/4*
65-61	E	1331	164	209	255	83	101	79	93	84	107	81	43	32
65-61/P	E	1187	234	228	206	89	71	64	71	66	67	42	26	23
65-61/Df	E	1147	184	196	194	83	71	58	51	55	95	74	43	43
P/P	E	2071	532	546	265	86	85	112	112	132	59	80	39	23
P/Df	E	1640	362	376	229	96	72	82	104	123	62	77	22	35
65-61/Cy	C	1444	567	872	5	0	0	0	0	0	0	0	0	0
P/Cy	C	1549	663	881	4	1	0	0	0	0	0	0	0	0
Df/Cy	C	997	591	406	0	0	0	0	0	0	0	0	0	0

Table 1b. Nondisjunction frequency of *y/y^{+}Y; mcl ; spa^{pol}* males to *y w sn ; C(4)RM ci ey /0* females

% Nondisjunction	nullo XY	diplo XY	nullo 4	diplo 4	XY	4	XY + 4*
65-61	33.3	11.9	24.0	21.8	45.2	45.8	19.8 (20.7)
65-61/Df	31.6	14.7	23.3	19.4	46.3	42.7	22.2 (19.8)
65-61/P	26.5	7.6	19.2	17.0	34.1	36.2	13.3 (14.8)
P/P	19.5	7.1	18.1	16.8	26.6	34.9	9.7 (9.3)
P/Df	22.4	9.3	14.5	21.3	31.7	35.8	12.0 (11.3)

*Frequencies of simultaneous sex and fourth chromosome nondisjunction. Observed and (Expected based on independence).

Because these tests only detected nondisjunction in meiosis I, I wanted to see if mutation in CG30383^{Z} also affected meiosis II behavior. To test this, I crossed *yw/y^{+}Y; CG30383; spa^{pol}* males to *C(1)RM y v /0* females (Table 2a-b). I found that mutation in CG30383^{Z} does affect meiosis II chromosome segregation. When I tested CG30383^{Z} in trans to a deficiency (*w^{1118}; Df(2R)BSC265/CyO*) nearly 30% (29.5%; table 2b) of nullo-gametes were recovered. These could have been produced from either MI or MII NDJ. I also observed diplo-exceptional progeny; the diplo-XY, resulting from MI NDJ, and diplo-XX gametes, resulting from MII NDJ were recovered at 15.1% and 5.1%, respectively. The diplo-XX progeny could only be produced from an aberrant equational division. A rare diplo-XXY class was also observed at a frequency of 0.6% (Table 2b) and is most likely a result of sequential nondisjunction: XY NDJ in the MI reductional division and NDJ of sister X chromatids in the equational division.

Table 2a. Progeny collected from cross of *yw/y⁺Y; CG30383 ; spa*^{pol} males to *C(1)RM y v /0* females test MII effects of CG30383^{Z} in trans to a Deficiency (Df)

Recovered male gametes:		*X*	*Y*	*0*	*XY*	*XX*	*XXY*
Paternal Genotype							
Z2-65-61 /Df	E	265	262	313	160	54	6
Z2-65-61 /Cy	C	268	200	1	1	0	0
Df /Cy	C	0	0	0	0	0	0

Table 2b. Frequencies of progeny NDJ *yw/y⁺Y; CG30383 ; spa*^{pol} males to *C(1)RM y v /0* females

65-61/Df	29.5	15.1	5.1	0.6	33.5	16.8
65-61/Cy	0.2	0.2				

*Frequencies of simultaneous sex and fourth chromosome nondisjunction. Observed and (Expected based on independence).

To ask if mutations in CG303083^{Z} affect both sexes, I crossed *yw; CG30383^{Z} ; spa*^{pol} females to *y w sn/Yy+ ; C(4)RM ci ey /0* males (Tables 3a-b). Females homozygous for CG30383^{Z} were sterile; however, crosses were fertile in trans-heterozygous allele combinations. This was likely due to an unrelated female sterile mutation at another locus on the chromosome. In progeny of *CG30383^{Z} in trans* to a deficiency (*w^{1118}; Df(2R)BSC265/CyO*), there was 37.5% and 48.8% nondisjunction of the X and fourth chromosome, respectively. In crosses with the P-element insertion, the progeny exhibited nondisjunction as well. CG30383^{Z}/P-element progeny showed a frequency of 44.9% and 55.1% for the X and fourth chromosome, respectively. Progeny from homozygous P-element insertion had a frequency of 31.8% and 47.5% for sex and fourth chromosome, respectively. Lastly, P-element in trans to a deficiency showed a

nondisjunction frequency of 37.2% for the X chromosome and 49.7% for the fourth chromosome.

