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Genetics

Haploidy and androgenesis in *Drosophila*

(NCD microtubule motor protein/pronuclear fusion)

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ABSTRACT **Androgenesis, development from paternal but not maternal chromosomes, can be induced to occur in some organisms, including vertebrates, but has only been reported to occur naturally in interspecific hybrids of the Sicilian stick insect. Androgenesis has not been described previously in *Drosophila*. We now report the recovery of androgenetic offspring from *Drosophila melanogaster* females mutant for a gene that affects an oocyte- and embryo-specific α-tubulin. The androgenetic exceptions are X,X diploid females that develop from haploid embryos and express paternal markers on all 4 chromosomes. The exceptional females arise by fusion of haploid cleavage nuclei or failure of newly replicated haploid chromosomes to segregate, rather than fusion of two inseminating sperm. The frequency of androgenetic offspring is greatly enhanced by a partial loss-of-function mutant of the NCD (nonclaret disjunctional) microtubule motor protein, suggesting that wild-type NCD functions in pronuclear fusion. Diploidization of haploid paternal chromosome complements results in complete genetic homozygosity, which could facilitate studies of gene variation and mutational load in populations.**

Diploid organisms usually arise by fusion of haploid maternal and paternal chromosome sets. In some instances, however, individuals develop from only maternal, or only paternal, chromosomes. The occurrence of diploid offspring with only maternal chromosomes, gynogenesis, has been reported in several organisms including *Drosophila*. In *Drosophila*, gynogenesis is associated with female- or male-sterile mutants that produce haploid embryos (1, 2). The best studied of these mutants are the maternal-effect mutant named "maternal haploid" (*mh*) and the paternal-effect mutant named "male sterile (3) of Hardy and Orevi" [*ms(3)K81*]. Both mutants are thought to cause failure of pronuclear fusion (2, 3), and both produce haploid embryos that express only maternal genetic markers. Diploid matroclinous offspring are produced at low frequency by matings of *ms(3)K81* males to wild-type females and are thought to arise by fusion of two of the haploid products of oocyte meiosis, usually the central two nuclei, or, less frequently, by doubling of a haploid cleavage nucleus (4).

Androgenesis has only been previously reported to occur naturally in interspecific hybrids of the Sicilian stick insect (5). These hybrids reproduce by eliminating the paternal species genome from oocytes, replacing it by fertilization with sperm from the paternal species (5, 6). Maintenance of the interspecific hybrids is by obligate backcrossing to the paternal species each generation, since hybrid males are sterile. Failure of the male and female pronuclei to fuse occurs frequently in hybrid embryos and can be followed by doubling of a single sperm or fusion of two sperm to produce diploid androgenetic offspring (5, 6). Androgenesis has also been induced in amphibia and fish species by irradiating oocytes to inactivate the germinal vesicle (7, 8) and applying hydrostatic pressure to suppress the first cleavage division of the fertilizing sperm (9, 10).

While the exceptional matroclinous offspring produced by *ms(3)K81* males provide evidence for gynogenesis in *Drosophila*, androgenesis has not been reported. We now report the recovery of androgenetic offspring from *Drosophila* females mutant for a gene, *αTub67C*, that results in an aberrant oocyte- and early-embryo-specific α-tubulin (11). We further find that androgenetic exceptions are produced at greatly enhanced frequency when the parental females are heterozygous for *αTub67C* and homozygous for ncd^D, a mutant allele of a gene (*ncd* for nonclaret disjunctional) that codes for a kinesin microtubule motor protein that functions in oocytes and early embryos (12).

MATERIALS AND METHODS

***Drosophila* Mutants.** Descriptions of mutants used in this work can be found in Lindsley and Zimm (13). The partial loss-of-function mutant, ncd^D, was originally isolated as a dominant mutant that caused elevated nondisjunction in females but now shows only recessive and weak semidominant effects on meiotic chromosome segregation (12). Although *ncd* null mutants cause frequent mitotic chromosome loss in early embryos in addition to high levels of meiotic nondisjunction and loss, ncd^D is nearly wild type for mitotic chromosome distribution in early embryos (12). The X chromosome and chromosome 2 in the ncd^D stock were replaced with Oregon R chromosomes several years ago by mating to balancer stocks. The *ncd* null alleles, ca^{nd}, and *ncd* were used in some of the experiments reported here.

