

# The second generation of the International Equine Gene Mapping Workshop half-sibling linkage map

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## Summary

A low-density, male-based linkage map was constructed as one of the objectives of the International Equine Gene Mapping Workshop. Here we report the second generation map based on testing 503 half-sibling offspring from 13 sire families for 344 informative markers using the CRIMAP program. The multipoint linkage analysis localized 310 markers (90%) with 257 markers being linearly ordered. The map included 34 linkage groups representing all 31 autosomes and spanning 2262 cM with an average interval between loci of 10.1 cM. This map is a milestone in that it is the first map with linkage groups assigned to each of the 31 autosomes and a single linkage group to all but three chromosomes.

**Keywords** chromosome, genome, horse, linkage group, mapping, markers, microsatellite, segregation.

## Introduction

Selection for performance traits and avoiding hereditary health problems are important aspects of horse breeding. Therefore, development of a horse gene map has valuable applications. Previously, three linkage maps have been reported with 140, 161 and 359 markers, respectively, and containing many markers in common (Lindgren *et al.* 1998; Guérin *et al.* 1999; Swinburne *et al.* 2000). To create an effective genetic map, the International Equine Gene

Mapping Workshop was formed under the auspices of the Dorothy Russell Havemeyer Foundation and the International Society of Animal Genetics, with one of the goals being the development of a low-density genetic map. A first map was reported based on 161 markers (Guérin *et al.* 1999). Within this manuscript, workshop participants report significant advances in the coverage and density, and the unequivocal assignment of linkage groups to all chromosomes.

## Materials and methods

### Participants

The 21 participating laboratories for the second phase of the workshop are identified in the list of authors.

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Accepted for publication 6 December 2002

## Reference family panel

Paternal half-sib families were chosen as the basis for the International Horse Reference Family Panel (IHRFP) as described previously (Guérin *et al.* 1999). The number of offspring per family ranged from 21 to 52. Stallions and their families were identified with alphabetic codes A, B, C, D, E, F, G, H, J, K, L, N and Q. Family Q was added only for this second phase of the study and was provided by the University of Queensland laboratory, containing a cross-bred stallion with 46 offspring. Nine additional offspring were added for family A. In total, 503 offspring were typed for this report. However, the offspring of the Queensland family and the nine additional offspring for family A were only tested for the 188 loci added since the last report, plus blood typing loci. Informative meioses for dams were not included in the analysis.

## Markers and analysis

A total of 344 polymorphic loci were tested on the reference family panel and included seven blood group loci, 10 biochemical loci, 325 microsatellites and two loci tested for nucleotide variation using single-strand conformational polymorphism (SSCP) techniques (*DRA* & *DQA*). Samples were available for dams in several of the families, and this information was incorporated into the CRIMAP analysis to assist in determining the contribution of the stallion. Among the markers tested, six had not been previously reported, specifically *HESTG04*, *HESTG08*, *HESTG13*, *HMS52*, *HMS53* and *SGCV42*. The data were treated as previously described (Guérin *et al.* 1999) except that the BUILD option in the multipoint analysis of the CRIMAP program (Ver. 2.4) (Green *et al.* 1990) was started with a higher threshold. Briefly, maximum likelihood estimates of recombination fraction ( $\theta$ ) were calculated using the TWOPOINT option with a significant LOD-score threshold  $>3$  to determine linkage groups. Multi-point analysis on data from loci included in these linkage groups was then performed using the BUILD option to produce a framework map with a LOD-score threshold  $>6$  instead of three in the preceding analysis. The best linear order finally determined with a LOD-score  $>1$  was checked with the FLIPS option and map distances were calculated using the Kosambi function. Assignment of linkage groups to chromosomes was made based on physical data published or reported in connection with other studies, especially synteny mapping and fluorescent *in situ* hybridization (FISH).

## Websites

Further detailed information and appendices regarding the work described in this manuscript (acronyms for participating laboratories, reference families, markers, primer sequences and linkage analysis) can be found at <http://www.uky.edu/AG/horsemap/Maps/Guerin2>.

Details on locus assignments, marker characterizations and general mapping information are available in the databases at <http://locus.jouy.inra.fr> and at <http://roslin.thearkdb.org>.

