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To RACHEL DEYSDALE	From KEVIN COOK	
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March 19, 1997

Dear Kathy,

Enclosed is a set of stocks containing new mutants on chromosome 2 and 3 that Jim Fristrom wrote to you about in February. Our paper describing all of these mutants will come out in the May 1997 issue of Genetics (Prout, et al. - pages not known yet). Wing clones homozygous for any of these mutations give a blistered phenotype. Some of the new loci have been given names (after flightless birds). Others are called 2L-A etc..for the chromosome arm to which they map. For all of them, the specific allele is given in superscript. Ostrich and 3L-B have a dominant wing blister phenotype. 2R-F, 3L-B and 3R-B are homozygous viable, but the stocks included here contain both homozygotes and heterozygotes for the balancer listed. There are 5 new loci on 2L, 10 on 2R, 2 on 3L, and 4 on 3R. One allele for each is enclosed, so there should be a total of 21 vials. The labels on the vials have some abbreviations. Below is a complete list with the full information about markers contained in each stock:

CyO Beta E3

2253 y w; P{white-un1}30C ***2L-A^{2L-2}*** P{neoFRT}40A / CyO P{mini-w$^+$ act-lacZ} P{w^{+mC} = act-lacZ}

e.g. the above would be labeled: yw; 2L-A^{2L-2} w$^+$ / CyO

Cst

2258 yw; P{white-un1}30C ***Ostrich^{2L-32}*** P{neoFRT}40A / CyO P{mini-w$^+$ act-lacZ}

cas

2269 yw; P{white-un1}30C ***cassowary^{2L-5}*** P{neoFRT}40A / CyO P{mini-w$^+$ act-lacZ}

pen

2271 yw; P{white-un1}30C ***penquin^{2L-14}*** P{neoFRT}40A / CyO P{mini-w$^+$ act-lacZ}

2273 yw; P{white-un1}30C ***2L-F^{2L-31}*** P{neoFRT}40A / CyO P{mini-w$^+$ act-lacZ}

2274 yw; P{neoFRT}42D ***kiwi^{2R-3}*** P{white-un1}47A / CyO P{mini-w$^+$ act-lacZ}

2276 yw; P{neoFRT}42D ***moa^{2R-6}*** P{white-un1}47A / CyO P{mini-w$^+$ act-lacZ}

pio

2278 yw; P{neoFRT}42D ***piopio^{2R-16}*** P{white-un1}47A / CyO P{mini-w$^+$ act-lacZ}

2279 yw; P{neoFRT}42D ***auk^{2R-4}*** P{white-un1}47A / CyO P{mini-w$^+$ act-lacZ}

2280 yw; P{neoFRT}42D ***2R-F^{2R-22}*** P{white-un1}47A / CyO P{mini-w$^+$ act-lacZ}

kit

2281 yw; P{neoFRT}42D ***kitikete^{2R-17}*** P{white-un1}47A / CyO P{mini-w$^+$ act-lacZ}

kak

2282 yw; P{neoFRT}42D ***kakapo$^{65-2}$*** P{white-un1}47A / CyO P{mini-w$^+$ act-lacZ}

xen

2290 yw; P{neoFRT}42D ***xenecid$^{72-3}$*** P{white-un1}47A / CyO P{mini-w$^+$ act-lacZ}

tak

2292 yw; P{neoFRT}42D ***takahe$^{82-8}$*** P{white-un1}47A / CyO P{mini-w$^+$ act-lacZ}

2295 yw; P{neoFRT}42D ***2R-L$^{84-2}$*** P{white-un1}47A / CyO P{mini-w$^+$ act-lacZ}

2296 yw; P{white-un1}70C ***rhea^{1}*** P{neoFRT}80B / TM6B, Tb Hu P{mini-w$^+$ Abd-A-βgal}

Presumably P{w^{+mC} = iab-2(1.7)lacZ}

2300 yw; ***3L-B^{3L-27}*** P{neoFRT}80B / TM6B, Tb Hu P{mini-w$^+$ Abd-A-βgal}

2301 yw; P{neoFRT}82B ***struthio^{3R-3}*** P{white-un1}90E / TM6B, Tb Hu P{mini-w$^+$ Abd-A-βgal}

2302 yw; P{neoFRT}82B ***steamer duck^{3R-17}*** P{white-un1}90E / TM6B, Tb Hu P{mini-w$^+$ Abd-A-βgal}

2305 +; P{neoFRT}82B ***3R-B^{3R-13}*** P{white-un1}90E / TM6B, Tb Hu P{mini-w$^+$ Abd-A-βgal}

2306 +; P{neoFRT}82B ***3R-C^{3R-11}*** P{white-un1}90E / TM6B, Tb Hu P{mini-w$^+$ Abd-A-βgal}

Most are eclosing now, a few were just set up in the last few days. I hope they arrive in good shape. Let me know if you have any questions or need any other information. Thanks.

Sincerely,

Mary

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P.S. I've also enclosed a table from the paper with some more information, in case you need it.

