Autosomal genetic maps of the Australian Sheep Blowfly, *Lucilia cuprina dorsalis* R.-D. (Diptera: Calliphoridae), and possible correlations with the linkage maps of *Musca domestica* L. and *Drosophila melanogaster* (Mg.)

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SUMMARY

Linkage data and revised maps for 52 autosomal loci in *L. cuprina* are presented. Examination of the linkage relationships of biochemically and morphologically similar mutations in *L. cuprina*, *Musca domestica* L. and *Drosophila melanogaster* (Mg) suggests that the major linkage groups have survived largely intact during the evolution of the higher Diptera.

1. INTRODUCTION

For several years the CSIRO Division of Entomology has conducted a major genetical and ecological programme aimed at the development and testing of genetic methods of controlling *Lucilia cuprina dorsalis* R.-D., the most important insect pest of sheep in Australia (Whitten *et al.* 1977 for a recent review). Basic to any genetic control programme is a thorough knowledge of the genetics of the species concerned; accordingly we have devoted considerable research effort to the elucidation of the formal genetics and cytogenetics of *L. cuprina*.

In this paper we present data concerning the linkage relationships of mutations at 52 loci in this species, give revised genetic maps of the five autosomal linkage groups, and discuss similarities between the linkage groups of *L. cuprina*, *Musca domestica* L. and *Drosophila melanogaster* (Mg.) Cytological maps of the polytene chromosomes of *L. cuprina*, and genetic data correlating these maps with the linkage maps, are presented elsewhere (Foster *et al.* 1980).

2. MATERIALS AND METHODS

Mutations. The symbols and names of the mutations mentioned in the present report are listed in Table 1. Descriptions of most of these mutations are presented by Whitten *et al.* (1975). Full descriptions of all known genetic mutations of *L. cuprina* are being prepared and will be published elsewhere.

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Table 1. *Symbols and names of* L. cuprina *mutations cited in the present paper. Mutations not listed by Whitten* et al. (*1975*) *are described briefly in parentheses. Mutations which have not been mapped are indicated by an asterisk following the mutant symbol*

Linkage group	Symbol	Name
2	bp, bp^2	Black puparium, dark puparium (alleles)
	$frag$	Fragmented veins (wing venation altered; certain veins missing, other veins fused)
	gla	Glazed eyes
	Jet	Jet wings (wings short and misshapen; humeral crossvein absent; associated with translocation $T(2;5)Jet$)
	Luc^*	Lucilins (12 loci associated with larval storage protein synthesis)
	ms	Missing bristles
	pb	Purple body
	pp	Pale puparium (puparium straw-coloured; smaller than wild type; viability poor)
	pt	Purple thorax
	sa	Sabre wings
	sb	Stubble bristles
	Spt	Spatule wings (wings small and distorted; variable in heterozygotes; homozygous viable and fertile)
3	ar	Arista
	drm	Double radial–median crossvein
	f	Forked bristles
	ru	Rusty body
	sbd	Stubbloid bristles
	w	White eyes
	wy	Wavy wings
	yw	Yellowish eyes
4	bu	Bubble wings
	cy	Curly wings
	gl	Golden body
	gp	Grape eyes
	hk	Hooked bristles
	ra	Radial vein gaps
	re	Reduced eyes
	Rop-1	Diazinon resistance
	Sh	Short bristles
	sv	Singed vibrissae
	tg	Tangerine eyes
	thv	Thick veins
5	bz	Bronze body
	cu	Curled wings
	dy	Dumpy wings (wings short and broad, often with thick veins; posterior margin of wing indented)
	Est^*	Esterase (electrophoretic variants)
	Jet	Jet wings (see linkage group 2)
	$mv(=m_1)$	Ml veinless
	ol	Olive body
	Rdl	Dieldrin resistance
	sby	Stubby bristles
	sk	Sockets
	to, to^2	Topaz eyes, topaz² eyes (alleles)

Table 1 (*cont.*)

Linkage group	Symbol	Name
6	*Amy**	Amylase (electrophoretic variants)
	Bl	Bristle
	dfw	Deformed wings
	ho	Held-out wings
	Rop-2	Diazinon resistance
	spr	Spread wings (wings held at right-angles to body and translucent rather than transparent)
	st	Stumpy bristles
	ti	Tiny bristles
	tri	Triangular veins (thickening of certain vein junctions)
	Tw	Twisted bristles
	vg	Vestigial wings
	y	Yellow eyes

Rearing methods. Ambient temperatures, at which larvae were reared and adult flies maintained, were generally within the range 25–30 °C. Each female was allowed to oviposit on a cube of sheep liver in a 12×50 mm vial. Egg masses (up to 300 eggs, depending on size and nutritional status of females (Foster *et al.* 1975; Barton Browne, van Gerwen & Williams, 1979)) were generally left in the oviposition vials overnight to hatch. On overage at 100 % R.H., eggs take 12·3 h to hatch at 25 °C and 9·3 h at 30 °C (Vogt & Woodburn, 1980).