Table 3a. Female NDJ was tested by crossing of *yw; mcl; spa*^{pol} females to *y w sn/Yy+; C(4)RM ci ey /0* males. Control crosses were tested, and heterozygous wildtype flies were recovered with Curly (Cy) wings

Recovered female gametes:			X; 4	X; 0	X; 44	XX; 4	XX; 0	XX; 44	0; 4	0; 0	0; 44
Maternal Genotype											
65-61	sterile 77/77										
65-61/Df	E	469	188	77	78	50	28	14	52	13	19
65-61/P	E	376	121	66	71	47	26	18	52	8	18
P/P	E	675	240	81	139	58	28	33	56	20	20
P/Df	E	596	202	104	76	56	33	27	44	35	21
65-61/Cy	C	229	229	0	0	0	0	0	0	0	0
P/Cy	C	864	855	1	6	1	0	0	1	0	0
Df/Cy	C	408	406	0	0	1	0	0	1	0	0

Table 3b. Calculations of nondisjunction frequency in progeny of *yw; mcl ; spa*^{pol} females to *y w sn/Yy+ ; C(4)RM ci ey /0* males

% Nondisjunction	nullo X	diplo XX	nullo 4	diplo 4	X	4	X + 4*
65-61	sterile						
65-61/Df	17.9	19.6	25.1	23.7	37.5	48.8	15.8 (18.3)
65-61/P	20.7	24.2	26.6	28.5	44.9	55.1	18.6 (24.73)
P/P	14.2	17.6	19.1	28.4	31.8	47.5	15.0 (15.1)
P/Df	17.7	19.5	28.9	20.8	37.2	49.7	19.5 (18.5)

*Frequencies of simultaneous sex and fourth chromosome nondisjunction. Observed and (Expected based on independence).

My next goal was to understand the meiotic mechanism that is disrupted due to mutation in CG30383^{Z} at the cytological level. Male meiosis has some benefits that help facilitate scoring of

cells. Inside the testes, 16 interconnected primary spermatocytes enter meiosis and divide synchronously to produce 32 secondary spermatocytes. After meiosis II, 64 spermatids are produced. Because secondary spermatocytes and spermatids are formed in the same vicinity, scoring meiotic cells based on developmental stage is clear and straightforward. Using FISH, the X and Y chromosomes were labeled and observed in meiotically dividing cells.

Interestingly, from cytologically characterizing meiotic cells, I found that sister chromatids of both the X and Y chromosomes had separated and segregated to opposite poles at meiosis I, producing secondary spermatocytes each with one X and one Y chromosome. This class of NDJ in which the X and Y chromosomes prematurely separated bi-directionally occurred 69.3% of the time (Figure 1b; table 4a). Normal division in secondary spermatocytes occurred 22.8% of the time and was characterized by paired X and Y chromosomes in separate nuclei (Fig. 1; Table 4a). Other exceptional classes of MI NDJ were rare and resulted from aberrant sister chromatid segregation of either the X or Y chromosome (Table 4a). Chromosome segregation in meiosis II was also affected and resulted in multiple disjunction phenotypes (Table 4b). A majority of aneuploid spermatids were observed to be nullo gametes (22.17%; fig. 4b) and diplo-XY gametes (19.34%; fig. 4b). Other aneuploid classes resulting from MII NDJ were less frequent

Table 4a. Results of FISH cytological analysis in secondary spermatocytes and spermatids.

MI	**Class of Disjunction**	**XX/YY**	**X/XYY**	**XXY/X**	**XY/XY**	**XXYY/nullo**	**XY/X**	**X/X**
	% frequency	**22.8**	**0.99**	**4.95**	**69.3**	**0**	**0.99**	**0.99**

Table 4b.

MII	**Class of Nondisjunction**	**X**	**Y**	**Nullo**	**XX**	**YY**	**XY**	**XYY**	**XXY**
	% frequency	**22.64**	**21.23**	**22.17**	**4.72**	**7.08**	**19.34**	**1.89**	**0.94**