TABLE 1

Properties of newly isolated mutants

Arm	Name		Alleles	Description[a]	Blister[b]	Lethal phase	Non-comp. deficiency	Interactions: *mys*^{1}/*mys*^{2}	Interactions: *mys*^{1}/*mys*^{1}
2L	*2L-A*		1	r, hl	A	Larva	See text	Moderate	None
	2L-F		1	r, hl	B	Pupa	None	None	None
	cassowary	*cas*	5	r, hl	A	Pupa	None	None	None
	dumpy		9	r, hl	A	Embryo/larva	25A1, 4	None	None
	Ostrich	*Ost*	2	D, hl	A[c]	Pupa	None	None	None
	penguin	*pen*	6	r, hl	A	Larva	None	None	None
2R	*2R-F*		1	r, hv	A[c]	Adult	None	Weak	None
	2R-L		1	r, hl	A	Larva	None	Weak	None
	auk		3	r, hl	C	Pupa	*Df(2R)trix*, 51A1, 2-51B6	Weak	None
	blistered		2	D, hl	D	Larva	*Df(2R)Px4*, 60B-C	Strong	Strong
	kakapo	*kak*	5	r, hl	A	Early larva	*Df(2R)CX1*, 49C1,4-50C23,D2	Weak	None
	kitikete	*kit*	3	r, hl	A	Larva	*Df(2R)Np3*, 44D2,E1-45B8, C1	Weak	None
	kiwi		17	r, hl	A	Embryo/larva	None	Weak	None
	mam		2	r, hl	D	Embryo	*Df(2R)CX1*, 49C1,4-50C23, D2	Weak	None
	moa		4	r, hl	A	Early larva	*Df(2R)_AB*, 59A1,3-B1,2	None	None
	piopio	*pio*	2	r, hl	C	Embryo	*Df(2R)Px2*, 60C5, 6-D9, 10	Weak	None
	takahe	*tak*	1	r, hl	A	Pupa	*Df(2R)bw-S46*, 59D8,11-60A7	None	None
	xenicid	*xen*	1	r, hl	A	Early larva	*Df(2R)03072*, 51A-51C	None	None
3L	*3L-B*		1	D, hl	*d*	Adult ND	ND	None	None
	rhea		2	r, hl	A	Embryo	*Df(3L)WR10*, 75A6,7-75C1,2	Strong	Strong
3R	*3R-B*		1	r, hv	B[c]	Adult	ND	None	None
	3R-C		1	r, hl	D	Pupa	ND	None	None
	Delta		3	D, hl	D	Embryo	91D-92A	Weak	Weak
	steamerduck	*stk*	2	r, hl	A	Early larva	*Df(3R)p712*, 84D4,6-85B6	Weak	Weak
	struthio	*Stru*	1	r, hl	A	Embryo	*Df(3R)e-N19*, 93B-94	None	None

ND, not determined.

[a] r, recessive; D, dominant; hl, homozygous lethal; hv, homozygous viable.

[b] Wing blister phenotypic classes are described in the text.

[c] Homozygotes have fluid filled or collapsed wings.

Heterozygotes have small wings with blisters.

[d] Blister phenotype determined in homozygotes (*3L-B*) or heterozygotes (*Ost*).

deficiency. As described below, the efficiency of recovery of mutations and the number of mutations recovered varied greatly among the four chromosome arms.

2L: Twenty-four independent mutations in six complementation groups were established from screens of 40,888 chromosomes. The efficiency of recovery was one established mutation per 1704 screened chromosomes. Four of the six complementation groups are composed of two or more alleles [*cassowary* (*cas*), *dp*, *Ostrich* (*Ost*), *penguin* (*pen*)]. All the new *dumpy* alleles recovered are lethal and include examples of dp^{l}, dp^{lw} and dp^{olu} mutations (CARLSON 1959). *Ost* alleles cause dominant blisters but are lethal in homozygous and heteroallelic combination. We have been unable to identify a deficiency on *2L* or *2R* that does not complement *Ost*, and so have not excluded the possibility that *Ost* is located on *2R*. A single mutant chromosome defining one complementation group (*2L-A*) is not complemented by a chromosome deficient for 21A1-21B6,7 (*Df(2L)net-PM47C*) and two chromosomes deficient for 28B2-28D1 (*Df(2L)XE2750; Df(2L)XE3801*). Whether the *2L-A* chromosome carries an inversion with breakpoints in the 21A-B6,7 and 28B2-D1 regions, or two independent lethals has not been established. The *2L-F* mutation complements for viability a chromosome deficient for 38A6,B1-40A4,B1 (*Df(2L)TW161*). The heterozygous adults, however, exhibit the held-out wing phenotype.

2R: Forty-one independent mutations in 12 complementation groups were established from screens of 16,969 chromosomes. Another mutation defining another complementation group was initially established but subsequently lost. The efficiency of recovery was one established mutation per 414 screened chromosomes. Eight of the 12 complementation groups are composed of two or more alleles [*auk*, *bs*, *kakapo* (*kak*), *kitikete* (*kit*), *kiwi*, *mam*, *moa* and *piopio* (*pio*)]. Two complementation groups, *xenicid* (*xen*) and *takahe* (*tak*), are each identified, respectively, by a single mutation that fails to be complemented by a single deficiency (Table 1).

3L: Three independent mutations in two comple-