After hatching, larvae were placed on sheep liver on a layer of vermiculite in a ventilated container which was screened with fine wire mesh to keep out parasites and larvae from other cultures. After 4–6 days the fully fed larvae left the liver and pupated in the vermiculite. Before the end of the pupal stage, which lasts 6–8 days, pupae were removed from the vermiculite and placed in cages supplied with sugar and water.

Virgin females were usually obtained reliably if isolated while less than 24 h old, although occasional matings involving younger females have been observed. If given unlimited access to protein (sheep liver as a paste or in strips) females can mature their first batch of eggs 4·5 days at 25 °C after emergence (Woodburn, Vogt & Kitching, 1978). In practice, eggs were usually obtained from 6–8-day-old females which had been fed fresh liver paste on 2 consecutive days after their second day of life.

Genetic mapping. The genetic mapping data were obtained during the period 1968–1979 from test crosses of heterozygous females to males homozygous for the relevant recessive mutations. The standard procedure used in mapping was to rear broods of progeny from individual females, score the broods separately, and pool the results of those broods in which there was no evidence of non-virginity, disproportionately poor expression of one or more mutations, or other anomaly. In some crosses there was evidence of incomplete penetrance of certain markers. In these cases only flies demonstrably mutant for the marker concerned were used to compute genetic map distances. Such crosses are identified in the Results section. Progeny from crosses involving insecticide resistance mutations were treated by topical application of the insecticide to adults and scored as described by Arnold & Whitten (1976) for *Rop-1* and *Rop-2*, and by Foster *et al.* (1978) for *Rdl*.

3. RESULTS

Chromosome 2 genetic mapping. The results of the chromosome 2 mapping crosses are presented in Table 2. The positions of *sb*, *pt* and *frag* relative to *pb* and *bp* were determined by progeny-testing recombinants between *pb* and *bp²* against *sb*, *pt* or *frag* respectively. In a cross of $pb + bp/ + sb + ♀♀ \times pb\ bp\ bp ♂♂$ 9 out of 35 *pb bp⁺* and 22 out of 28 *pb⁺ bp* recombinant chromosomes tested carried *sb*. In a cross of $pb + bp/ + pt + ♀♀ \times pb\ bp\ bp\ ♂♂$, 2 out of 24 *pb⁺ bp* and 29 out of 32 *pb⁺ bp* recombinant chromosomes tested carried *pt*. In a cross of $pb + bp/ + frag + ♀♀ \times pb\ bp ♂♂$, 4 out of 15 *pb bp⁺* and 12 out of 17 *pb⁺ bp* recombinants tested carried *frag*. These results indicate that all three mutants lie between *pb* and *bp*, and suggest the following map distances: *pb*–(6)–*frag*–(15)–*bp*. A composite genetic map of chromosome 2 is presented in Fig. 1 (*a*).

Chromosome 3 genetic mapping. The results of the chromosome 3 mapping crosses are presented in Table 3, and the composite map derived from these data in Fig. 1 (*b*). The data in Table 3 do not critically establish that *f* and *drm* lie to the left of

Table 2. *Results of mapping crosses with chromosome 2 mutations*

♀ Genotype	N	Number and type of single crossovers[1]			Number and type of double crossovers			Number of triple crossovers
		1	2	3	1, 2	1, 3	2, 3	
pb bp² gla/+ + +	798	88	281	–	62	–	–	–
pb + /+ bp²	884	174	–	–	–	–	–	–
bp² ms gla/+ + +	555	65	144	–	2	–	–	–
bp² +/+ ms	1128	151	–	–	–	–	–	–
sa +/+ bp²	615	112	–	–	–	–	–	–
pb sa bp²/+ + +	770[2]	41	122	–	0	–	–	–
pb sb/+ +	845	139	–	–	–	–	–	–
sb bp²/+ +	224	11	–	–	–	–	–	–
	699	6	–	–	–	–	–	–
sb +/+ bp²	173	7	–	–	–	–	–	–
	561	1	–	–	–	–	–	–
sb +/+ bp	1061	8	–	–	–	–	–	–
pt bp²/+ +	1574	36	–	–	–	–	–	–
pt +/+ bp²	827	16	–	–	–	–	–	–
pb pt/+ +	715	139	–	–	–	–	–	–
pt pp gla/+ + +	1036	145	298	–	15	–	–	–
Spt +/+ bp	727	0	–	–	–	–	–	–
pb + bp² gla/+ Spt + +	869	83	1	287	0	54	0	–
sb+ bp/+ Spt +	544	8	3	–	0	–	–	–
pb bp²+gla/+ +Jet +	863	147	28	176	11	56	4	2
	247	41	5	46	2	18	0	0

[1] Crossover regions are identified by number according to the following examples. In the *pb bp² gla/+ + + cross*, region 1 is *pb – bp²* and region 2 is *bp² – gla*. In the *pb + bp² gla/+ Spt + +* cross, region 1 is *pb – Spt*, region 2 is *Spt – bp²* and region 3 is *bp² – gla*. In the latter cross, simultaneous crossing-over in regions 1 and 2 would be indicated as double crossovers 1, 2 (and so forth).