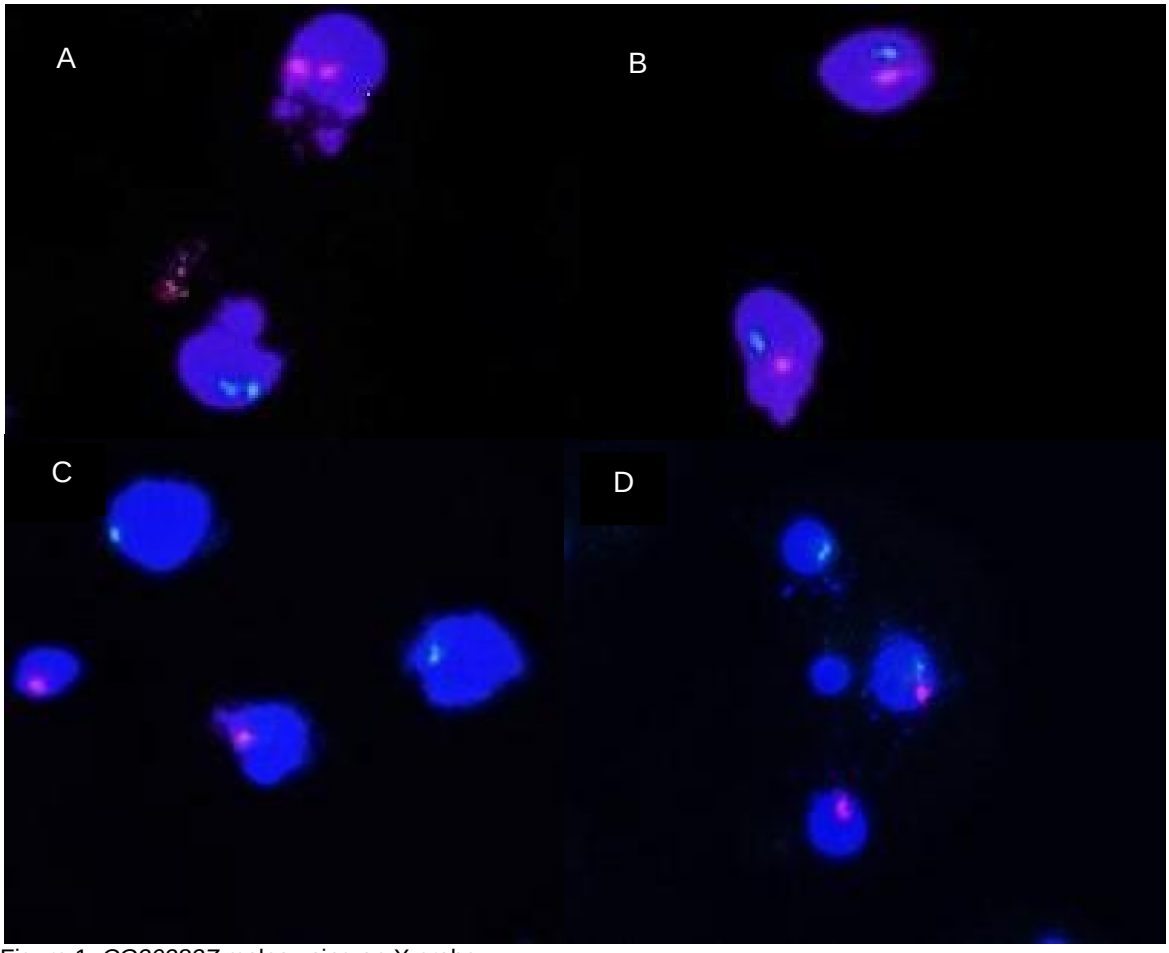
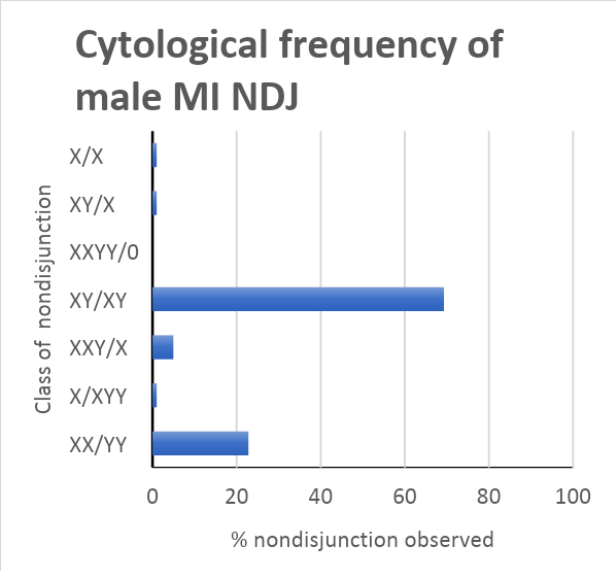


Figure 1. *CG30383Z* males using an X probe
(Red) and an AATAC repeat Y probe (Green). Nuclei are stained blue with DAPI **(**4′,6-diamidino-2-phenylindole). (A) Normal MI XY segregation resulting in secondary spermatocytes (B) exceptional secondary spermatocytes in which the sex chromosome sister chromatids have segregated equationally (C) normal MII resulting in spermatids (D) Aberrant MII division resulting in spermatid NDJ.