The *αTub67C* mutants carrying alleles $\alpha Tub67C^1$, $\alpha Tub67C^2$, and $\alpha Tub67C^3$ (14) were obtained from the Bloomington Stock Center. The tip of chromosome 3R distal to *ca* in the three *αTub67C* mutant stocks was replaced by recombination with a wild-type (Oregon R) chromosome 3, and the X chromosome and chromosome 2 were replaced with Oregon R chromosomes by crossing to balancer lines. For *αTub67C* ncd^D chromosomes, ncd^D was crossed onto the original or recombinant *αTub67C* chromosomes 3, and the X chromosome and chromosome 2 were replaced with Oregon R chromosomes by matings to balancer lines, as for the *αTub67C* stocks. The lethal mutation associated with $\alpha Tub67C^3$ (14) was removed by the above recombination with the Oregon R chromosome 3. *Df(3L)AC1* is a deficiency, cytologically Df(3L)67A2;67D11-13, that uncovers *αTub67C* (14) and was used in some of the tests reported here.

Tests of meiotic and early mitotic chromosome segregation were carried out by mating wild-type (Oregon R) or mutant females to $y^2\ w^{bf}/B^SY$ males, and the offspring were analyzed phenotypically as described previously (12). In calculations of the total number of embryos produced by each mating, the number of X/X/Y females and X/0 males was multiplied by 2 to adjust for inviability of half of the nondisjunctional and chromosome loss embryos as X/X/X or 0/Y embryos.

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Abbreviation: DAPI, 4′,6-diamidino-2-phenylindole dihydrochloride.

Cytology. Embryos were collected for 30 min from wild-type females or 90 min from $\alpha Tub67C^3/+$ or $\alpha Tub67C^3\ ncd^D/ncd^D$ females, followed by dechorionation, fixation in methanol/EGTA without taxol, and removal of vitelline membranes as described (15). Mitotic spindles in early whole-mount embryos were visualized by staining embryos with a rhodamine-conjugated anti-α-tubulin antibody that cross-reacts with all α-tubulin isoforms (15, 16). Whole-mount embryos were also stained with the DNA-specific dye DAPI (4',6-diamidino-2-phenylindole dihydrochloride) to visualize chromosomes. Images of antibody-stained spindles were collected by using a Bio-Rad MRC600 scanning confocal system attached to a Zeiss Axiophot microscope with a ×63/1.4 n.a. Planapochromat objective.

To image chromosomes, whole-mount embryos stained with anti-tubulin antibody and DAPI were mounted in TE buffer (10 mM Tris·HCl, pH 7.9/1 mM EDTA) on Denhardt solution-treated slides (17) under siliconized coverslips; Denhardt solution is 0.02% bovine serum albumin/0.02% Ficoll/0.02% polyvinylpyrrolidone. Embryos were staged by examining spindles and chromosomes under fluorescence. Embryos were squashed *in situ* onto slides, coverslips were removed, and slides were post-fixed 30–60 min in cold absolute ethanol. Slides were wet-mounted in TE containing 5 μg of DAPI per ml, and chromosomes were photographed onto 4 × 5 inch (10.2 × 12.7 cm) Tri-X film. Negatives were scanned into digital images by using a Sharp JX-320 scanner. Confocal and light-micrograph image contrast was adjusted with Adobe PHOTOSHOP 2.5.1, and images were printed with a Tektronix Phaser IISDX printer.