[2] Only *sa* mutant flies were used for genetic map purposes, due to incomplete penetrance of this mutation in this cross.

Table 3. *Results of mapping crosses with chromosome 3 mutations*

♀ genotype	N	Number and Type of Single crossovers[1]			Number and type of double crossovers			Number of triple crossovers
		1	2	3	1, 2	1, 3	2, 3	
w ru f drm/+ + + +	302[2]	43	20	10	0	0	0	0
	376	60	23	21	0	0	0	0
w + drm/+ ru +	615	93	43	–	0	–	–	–
w ru drm/+ + +	814	136	64	–	0	–	–	–
w ru ar/+ + +	1418	217	387	–	35	–	–	–
w + ar/+ ru +	1413	163	479	–	19	–	–	–
w ru ar wy/+ + + +	819	70	144	173	13	40	74	5
w ru ar sbd/+ + + +	862	63	159	192	2	50	74	1
ru ar yw wy/+ + + +	238	40	12	81	0	22	6	1
ru yw wy/+ + +	885	206	207	–	137	–	–	–
w sbd wy/+ + +	390[3]	201	6	–	7	–	–	–

[1] See footnote (1), Table 2.

[2] In all crosses involving *drm* only *drm* mutant flies were used, because of incomplete penetrance of this marker.

[3] Only *sbd* mutant flies were used in this cross, because of incomplete penetrance of this marker.

Table 4. *Results of mapping crosses with chromosome 4 mutations*

♀ Genotype	N	Number and type of single crossovers[1]				Number and type of double crossovers			Number of triple crossovers
		1	2	3	4	1, 2	1, 3	2, 3	
sv bu + ra tg/+ + *Rop-1 + +*	296[2]	56	13	45	64	–[3]	–	–	–
sv + /+ bu	395	68	–	–	–	–	–	–	–
sv bu tg/+ + +	447	56	151	–	–	11	–	–	–
sv ra tg gl/+ + + +	581	93	57	127	–	25	74	44	22
cy tg Sh/+ + +	229	46	3	–	–	0	–	–	–
tg Sh gl/+ + +	1446	13	653	–	–	4	–	–	–
tg + gl/+ Sh +	703	9	304	–	–	2	–	–	–
tg + +/+ Sh gl	389	0	158	–	–	0	–	–	–
re Sh/+ +	819	69	–	–	–	–	–	–	–
re gl gp/+ + +	703	313	30	–	–	9	–	–	–
Rop-1 + / + re	365	102	–	–	–	–	–	–	–
Sh + / + thv	725	130	–	–	–	–	–	–	–
thv gl gp/+ + +	174	32	5	–	–	0	–	–	–
Sh gl gp/+ + +	714	319	22	–	–	2	–	–	–
gl gp/+ +	529	20	–	–	–	–	–	–	–
gl hk/+ +	551	42	–	–	–	–	–	–	–
Sh thv gl gp/ *+ + + +*	1164	169	285	66	–	7	4	2	0
Sh + + + + / + *thv gl gp*	198	34	50	11	–	2	5	0	0

[1] See footnote (1), Table 2.   [2] Unpublished data of P. Hughes.

[3] Multiple crossovers excluded– see text for details.

Table 5. *Results of mapping crosses with chromosome 5 mutations*

♀ Genotype	N	Number and type of single crossovers[1]				Number and type of double crossovers						Number of triple crossovers
		1	2	3	4	1,2	1,3	2,3	1,4	2,4	3,4	
Rdl + + + + +/+ to² bz mv sby	554	40	84	18	97	5	4	0	37	60	7	6²
to² bz mv sk/+ + + + +	434	140	32	4	—	3	2	0	—	—	—	0
to bz mv/+ + +	682	203	24	—	—	0	—	—	—	—	—	—
bz + +/+ mv sk	393	23	12	—	—	0	—	—	—	—	—	—
bz mv sk/+ + +	765	42	5	—	—	1	—	—	—	—	—	—
	83	13	11	—	—	0	—	—	—	—	—	—
mv sby/+ +	802	312	—	—	—	—	—	—	—	—	—	—
mv sk/+ +	848	69	—	—	—	—	—	—	—	—	—	—
ol mv sk/+ + +	681	101	30	—	—	5	—	—	—	—	—	—
ol + /+ mv	713	99	—	—	—	—	—	—	—	—	—	—
	1681	280	—	—	—	—	—	—	—	—	—	—
mv + sk/+ cu +	46	1	8	—	—	0	—	—	—	—	—	—
to dy mv/+ + + +	957	257	93	—	—	2	—	—	—	—	—	—
dy mv/+ +	790	62	—	—	—	—	—	—	—	—	—	—
Jet+ + /+ to cu	785³	108	227	—	—	34	—	—	—	—	—	—
to cu/+ +	382	140	—	—	—	—	—	—	—	—	—	—

[1] See footnote (1), Table 2.