## Results and discussion

### Genotyping

Genotype data for 156 loci across 12 families used in the first workshop (Guérin *et al.* 1999) were combined with data for an additional 188 loci with 503 offspring in 13 families to make this analysis and report. The number of observed segregations, that is the number of families in which a stallion was heterozygous for a given marker, was 2491, equivalent to 56% of the total. The typing of the combined 344 markers produced 87 649 genotypes. Percentage of heterozygous loci per stallion was: A, 62%; B, 58%; C, 59%; D, 66%; E, 46%; F, 58%; G, 63%; H, 61%; J, 46%; K, 64%; L, 52%; N, 60%; and Q, 53%. The genetic map was developed from 61 699 meioses and the average number of informative meioses per locus was 175.

### Linkage groups and map coverage

The CRIMAP program was used to compute LOD scores based on the reported segregation data and to construct the most probable linkage map presented in Table 1. Of the 344 markers which were informative, 310 (90%) showed significant linkage to other markers and were reported in the final map (Table 1). Among these, 257 were unambiguously ordered and all 310 were distributed among 34 linkage groups. Another 34 markers showed only weak linkage to other loci or linkage to multiple loci on different chromosomes indicating that available data fell outside the range for statistical assignment to a linkage group. The markers spanned 2262 cM and the average interval between loci was 10.1 cM, ranging from 0 to 38.4.

All linkage groups were assigned to one of the 31 autosomal chromosomes, specifically 28 by FISH and synteny mapping, one by FISH alone and five by synteny mapping alone. Three chromosomes (ECA08, ECA20 and ECA31) contained two distinct linkage groups. The orientation of 15 linkage groups was assigned using FISH mapping data for multiple loci; the orientation of eight other linkage groups could be deduced because terminal or subterminal markers in the groups were assigned to terminal or subterminal cytogenetic bands; and 11 have yet to be determined (Table 1). The specific assignment of linkage groups to chromosomes led to replacement of WS nomenclature with chromosome designations (ECA).

Because the linkage analysis was performed based on male meioses, it was not possible to investigate linkage distances for the two markers, *LEX013* and *VHL81*, on the X chromosome. No other markers showed linkage to those

**Table 1** Linkage of 310 loci tested on 13 half-sibling families that included 503 offspring<sup>1</sup>.

Chromosome <sup>2</sup>	Locus <sup>3</sup>	FISH/syteny <sup>4</sup>	cM <sup>5</sup>	cM cum <sup>6</sup>	Other loci <sup>7</sup> /orientation <sup>8</sup>
ECA01	<i>COR054</i>	01	0		
ECA01	<i>LEX030</i>	01	21.5	21.5	
ECA01	<i>VIASH34</i>	01	12.6	34.1	1CA30
ECA01	<i>HP27.1</i>	01	8.4	42.5	
ECA01	<i>LEX020</i>	01	14	56.5	
ECA01	<i>NVHEQ100</i>	01	28.9	85.5	
ECA01	1CA12		4.4	89.9	
ECA01	<i>COR100</i>	01	8.5	98.4	
ECA01	<i>VHL134</i>	01	6.1	104.5	1CA01
ECA01	<i>TKY297</i>		1.2	105.7	1CA01
ECA01	<i>UCDEQ487</i>	01	3.3	109.1	
ECA01	<i>TKY015</i>	01q12	9.2	118.3	LEX077, COR046, 1CA24
ECA01	<i>LEX058</i>	01	22.7	141	
ECA01	<i>TKY374</i>		6.2	147.2	
ECA01	<i>ASB08</i>	01q16–q17	1.4	148.6	
ECA01	1CA32		1.7	150.3	
ECA01	<i>TKY002</i>	01q17.2	11.6	161.9	
ECA01	1CA25		5.7	167.6	LEX049
ECA01	<i>UCDEQ440</i>	01	17	184.7	
ECA01	<i>HMS15</i>	01q21–q23	6.4	191.1	
ECA01	<i>TKY295</i>		15.1	206.2	
ECA01	<i>HMS07</i>	01	5.1	211.3	/Y
ECA02	<i>VHL078</i>	02	0		
ECA02	<i>HMS54</i>	02p14–p15	26.5	26.5	TKY003
ECA02	<i>ASB17</i>	02p14–p15	3	29.4	TKY003
ECA02	<i>HMS51</i>	02p14	4.9	34.4	TKY003
ECA02	<i>UCDEQ380</i>	02	7.6	42	
ECA02	<i>TKY340</i>		7.6	49.6	
ECA02	<i>PGD</i>	02p12–p13	6.1	55.7	
ECA02	<i>TKY358</i>		12.2	67.9	ASB13, EAK
ECA02	<i>A14</i>	02q14–q21	8.3	76.2	
ECA02	<i>HESTG13</i>		21	97.2	
ECA02	<i>VHL123</i>		15.3	112.5	/Y
ECA03	<i>ES</i>	03	0		
ECA03	<i>COR028</i>	03	13.2	13.2	
ECA03	<i>COR033</i>	03	10.1	23.2	
ECA03	<i>SGCV18</i>	03p13–p14	4	27.2	
ECA03	<i>UCDEQ437</i>	03	21.8	49	
ECA03	<i>LEX057</i>	03	6	55	
ECA03	<i>TKY353</i>		7.9	62.9	GC
ECA03	<i>ASB23</i>	03q22.1–q22.3	10.5	73.4	GC, ALB
ECA03	<i>LEX007</i>	03	7.9	81.2	
ECA03	<i>HTG02</i>	03	27.3	108.5	/Y
ECA04	<i>NVHEQ029</i>		0		
ECA04	<i>HMS06</i>	04	10	10	
ECA04	<i>TKY223</i>		1.8	11.8	
ECA04	<i>ASB03</i>	04p12–p13	21.5	33.3	
ECA04	<i>TKY337</i>		8.4	41.7	
ECA04	<i>COR057</i>	04	4.4	46	LEX061
ECA04	<i>HMS22</i>	04	7.7	53.7	
ECA04	<i>LEX050</i>	04	8.1	61.8	COR089
ECA04	<i>ASB22</i>	04q21	8.5	70.3	
ECA04	<i>LEX033</i>	04	3.4	73.6	
ECA04	<i>LEX072</i>	04	5.3	79	
ECA04	<i>HTG07</i>	04	5.2	84.1	
ECA04	<i>HMS19</i>	04q21	6.7	90.8	