RESULTS

Androgenesis was first detected in genetic tests for interactions between tubulin mutants and mutants of the NCD microtubule motor protein. Females heterozygous for $\alpha Tub67C^3$, a mutant allele encoding an oocyte- and early-embryo-specific α-tubulin (11, 14, 18), or for both $\alpha Tub67C^3$ and ncd^D, a partial loss-of-function microtubule motor protein mutant (12), were tested for X chromosome segregation by mating to $y^2\ w^{bf}/B^SY$ males. These tests resulted in the unexpected recovery of two exceptional, patroclinous yellow-2 white-buff females in addition to regular, phenotypically wild-type female offspring (Table 1). The yellow-2 white-buff females are not regular offspring of these crosses (Fig. 1) and were recovered at low frequency, 1 of 662 and 1 of 911 total embryos of $\alpha Tub67C^3/+$ and $\alpha Tub67C^3\ ncd^D/+$ females, respectively. Further tests showed that the exceptional patroclinous females were produced by females heterozygous for $\alpha Tub67C^3$ and homozygous for ncd^D (i.e., $\alpha Tub67C^3\ ncd^D/ncd^D$ females) at greatly increased frequency, ≈1 or 2 per 100 offspring, but were not produced by females wild type for $\alpha Tub67C$ (Table 1). The frequency of patroclinous females produced by $\alpha Tub67C^3\ ncd^D/ncd^D$ females was significantly increased compared with $\alpha Tub67C^3/+$ females.

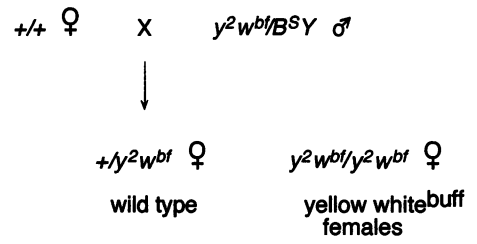

FIG. 1. yellow-2 white-buff ($y^2\ w^{bf}$) females are exceptional progeny that express recessive paternal markers. The schematic diagram shows regular, phenotypically wild-type and exceptional yellow-2 white-buff female offspring of the mating. Only female offspring and X and Y chromosome markers are shown. B^S, Bar of Stone mutant.

The exceptional patroclinous females were also produced at low frequency by $\alpha Tub67C/+$ females carrying either of the two other $\alpha Tub67C$ mutant alleles, $\alpha Tub67C^1$ or $\alpha Tub67C^2$, but only $\alpha Tub67C^1$ and $\alpha Tub67C^3$ caused a significant increase in frequency of the exceptional females in the presence of homozygous ncd^D (data not shown). Tests of $\alpha Tub67C^3/+$ females that were homozygous for two other *ncd* alleles, ca^{nd} or *ncd*, resulted in sterility. In addition, females homozygous for $\alpha Tub67C^2$ or $\alpha Tub67C^3$ and either wild type for *ncd* or homozygous for ncd^D were sterile. $\alpha Tub67C^1$ is associated with a lethal mutation and could not be tested in homozygous form; however, $\alpha Tub67C^1/\alpha Tub67C^3$ and $\alpha Tub67C^1$/deficiency (*Df*) females were tested and found to be sterile. Female sterility of heteroallelic $\alpha Tub67C$ combinations and $\alpha Tub67C$/deficiency has been reported previously (14).

The exceptional patroclinous females were recovered as larvae for karyotyping. Five yellow-2 female larvae that were examined showed normal X,X diploid karyotypes in neuroblast tissue (Fig. 2). Two further yellow-2 female larvae were diploid for the X chromosome and chromosomes 2 and 3 but were haploid for chromosome 4.

The yellow-2 white-buff adult females were of normal size and morphology, rather than the small size and irregular morphology expected for haploid offspring (19). Five of eight exceptional females that were tested in single-pair matings to $+/B^SY$ males for possible segregating mutations showed normal X chromosome segregation, indicative of diploidy. Three females were sterile, giving a higher-than-usual frequency of sterility. The yellow-2 white-buff females were concluded to be X,X diploid females that were homozygous for the paternal X chromosome.