[2] Triple crossovers: 1(1,3,4); 4(1,2,4). Quadruple crossover: 1.

[3] Results from a cross in which females were egged *en masse* rather than singly.

ar. However, cytogenetic studies (Foster *et al.* 1980) confirm this gene order for these mutations.

Chromosome 4 genetic mapping. The results of the chromosome *4* mapping crosses are presented in Table 4, and the composite map derived from these data in Fig. 1(*c*). A total of 40 apparent multiple crossovers from the *sv bu + ra tg/+ + Rop-1 + +* females were excluded from the analysis, since the data strongly suggested that penetrance of *bu* and *ra* was incomplete and that the dose of insecticide used did not absolutely discriminate between resistant and susceptible. All three factors would lead to misclassification of certain non-, single- and double-crossover

Table 6. *Results of mapping crosses with chromosome 6 mutations*

♀ Genotype	N	Number and type of single crossovers[1]			Number and type of double crossovers			Number of triple crossovers
		1	2	3	1,2	1,3	2,3	
vg + y/+ Bl +	829	212	122	–	3	–	–	–
Bl +/+ y	417	65	–	–	–	–	–	–
Bl +/+ ho	756	203	–	–	–	–	–	–
y +/+ ho	388	31	–	–	–	–	–	–
vg y +/+ + Tw	649	253	57	–	9	–	–	–
	729	308	73	–	21	–	–	–
y +/+ Tw	629	54	–	–	–	–	–	–
y + dfw/+ Tw +	95[2]	4	11	–	0	–	–	–
y dfw +/+ Rop-2	305[3,4]	47	36	–	5	–	–	–
y + dfw +/+ Tw + Rop-2	127[4]	8	27	9	3	0	4	0
y +/+ st	986	139	–	–	–	–	–	–
vg y st/+ + +	803	282	79	–	27	–	–	–
	908	315	168	–	48	–	–	–
spr y st/+ + +	764	197	90	–	31	–	–	–
y dfw ti/+ + +	302	51	76	–	8	–	–	–
y + tri/+ Tw +	1242	76	426	–	51	–	–	–

[1] See footnote (1), Table 2.
[2] Unpublished data of D. Berman.
[3] Data from table 2 of Arnold & Whitten (1976).
[4] Only *Rop*-2 mutant flies (survivors of topical dose applied to adults) used.

classes as doubles, triples and quadruples respectively. However, the single-crossover progeny indicate unambiguously that *Rop-1* lies between *bu* and *ra*. This is consistent with the finding of Arnold & Whitten (1976) that *Rop*-1 maps 5·3 units to the right of *bu*. The estimates of genetic distance between *bu*, *Rop-1* and *ra* (Fig. 1c) may be underestimates because of the exlcusion of multiple crossovers.

Chromosome 5 genetic mapping. The results of the chromosome 5 mapping crosses are presented in Table 5, and the composite map derived from these data in Fig. 1(*d*).

Chromosome 6 genetic mapping. The results of the chromosome 6 mapping crosses are presented in Table 6, and the composite map derived from these data in Fig. 1(*e*).

The relative order of *spr* and *vg* has not been determined. Fourteen crossovers between *vg* and *y* from the cross $vg + y/+ spr + ♀ × vg \ y \ ♂$ were progeny tested for *spr*, and none proved to have recombined between *vg* and *spr*. The position of *ho* with respect to *y* has not been determined in a three-point testcross, but the genetic distance between *B1* and *ho* (Table 6) suggests that the *ho* locus lies to the right of *y*. Similarly, most of the mutations in the right half of the linkage map of chromosome *6* have not been critically ordered with one another, but the order (*ho*, *Tw*)–*st*–*dfw*–(*Rop-2*–, *tri*, *ti*) appears most likely. Cytogenetic studies indicate that *st* lies to the left of *ti* (Foster *et al.* 1980).

The data indicate that both *Rop-2* and *ti* lie to the right of *dfw* (Table 6), however, as noted elsewhere (Whitten *et al.* 1975) we have been unable to demonstrate linkage directly between *Rop-2* and *ti* in crosses involving both markers (unpublished data). It is possible that this may reflect some interference with the expression of *Rop-2* on the part of *ti*, although we have no direct experimental evidence to support this hypothesis.