Table 1 (Continued)

Chromosome <sup>2</sup>	Locus <sup>3</sup>	FISH/synteny <sup>4</sup>	cM <sup>5</sup>	cM cum <sup>6</sup>	Other loci <sup>7</sup> /orientation <sup>8</sup>
ECA04	<i>HMS09</i>	04	8.9	99.7	
ECA04	<i>HTG22</i>		22.9	122.6	
ECA04	<i>SGCV23</i>	04q27	14	136.5	/Y
ECA05	<i>TKY271</i>		0		
ECA05	<i>HMS52</i>	05q12	7.2	7.2	
ECA05	<i>HMS05</i>	05	16.2	23.4	
ECA05	<i>LEX069</i>		7.3	30.7	<i>UCDEQ304</i>
ECA05	<i>LEX034</i>	05	17.7	48.4	
ECA05	<i>ASB10</i>	05	8	56.4	
ECA05	<i>LEX014</i>	05q16–q17	13.9	70.3	
ECA05	<i>HESTG08</i>		7.4	77.7	
ECA05	<i>TKY344</i>		2.9	80.6	/Y
ECA06	<i>HMS55</i>	06	0		<i>COR088</i>
ECA06	<i>NVHEQ082</i>		3.4	3.4	
ECA06	<i>TKY312</i>		2.7	6.1	
ECA06	<i>LEX065</i>	06	9.2	15.3	
ECA06	<i>COR070</i>	06	36.3	51.6	
ECA06	<i>UCDEQ465</i>	06	9.3	60.9	
ECA06	<i>TKY028</i>	*9	3.9	64.8	
ECA06	<i>TKY284</i>		8	72.8	/N
ECA07	<i>SGCV28</i>	07	0		
ECA07	<i>TKY305</i>		6.9	6.9	
ECA07	<i>AHT19</i>	07	8.5	15.4	
ECA07	<i>ASB40</i>	07q18–q19	6.8	22.2	/L
ECA08a	<i>AHT05</i>	08	0		
ECA08a	<i>COR097</i>	08	7.2	7.2	/N
ECA08b	<i>UCDEQ046</i>	08	0		
ECA08b	<i>LEX029</i>	08	7.4	7.4	<i>UM034</i>
ECA08b	<i>COR012</i>	08	8.8	16.2	<i>LEX023</i>
ECA08b	<i>SGCV32</i>	08q14–q16	10.1	26.3	<i>SGCV42</i>
ECA08b	<i>COR003</i>	08	6.6	32.9	
ECA08b	<i>UM033</i>	08	16.1	49	
ECA08b	<i>COR056</i>	08	11.3	60.3	/N
ECA09	<i>HTG04</i>	09	0		
ECA09	<i>HMS03</i>	09	38.4	38.4	<i>VHL126</i>
ECA09	<i>COR008</i>	09	4.5	42.9	
ECA09	<i>PK9TET</i>	9p12	9.3	52.3	
ECA09	<i>LEX070</i>	09	2.3	54.6	
ECA09	<i>ASB04</i>	09q16–q18	28.4	83.1	
ECA09	<i>ASB05</i>	09q16–q18	23.8	106.8	
ECA09	<i>ASB21</i>		16.7	123.5	
ECA09	<i>LEX019</i>	09	7.6	131.1	/L
ECA10	<i>COR045</i>	10	0		
ECA10	<i>UCDEQ482</i>	10	12.3	12.3	
ECA10	<i>COR048</i>	10	1.7	14	
ECA10	<i>ASB06</i>	10p13	8.3	22.3	<i>HMS56</i>
ECA10	<i>NVHEQ018</i>	10	5.6	27.9	<i>HMS56</i>
ECA10	<i>NVHEQ007</i>	10	15.5	43.4	
ECA10	<i>LEX066</i>	10	6.4	49.8	
ECA10	<i>LEX008</i>	10	3.1	52.9	
ECA10	<i>COR015</i>	10	5.4	58.3	
ECA10	<i>LEX017</i>	10	3.1	61.4	
ECA10	<i>UCDEQ412</i>	10	1.9	63.3	
ECA10	<i>SGCV30</i>	10qter	3	66.3	
ECA10	<i>HMS02</i>	10	15.7	82	<i>UM040</i>
ECA10	<i>ASB09</i>	10q21–q23	3.2	85.2	