To determine if the exceptional females were homozygous for paternal autosomes as well as the paternal X chromosome, $\alpha Tub67C^3\ ncd^D/ncd^D$ females were mated to males carrying recessive markers on the X chromosome and autosomes. The

Table 1. Androgenetic offspring are produced by females mutant for $\alpha Tub67C$

Female parent	Number of offspring with the indicated markers							Total embryos	Frequency of androgenetic females
	+ ♀	B^S ♀	B^S ♂	$y^2\ w^{bf}$ ♂	gyn	+ ♂	$y^2\ w^{bf}$ ♀		
+/+	725		604			1		1330	<0.001
$ncd^D/+$	699	2	607		2	1		1313	<0.001
$\alpha Tub67C^3/+$	373		271	3	8	3	1	662	0.002
$\alpha Tub67C^3\ ncd^D/+$	496	2	397		12	1	1	911	0.001
$\alpha Tub67C^3\ ncd^D/ncd^D$	285	78	243	54	12	1	12	817	0.015
ncd^D/ncd^D	765	13	551	18	2	1		1381	<0.001

+, Oregon R. The ncd^D and $\alpha Tub67C^3\ ncd^D$ chromosomes were marked with *st*. Females of the indicated genotypes were mated to $y^2\ w^{bf}/B^SY$ males, and offspring were scored for phenotype. The exceptional androgenetic $y^2\ w^{bf}$ females express recessive paternally derived X chromosome markers (Fig. 1) and are significantly increased in frequency among offspring of $\alpha Tub67C^3\ ncd^D/ncd^D$ females. Regular offspring are + females and B^S males. B^S (X/X/Y) females, $y^2\ w^{bf}$ (X/0) males, gynandromorphs (gyn), and + males arise upon X chromosome nondisjunction or loss in meiosis and mitosis (11). Minute (haplo-4) and Minute mosaic offspring were scored but were omitted from the table because of their highly variable recovery. The total number of embryos was calculated as described in *Materials and Methods*.

FIG. 2. Androgenetic females are X,X diploids. The chromosome spread prepared from the neuroblast of an androgenetic yellow-2 female larva shows the paired X chromosomes and autosomes typical of normal diploids. The X chromosomes are indicated. (DAPI; ×1070.)

mating produced 9 androgenetic females (yellow-2 white-buff cinnabar ebony sparkling) from an estimated 4259 embryos. All of the androgenetic females expressed recessive paternal markers on all four chromosomes (Table 2). The lower frequency of androgenetic exceptions produced by $\alpha Tub67C^3$ ncd^D/ncd^D females in this and other experiments involving matings to males with multiple chromosome markers is attributable to the multiply mutant constitution of the exceptional females. Together with the diploid chromosome number, the expression of recessive paternal markers on all four chromosomes indicates that the androgenetic females arise by doubling of paternal chromosome complements.

Whole-mount embryos were examined cytologically to determine the mutant effects of $\alpha Tub67C^3$ or both $\alpha Tub67C^3$ and ncd^D on early mitotic divisions. Early embryos produced by $\alpha Tub67C^3/+$ females sometimes contained two adjacent pronuclei or, more frequently, two side-by-side groups of chromosomes, one associated with an abnormally small mitotic spindle and the other with microtubules resembling a polar body ($n=3$, total=20). The two groups of chromosomes were interpreted to be the male and female chromosome complements. Approximately 70% of metaphase or anaphase cycle 1–12 embryos of $\alpha Tub67C^3/+$ females contained abnormally small mitotic spindles ($n=25$, total=35), and some embryos contained both small and normal-size spindles. The small spindles were also observed in embryos of $\alpha Tub67C^3/\alpha Tub67C^3$ females (Fig. 3) and have been reported previously to occur in embryos of $\alpha Tub67C^3$/deficiency females (14).

Small mitotic spindles were also observed in embryos of $\alpha Tub67C^3$ ncd^D/ncd^D females (Fig. 3), some of which contained both small and normal-sized spindles. The small spindles were formed around haploid chromosome complements, based on the diminished size of the spindles relative to wild type and the number of chromosomes associated with the small spindles in embryo squashes (Fig. 3). The presence of centrosomes at spindle poles indicated that the small spindles were assembled around paternal chromosomes, since centrosomes in *Drosophila* embryos are normally paternally-derived (20). More than one decondensing sperm could be observed in embryos mutant for $\alpha Tub67C^3$ or both $\alpha Tub67C^3$ and ncd^D, indicating the occurrence of polyspermy (insemination by more than one sperm).