Interference. As in *D. melanogaster*, both centromere (Beadle, 1932) and chiasma (Stevens, 1936) interference appear to occur in *L. cuprina*. There is some evidence of clustering of mutations in centromere regions, particularly *sb*, *Spt*, *pt* and *bp* on chromosome *2* and *bz*, *mv*, *cu* and *sk* on chromosome *5* (Fig. 1*a*, *d*; Foster *et al.* 1980), suggesting suppression of crossing-over in these regions. Interference exists against double exchange in adjacent regions on the same side of a centromere, but not between regions on opposite sides of a centromere (Fig. 2). A knowledge of the degree of interference present in different regions of the genome is useful in the construction of crossover-suppressing chromosomes. With interference becoming virtually complete below 30 map units (Fig. 2) it should be possible to construct effective balancers by the sequential induction of overlapping inversions with break points at or near the centromere and every 30 map units or so along each arm.

4. DISCUSSION

As the knowledge of *L. cuprina* genetics becomes more extensive, the suitability of this species for other studies increases. For example, the availability of a series of eye-colour mutations has been utilized by Summers & Howells (1978, 1980) to elucidate steps in the synthesis of eye pigments in *L. cuprina*. Knowledge of the action of these mutations, and the particular enzymes involved, also enables the identification of homologies between specific loci across species, as noted by Milani (1967).

The use of market genes which cause similar phenotypic manifestations allows us to pursue, to a limited extent, the type of analysis conducted by Sturtevant & Novitski (1941) on the homologies of the chromosome elements in the genus *Drosophila*. From a comparison of the linkage relationships of phenotypically similar mutant genes they concluded that the 6 chromosome arms of *D. melanogaster* (*X*, *2L*, *2R*, *3L*, *3R*, *4*) retained their essential identity among the 14 species of *Drosophila* included in their study. Such conservation is no doubt a consequence of the relative infrequency of non-centromeric translocations and pericentric inversions, in contrast to paracentric inversions, which have become fixed frequently during the evolution of the genus *Drosophila*.

Establishment of genic homologies between species is most likely to be valid where an enzyme change is known to accompany a particular mutation, although similarities between certain types of morphological mutations may also be acceptable as evidence of homology. We have examined the linkage groups of *L. cuprina, M. domestica* and *D. melanogaster* for biochemically and morphologically similar mutations. As summarized in Table 7, there are substantial correlations between the autosomes of the first two species and the major chromosome arms of *D. melanogaster*.
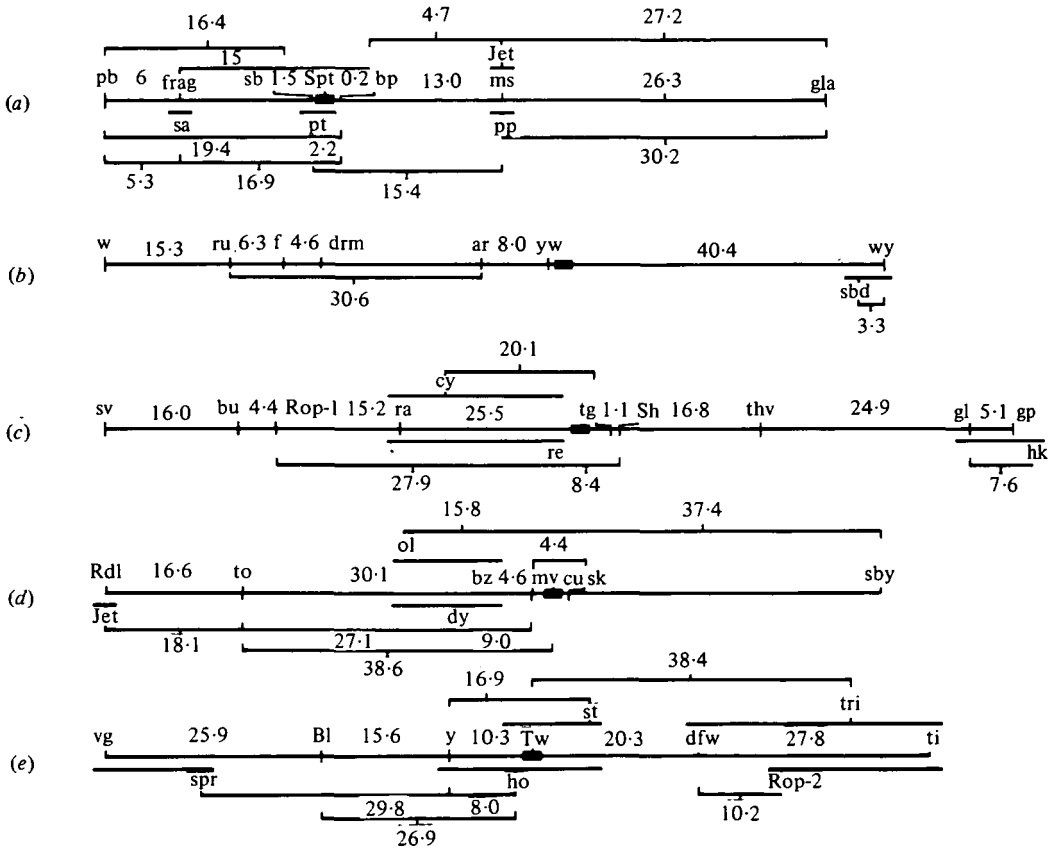
(a)