Table 1 (Continued)

Chromosome <sup>2</sup>	Locus <sup>3</sup>	FISH/syteny <sup>4</sup>	cM <sup>5</sup>	cM cum <sup>6</sup>	Other loci <sup>7</sup> /orientation <sup>8</sup>
ECA10	SGCV17	10q21–q23	5.8	91	UM040/Y
ECA11	AHT44	11	0		UCDEQ062, UCDEQ439
ECA11	NVHEQ040	11	15.7	15.7	
ECA11	LEX068	11	3.8	19.5	UCDEQ439
ECA11	TKY343		4.3	23.8	
ECA11	SGCV24	11p12	10.3	34.1	
ECA11	D8	11p12–p13	2.3	36.4	
ECA11	SGCV13	11q12	12.7	49.1	/Y
ECA12	SGCV10	12p13	0		
ECA12	SGCV08	12	13.4	13.4	
ECA12	UCDEQ411	12	13.5	26.9	
ECA12	COR058	12	14.4	41.3	
ECA12	AHT17	12	19	60.2	
ECA12	UCDEQ497	12	3.2	63.4	/L
ECA13	HB	13pter	0		
ECA13	ASB01	13	18.2	18.2	
ECA13	AHT30	*	19.2	37.5	
ECA13	VHL047	13	7.7	45.1	LEX041, VHL161
ECA13	ASB37	13q11–q12	5.1	50.3	
ECA13	SGCV03	13q12	5.5	55.8	VHL161/Y
ECA14	LEX043	14	0		HTG18
ECA14	AHT29	14q13	25.6	25.6	
ECA14	TKY267	14q13	14.2	39.8	
ECA14	VHL204		13	52.7	
ECA14	UM010	14	2.5	55.3	
ECA14	TKY376		2.6	57.9	VHL209
ECA14	LEX047	14	7.1	64.9	
ECA14	TKY310		14.1	79.1	HTG18
ECA14	HP4.2–2	14	11.5	90.5	
ECA14	LEX078	14q27	30.3	120.8	
ECA14	COR002	14	10.8	131.7	
ECA14	EAD		11.5	143.2	/Y
ECA15	B8	15q14–q21	0		
ECA15	LEX051	15	17.5	17.5	
ECA15	LEX046	15	2.7	20.3	
ECA15	ASB15	15q21.3–q23	16.9	37.1	SGCV06
ECA15	ASB02	15q21.3–q23	5.4	42.5	SGCV06
ECA15	ASB19	15q21.3–q23	3.6	46.1	
ECA15	HTG06	15q26–q27	17.6	63.7	
ECA15	HMS01	15	17.9	81.6	
ECA15	COR014	15	2.6	84.2	
ECA15	COR075	15	1.6	85.8	/Y
ECA16	AHT37	16q13	0		
ECA16	TKY279		8.3	8.3	
ECA16	HTG03	16q13	6.9	15.1	TKY311
ECA16	HMS20	16	18.2	33.3	LEX059
ECA16	COR011	16	12.7	46	
ECA16	VHL145	16	30.9	77	
ECA16	LAMBDA15.2	16	9.3	86.2	ASB42, UCDEQ505
ECA16	LEX056	16	6.5	92.8	AHT14
ECA16	TF	16q23	4.5	97.2	
ECA16	I18	16q23–q25	17.3	114.6	
ECA16	TKY341		13.1	127.7	/Y
ECA17	COR105		0		
ECA17	COR007	17	3.5	3.5	
ECA17	COR072	17	3.8	7.4	