Two possible mechanisms by which diploid androgenetic females could arise, fusion of two inseminating sperm or fusion of haploid cleavage nuclei, were tested by mating $\alpha Tub67C^3$ ncd^D/ncd^D females to males that were heterozygous for recessive markers [ebony (*e*), and roughoid (*ru*), and hairy (*h*)] on chromosome 3 (Table 3). Of the 17 androgenetic females that were recovered from an estimated 8323 embryos, 9 were ebony and 8 were roughoid hairy. Assuming random insemination by *ebony* and *roughoid hairy* sperm, diploidy due to fusion of two sperm nuclei is predicted to produce a ratio of 1:2:1 offspring with one, both, or the other chromosome 3 marker. The finding of androgenetic females expressing recessive markers on one or the other paternally derived chromosome 3, but not both, rules out fusion of two sperm nuclei as a mechanism of diploidization and indicates that the mechanism involves single haploid paternal chromosome complements.

The most probable mechanism is fusion of two haploid nuclei or, alternatively, failure of haploid chromosomes to form two separate nuclei after replication. Fusion of haploid cleavage nuclei or failure of haploid complements to disjoin would result in offspring homozygous for recessive chromosome 3 markers, consistent with the phenotypes of the androgenetic females that were recovered. The failure to recover androgenetic X,Y male offspring also supports a mechanism involving duplication of single haploid chromosomal complements.

Since fusion or nondisjunction of Y-bearing cleavage nuclei is expected to produce inviable Y,Y embryos, the frequency of androgenesis can be estimated to be twice the observed frequency of androgenetic females. The presence of recessive deleterious mutations in the tester male stocks almost certainly further reduces the observed frequency of androgenesis.

Gynogenetic exceptions that carry maternal, but not paternal, chromosomes are not produced at significant frequency by $\alpha Tub67C^3$ ncd^D/ncd^D females, as indicated by results of the experiment just described (Table 3). The parental females carried the recessive marker scarlet (*st*) on both chromosomes 3. Gynogenesis is expected to produce scarlet offspring. The absence of scarlet progeny among an estimated 8323 embryos indicates that gynogenesis is extremely infrequent (<0.00012) under these genetic conditions.

DISCUSSION

Several mutants of Drosophila are now known to produce haploid embryos. These mutants include *mh, ms(3)K81*, and a

Table 2. Androgenetic females express paternal markers on all four chromosomes

$\alpha Tub67C^3$ *st* ncd^D/*st* ncd^D ♀ × y^2 w^{bf}/B^SY; *cn; e; spa*^{pol} ♂							
Number of offspring with certain markers							
+ ♀	B^S ♀	B^S ♂	y^2 w^{bf} ♂	gyn	+ ♂	y^2 w^{bf} cn e spa ♀	Total embryos
1794	349	1291	216	31	4	9	4259

$\alpha Tub67C^3$ ncd^D/ncd^D females were mated to males with a recessively marked X chromosome and autosomes, and the offspring were scored for phenotype. The exceptional androgenetic y^2 w^{bf} cn e spa females express recessive paternally derived phenotypic markers on all four chromosomes. Regular offspring are + females and B^S males. The B^S (X/X/Y) females, y^2 w^{bf} (X/0) males, gynandromorphs (gyn), and + males arise as described in Table 1. Minute (haplo-4) and Minute mosaic offspring were scored but were omitted from the table, and the total number of embryos was calculated as described in *Materials and Methods*. The *cn* (cinnabar) marker can be recognized in a w^{bf} *cn* fly as a white eyecolor, whereas w^{bf} (white-buff) flies have buff (pale apricot-tan) eyecolor.