(b)

(c)

(d)

(e)

Fig. 1. Linkage maps of the autosomes of *L. cuprina*. (a) Chromosome 2, (b) Chromosome 3, (c) chromosome 4, (d) chromosome 5, (e) chromosome 6. Mutations sited on the main map of each chromosome have been positioned critically with respect to their nearest neighbours. The extent to which mutations not on the main map have *not* been positioned critically is indicated by the length of the satellite map lines. The positions of the centromeres as determined by Foster *et al.* (1980) are indicated by the thickened portions of the maps. *Spt, cu, ho, Tw* and *st* have not been positioned relative to their respective centromeres.

The *L. cuprina* chromosome 6 mutation *y* involves a deficiency of kynurenine hydroxylase, and is probably homologous with *ocra* in *M. domestica* (chromosome 5) and *cn* of *D. melanogaster* (chromosome 2R) (Summers & Howells, 1978). The same chromosomes of the three species also contain amylase loci (Berman, 1975; Hiroyoshi, 1977; O'Brien & Macintyre, 1978), the morphologically similar muta-

tions *vg* in both *L. cuprina* and *D. melanogaster* (Whitten *et al.* 1975; Lindsley & Grell, 1968) and *apt* in *M. domestica* (Milani, 1975), and insecticide resistance loci (Arnold & Whitten, 1976; Milani, 1975; Lindsley & Grell, 1968). Note that the *D. melanogaster* mutation *Rst(2)DDT* confers resistance to both DDT and organophosphorus insecticides (Lindsley & Grell, 1968).

The *L. cuprina* chromosome *3* mutation *w* is phenotypically identical to *w* in *M. domestica* (chromosome *3*) and *D. melanogaster* (*X* chromosome) (Whitten *et al.* 1975; Milani, 1975; Lindsley & Grell, 1968), all of which may involve inability
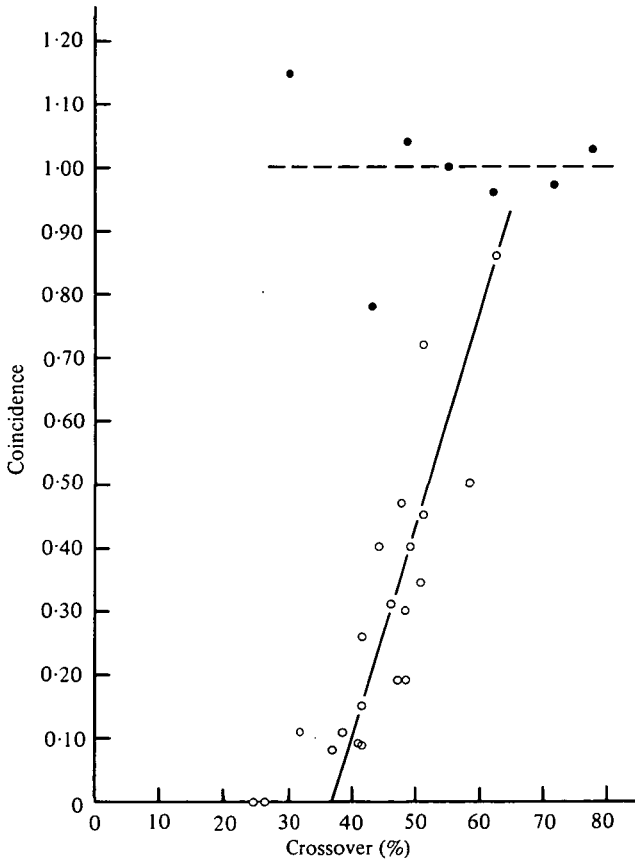

Fig. 2. Coincidence of double exchange in relation to map distance. Coincidence, calculated according to the formula of Stevens (1936), has been plotted against the total crossover percentage observed between the distal markers (singles + 2 × doubles ÷ *N*). Data were taken from crosses in which the expected numbers of doubles was 10 or more. ●–●, Regions on different sides of centromere; ○—○, both regions on same side of centromere.