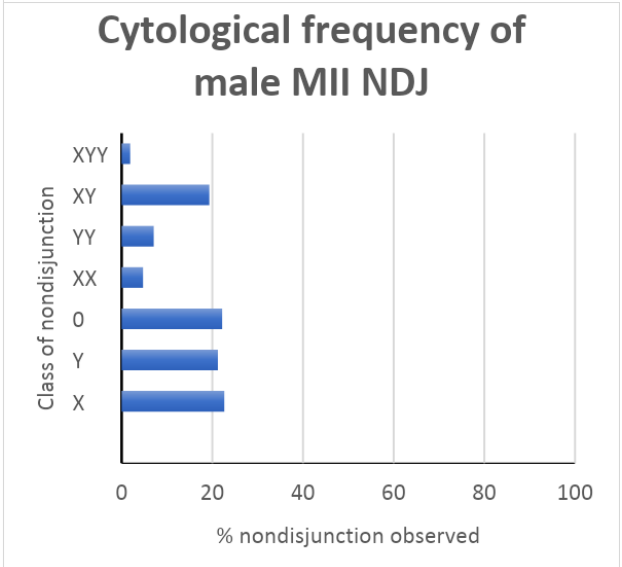


Figure 2. The frequency of nondisjunction (NDJ) is shown above and was cytologically observed from mutant CG30383^{Z} male testes using FISH. XX/YY is a normal division in MI resulting in normal secondary spermatocytes. X or Y is a normal division in MII that results in normal spermatids.

Discussion

The aim of this work was to understand one of the oldest issues in genetics: how sister chromatids co-orient during meiosis. Furthermore, I wanted to examine the *Drosophila*-specific mechanism responsible for moving sisters to the same pole in meiosis I. Two properties unique to meiosis but absent in mitosis are the attachment of sister chromatids to a single pole (monopolar attachment) and enduring centromere cohesion in the first meiotic division. To understand this process more clearly it is necessary to find the meiosis-specific proteins involved and discover their functions. CG30383^{Z} is a key player in mediating monopolar attachment of sister chromatid kinetochores at meiosis, thus ensuring proper chromosome segregation.

CG30383^{Z} mutants had characteristic nondisjunction in which sister chromatids no longer orient to the same pole but form amphitelic (bipolar) connections at the meiosis I spindle. Aberrant, amphitelic microtubule attachments to sister kinetochores resulted in precocious separation of sisters in the first meiotic division around 70 percent of the time. Precocious bi-directional segregation of sister chromatids in meiosis I indicates that CG30383^{Z} has an important role regarding this aspect. It is likely that CG30383^{Z} is a component of the fly monopolin complex and plays some role in mediating sister kinetochore orientation to ensure that paired sisters segregate together at the reductional division. Mutation in CG30383^{Z} led to aneuploidy error types (trisomy, monosomy) for both the autosomes and sex chromosomes in males and females suggesting that proper mono-orientation of sisters has a conserved mechanism for both sexes.

Searches based on sequence homology to budding yeast monopolin components, Csm1 and Lrs4, only find obvious homologs in other fungi (McCollum 2012). There is even low sequence homology for monopolin when compared to other fungal species, perhaps, the reason monopolin has not been discovered in many higher systems is because of poor conservation. Structural studies have proposed that the globular domain of Csm1 is likely to be evolutionarily related to Spc24/25 components of the NDC80 complex (Corbett and Harrison, 2012). This kinetochore complex is conserved in both fungi and animals and is necessary for proper kinetochore-microtubule binding and chromosome segregation. Thus, alternative structural homologs may exist in other organisms.

The CG30383^{Z} gene may be a species-specific component or mediator of sister kinetochore co-orientation in *Drosophila* that performs a similar role to that of monopolin in fungi. The similar phenotypic characteristics to monopolin that CG30383^{Z} presents suggest that this gene may be the fruit fly's analogous mechanism to properly orient chromosomes in the first meiotic division. Further research needs to elucidate the molecular structure of CG30383^{Z} to understand the mechanism by which *Drosophila* chromosomes are mono-oriented and to discover other possible elements in the fly's monopolin complex.

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