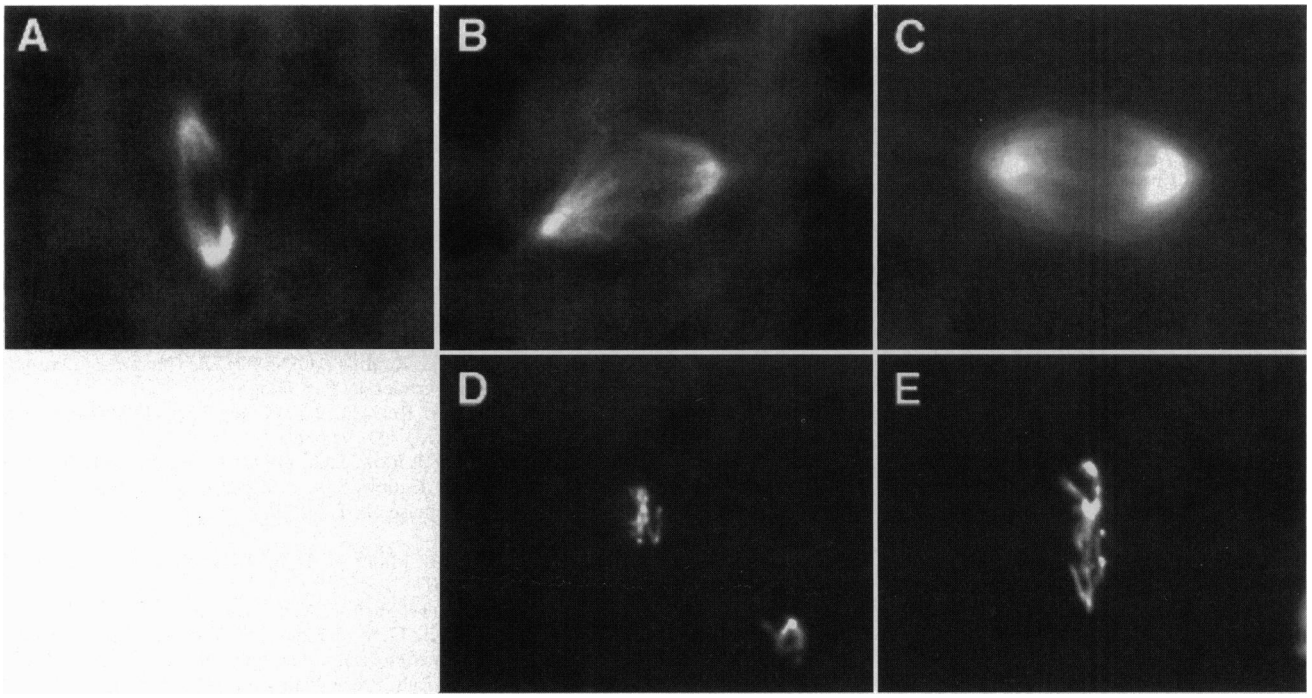

FIG. 3. Females mutant for *αTub67C* produce haploid embryos. (*A*) Small mitotic spindle in a cycle 4–5 $\alpha Tub67C^3/\alpha Tub67C^3$ mutant embryo. The embryo contained 12 small spindles like the one shown. Similar spindles were observed in embryos of $\alpha Tub67C^3/+$ females. (*B*) Small mitotic spindle in a cycle 4 $\alpha Tub67C^3\ ncd^D/ncd^D$ mutant embryo. (*C*) Normal-size cycle 4 mitotic spindle in a wild-type embryo. (*D*) Haploid set of chromosomes in a cycle 6–7 $\alpha Tub67C^3\ ncd^D/ncd^D$ mutant embryo. The chromosomes were associated with a small mitotic spindle like the one shown in *B*. The embryo contained ≈45 spindles, approximately half of which were normal-size and half abnormally small. A second haploid set of chromosomes is at the lower right. (*E*) Diploid set of chromosomes associated with a normal-size spindle in a cycle 6 wild-type embryo. (*A–C*) Confocal images of spindles (rhodamine-conjugated anti-tubulin antibody; ×2470.) (*D* and *E*) Light micrographs of chromosomes in embryo squashes. (DAPI; ×1175.)

few other female-sterile mutants that have not been characterized further (1). Haploid embryos are also produced by females mutant for a gene, *αTub67C*, that affects an oocyte- and early-embryo-specific α-tubulin of *Drosophila* (Fig. 3). Two of the mutants that produce haploid embryos, *mh* and *ms(3)K81*, are defective in pronuclear fusion: the female and male pronuclei fail to conjugate in embryos of *mh* females (3), while *ms(3)K81* is thought to result in defective pronuclear fusion based on the occurrence of haploid embryos. The oocyte meiotic divisions do not occur in embryos of *αTub67C*/deficiency females (14), and the female pronucleus therefore does not form. In embryos of $\alpha Tub67C^3/+$ females, the meiotic divisions occur, but the female pronucleus is greatly delayed or defective in forming: two side-by-side groups of chromosomes were frequently observed, one with a small mitotic spindle and one with a polar body-like array of microtubules, interpreted to be the male and female chromosome complements.