to transport or store ommochrome and pteridine precursors (Howells, Summers & Ryall, 1977; Summers & Howells, 1978). The same chromosomes of the three species contain eye-colour mutations resulting in a deficiency of tryptophan oxygenase, *yw*, *ge* and *v*, respectively (Summers & Howells, 1978). Chromosome *3* of *M. domestica* contains the homozygous lethal *N* mutation, which causes serrated

wings and appears to involve a *w* deficiency (Hirroyoshi, 1977), thus being comparable to some *D. melanogaster* *N* mutations (Lindsley & Grell, 1968). The *L. cuprina* chromosome *3* mutation *ru* may cause some defect in tyrosine metabolism, as Hackman & Goldberg (1968) found both an altered activator of a pro-*o*-diphenoloxidase, and an altered pro-*o*-diphenoloxidase in a strain homozygous for both *ru* and *bp*. In *M. domestica* the chromosome *3* mutation *bwb* gives enhanced diphenoloxidase activity (Milani, 1975), and in *D. melanogaster* the *X*-chromosome mutations *y* and *t* both appear to involve defects in tyrosine metabolism (O'Brien & MacIntyre, 1978). Finally, both chromosome *3* in *L. cuprina* and the *X*-chromosome in *D. melanogaster* contain *wy* mutations (wavy wings) (Whitten *et al.* 1975; Lindsley & Grell, 1968).

Table 7. *Possible correlations between the chromosomes of* L. cuprina, M. domestica *and* D. melanogaster. *See text for references. The linkage group numbering system for* M. domestica *is that established by Wagoner* (*1967*)

L. cuprina	M. domestica	D. melanogaster
Chromosome *2*	Chromosome *1*	Chromosome *2L*
bp	bp	1(2) amd, Ddc
Luc-1, Luc-3	—	LSP-1ᵝ
Chromosome *3*	Chromosome *3*	X-chromosome
w	w	w
yw	ge	v
—	N	N
ru	bwb	y, t?
wy	—	wy
Chromosome *4*	Chromosome *2*	Chromosome *3R*
—	a, Deh,	Ali-est
Rop-1	Rdz, Ox²	—
—	Est A, B	Est C
—	ar	ssᵃ
—	atp	Antp
—	Phos	Acph-1
tg	car, cm, bu?	cd, kar?
gp	—	p, ca, ma?
Sh	—	Sb
Chromosome *5*	Chromosome *4*	Chromosome *3L*
to, to²	ye, rb	st
Rdl	dieldrin resistance	—
Est	—	Est-1
Chromosome *6*	Chromosome *5*	Chromosome *2R*
y	ocra	cn
Amy	Amy-A	Amy-1
	Amy-B	—
vg	apt	vg
Rop-2	DDT-md	Rst(2)DDT

Both *L. cuprina* chromosome *4* and *M. domestica* chromosome *2* contain major loci involved in diazinon resistance (Arnold & Whitten, 1976; Hiroyoshi, 1977). *M. domestica* chromosome *2* and *D. melanogaster* chromosome *3R* contain loci for aliesterases (which in *M. domestica* are involved in resistance to organophosphates and possibly DDT) (Milani, 1975; Hiroyoshi, 1977), esterase loci and acid

3-2

phosphatase loci (Hiroyoshi, 1977; O'Brien & MacIntyre, 1978), and two homoeotic loci, antennapedia and aristapedia (Milani, 1967; Lindsley & Grell, 1968). The *L. cuprina* chromosome *4* mutation *tg*, like *cd* (chromosome *3R*) of *D. melanogaster*, gives rise to reduced ommochrome levels and wild-type larval malpighian tubules, and a block in the final step of xanthommatin synthesis (Howells *et al.* 1977; Summers & Howells, 1978). In *M. domestica* three chromosome *2* mutations, *car*, *cm* and *bu*, cause reduced ommochrome levels (Milani, 1975), and could be comparable to *tg* (*L. cuprina*) and *cd* or *kar* (*D. melanogaster*). The *L. cuprina* chromosome *4* mutation *gp* causes reduced levels of pteridines and ommochromes, but does not affect XDH levels (Summers & Howells, 1978, 1980). This mutation may be comparable to *p*, *ca* or *ma* on *D. melanogaster* chromosome *3R*, all of which reduce the levels of both types of pigments but not XDH (Lindsley & Grell, 1968). Finally, the *L. cuprina* chromosome *4* homozygous lethal mutation *Sh* has a phenotypic expression very similar to that of *Sb on D. melanogaster* chromosome *3R*.

The *L. cuprina* chromosome *5* mutations *to* and *to²* reduce ommochrome levels without blockage of the tryptophan–ommochrome pathway, but possibly through a defect in transport or storage of ommochrome precursors (Summers & Howells, 1978, 1980). In some or all of these respects they are similar to *st* on *D. melanogaster* chromosome *3L* (Lindsley & Grell, 1968; Howells *et al.* 1977) and the *M. domestica* chromosome *4* mutation *ye* (Milani, 1975), which is allelic to *rb* (Hiroyoshi, 1977). Esterase loci have been reported on *L. cuprina* chromosome *5* (Berman, 1975) and *D. melanogaster* chromosome *3L* (O'Brien & MacIntyre, 1978). The *M. domestica* chromosome *4* dieldrin resistance locus (Milani, 1975) may be homologous with *Rdl* on chromosome *5* of *L. cuprina*.

L. cuprina chromosome *2* and *M. domestica* chromosome *1* both contain phenotypically similar *bp* mutations. In *L. cuprina*, *bp* is associated with an altered diphenoloxidase in the tyrosine metabolic pathway (Hackman & Goldberg, 1968). Similar studies have not been done in *M. domestica*, although the pupal sheath of *bp* mutant individuals of this species is reported to lack β-alanine (Hiroyoshi, 1977), which could implicate the tyrosine pathway. Similar mutations have not been reported in *D. melanogaster*. However, chromosome *2L* of this species, which so far has not been correlated with any chromosome of *L. cuprina* or *M. domestica*, contains two mutations involved in tyrosine metabolism (O'Brien & MacIntyre, 1978).

The linkage relationships of the genes coding for the homologous (Roberts, Wolfe & Akam, 1977) larval storage proteins lucilin and drosophilin (*Luc* and *LSP-1*, respectively), constitute an exception to the above correlation. Thomson *et al.* (1976) mapped *Luc-1* and *Luc-3* to chromosome *2* of *L. cuprina*, and concluded from their data that the other *Luc* loci for which genetic variants are known also map on chromosome *2*. Roberts & Evans-Roberts (1979) have mapped *LSP-1β* to chromosome *2L* of *D. melanogaster*, in agreement with the postulated correlation (Table 7). However, they found that *LSP-1α* maps on the *X*-chromosome and *LSP-1γ* maps on *3L*. The dispersal of *LSP-1* loci in the Drosophila

genome may have resulted from gross chromosomal rearrangement, but Roberts & Evans-Roberts (1979) suggest that association with a transposing element is a more likely explanation.

Thomson *et al.* (1976) postulated the existence of three *Luc* loci which apparently lack allelic variation. Since they were thus unable to map these loci, the possibility remains that they may not be linked to chromosome *2*. However, their conclusion that all the *Luc* loci are in the same linkage group is supported by the work of Kemp *et al.* (1978) on another calliphorid, *Calliphora vicina*. DNA prepared from *C. vicina* calliphorin mRNA hybridized to a single polytene chromosome region, suggesting that the structural genes for calliphorin are all on one chromosome.

Since the specific biochemical or developmental lesions responsible for many of the mutations listed above are not known, caution should be exercised in arguing that the linkage groups of these three species are homologous. Nevertheless, the apparent correlations suggest substantial conservation of linkage groups in the higher Diptera, although translocations between the muscoid and drosophilid linkage groups cannot yet be entirely ruled out.

The relationship between the sixth linkage group of *D. melanogaster*, chromosome *4*, and those of *L. cuprina* and *M. domestica* remains obscure. In *L. cuprina* a small linkage group resides in the *X* chromosome, and contains body-colour (Whitten *et al.* 1975) and wing venation mutations (unpublished) like the screw-worm *Cochliomyia hominivorax* (La Chance, Dawkins & Hopkins, 1966). In addition there is evidence that certain *X*-chromosome deficiencies in *L. cuprina* may be inviable in hemizygotes (Maddern & Bedo, unpublished), suggesting the existence of one or more vital genes on the *X*. No genetic factors are known to exist on the *Y* chromosome of *L. cuprina* other than that which determines maleness (Ullerich, 1963). No sex-linked locus other than sex itself has been identified in the *XY* ('standard') strains of *M. domestica* (Milani, 1975; Hiroyoshi, 1977). The *X* chromosome of the muscoids may correspond to the *D. melanogaster 4*, but in the absence of evidence this remains pure speculation.

Comparisons between species can facilitate identification of particular steps in the biochemical pathways for which mutants do not yet exist. Screening procedures could then be devised for isolating mutations at these presumptive loci. For example, an eye-colour mutation sensitive to dietary purine, such as *mal* (*X* chromosome) or *ry* (*3R*) of *D. melanogaster* (Lindsley & Grell, 1968), could be used in conjunction with sex-linked translocations to construct a female-killing system (cf. Whitten & Foster, 1975; Whitten *et al.* 1977), but such a mutation is not yet known in *L. cuprina*. From the correlation listed in Table 7, screening chromosomes *3* and *4* of *L. cuprina* would seem to offer the best chances of success in a search for this type of mutation.

It is clear from polytene chromosome studies (Foster *et al.* 1980; Foster & Boon, in preparation) that extensive homologies exist between the Caliphoridae and the Sarcophagidae. The correlations listed in Table 7 suggest that the homologies may extend to other muscoid Diptera, nearly all of which have five pair of autosomes

(Boyes, 1967). If the suggested correlations between *Lucilia*, *Musca* and *Drosophila* are supported by further research, the speculation of Sturtevant & Novitski (1941) that the linkage 'elements' (of *Drosophilia* may extend to other groups, will have proved to be prophetic.

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