After failure of the oocyte pronucleus to fuse with the male pronucleus or to form, haploid embryos arise upon division of the functional pronucleus. The haploid embryos produced by *mh* and *ms(3)K81* are gynogenetic, arising from divisions of the female, but not the male, pronucleus. In *αTub67C* mutants, the paternal chromosomes divide to produce haploid androgenetic embryos.

Haploid nuclei in gynogenetic or androgenetic oocytes or embryos can fuse or nondisjoin, producing diploid embryos that develop into gynogenetic or androgenetic adults. The frequency of fusion or nondisjunction of haploid nuclei is enhanced in embryos mutant for both $\alpha Tub67C^3$ and ncd^D, compared with embryos mutant only for $\alpha Tub67C^3$. Although the basis of the enhanced frequency of androgenesis in $\alpha Tub67C^3$ and ncd^D doubly mutant embryos is not certain, the NCD^D motor might increase the frequency of androgenesis by moving haploid nuclei together for fusion in early embryos. NCD is a member of an unusual subfamily of kinesin motor proteins that moves toward microtubule minus, rather than plus, ends (21). NCD^D could function in moving haploid nuclei together for fusion in the same manner as another minus-end kinesin motor, yeast Kar3 (22), which mediates nuclear fusion during mating. Kar3 has been proposed to crosslink antiparallel microtubules that lie between the two haploid nuclei of

Table 3. Androgenetic females arise by fusion or nondisjunction of haploid cleavage nuclei

$\alpha Tub67C^3\ st\ ncd^D/st\ ncd^D$ ♀ × $y^2\ w^{bf}/B^SY;\ e/ru\ h$ ♂									
Number of offspring with indicated markers									
						$y^2\ w^{bf}$ ♀			
+ ♀	B^S ♀	B^S ♂	$y^2\ w^{bf}$ ♂	gyn	+ ♂	e	$e^+\ ru^+\ h^+$	ru h	Total embryos
3309	598	2858	456	23	8	9	0	8	8323

$\alpha Tub67C^3\ ncd^D/ncd^D$ females were mated to males heterozygous for recessive chromosome 3 markers, and offspring were scored for phenotype. The exceptional androgenetic $y^2\ w^{bf}$ females were either e or ru h, but not $e^+\ ru^+\ h^+$. Regular offspring are + females and B^S males. The B^S (X/X/Y) females, $y^2\ w^{bf}$ (X/0) males, gynandromorphs (gyn), and + males arise as described in Table 1. Minute (haplo-4) and Minute mosaic offspring were scored but are omitted from the table, and the total number of embryos was calculated as described in *Materials and Methods*.

mating cells and move toward microtubule minus ends, pulling the nuclei together (22). If NCD^D acts to enhance androgenesis by moving nuclei together for fusion, it could reflect wild-type NCD function in pronuclear conjugation during karyogamy in *Drosophila*, paralleling the role of Kar3 in mating yeast cells. Pronuclear fusion in *Drosophila* is likely to require microtubules (20), but the involvement of a microtubule motor protein has not been demonstrated.

The single gene mutations, *mh*, *ms(3)K81*, and *αTub67C*, cause the formation of haploid embryos, and a small but significant fraction of the haploid embryos produced by matings of *ms(3)K81* males or *αTub67C/+* females becomes diploid and develops into adults. In contrast to the gynogenesis caused by *ms(3)K81*, which usually involves fusion of haploid products of oocyte meiosis, androgenesis in *Drosophila* results in complete genetic homozygosity, since it involves duplication of single haploid chromosome complements. This should be useful for constructing isogenic stocks or making specific markers homozygous and could be applied to studies of genetic variation and mutational load in wild-type and mutant populations. Androgenesis in *Drosophila* represents a special case of thelitoky, parthenogenesis resulting in only female offspring, and also has implications for genome transmission and the evolution of reproductive mechanisms.

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