Table 1 (Continued)

Chromosome <sup>2</sup>	Locus <sup>3</sup>	FISH/syteny <sup>4</sup>	cM <sup>5</sup>	cM cum <sup>6</sup>	Other loci <sup>7</sup> /orientation <sup>8</sup>
ECA17	<i>TKY287</i>		2.2	9.5	
ECA17	<i>HMS41</i>	17q21.3–q22	20.8	30.3	
ECA17	<i>NVHEQ079</i>		5.4	35.7	
ECA17	<i>UCDEQ014</i>	17	8.8	44.6	/N
ECA18	<i>TKY019</i>	18q13	0		
ECA18	<i>UCDEQ136</i>	18	4	4	
ECA18	<i>LEX054</i>	18	10.8	14.9	<i>HTG28, SGCV07</i>
ECA18	<i>HMS46</i>	18	17.1	32	
ECA18	<i>HTG17</i>	18	31.7	63.7	
ECA18	<i>UCDEQ387</i>	18	14.4	78.1	/L
ECA19	<i>COR062</i>	19	0		
ECA19	<i>AHT41</i>	19q13	6.9	6.9	
ECA19	<i>I12</i>	19q12–q14	8.6	15.5	
ECA19	<i>LEX036</i>	19	18	33.5	<i>HTG23</i>
ECA19	<i>ASB07</i>	19q14–q16	5.4	38.9	
ECA19	<i>LEX073</i>	19	9.9	48.8	
ECA19	<i>LEX035</i>	19	3	51.8	
ECA19	<i>HMS08</i>	19	22.2	74	
ECA19	<i>NVHEQ011</i>	19	9.7	83.7	
ECA19	<i>ASB11</i>	19q21–q22	12.4	96.1	/Y
ECA20a	<i>VHL137</i>		0		
ECA20a	<i>HTG05</i>	20	11.6	11.6	<i>NVHEQ005</i>
ECA20a	<i>AHT18</i>	20	17.2	28.8	
ECA20a	<i>LEX052</i>	20	11	39.8	
ECA20a	<i>LEX064</i>	20	4.4	44.3	<i>DRA, EAA</i>
ECA20a	<i>DQA</i>		14.9	59.2	/N
ECA20b	<i>COR050</i>	20	0		
ECA20b	<i>TKY321</i>		16.8	16.8	
ECA20b	<i>NVHEQ021</i>		3.7	20.5	
ECA20b	<i>HMS42</i>	20q24	9.2	29.6	/N
ECA21	<i>SGCV14</i>	21q13	0		
ECA21	<i>HTG10</i>	21	13.9	13.9	<i>SGCV16</i>
ECA21	<i>COR073</i>	21	4.9	18.8	
ECA21	<i>LEX060</i>	21	1.7	20.5	
ECA21	<i>COR068</i>	21	4.5	25	
ECA21	<i>HTG32</i>		18.6	43.6	<i>LEX031/L</i>
ECA22	<i>HMS53</i>		0		
ECA22	<i>HTG21</i>	22	4.5	4.5	
ECA22	<i>COR016</i>	22	11.2	15.7	
ECA22	<i>HMS47</i>	22q19	22.3	38	
ECA22	<i>SGCV19</i>	22q19	16	54	<i>NVHEQ067/Y</i>
ECA23	<i>COR055</i>	23	0		
ECA23	<i>TKY346</i>		7.1	7.1	
ECA23	<i>ASB39</i>	23q15	2.7	9.8	
ECA23	<i>HP13.2</i>	23	10.5	20.3	<i>TKY301</i>
ECA23	<i>LEX063</i>	23	2.4	22.7	
ECA23	<i>LEX053</i>	23	8.7	31.4	
ECA23	<i>TKY269</i>		10.4	41.7	
ECA23	<i>SGCV04</i>	23q19	22.8	64.5	/Y
ECA24	<i>EAU</i>	24	0		
ECA24	<i>AHT04</i>	24q14	18.2	18.2	<i>LEX042</i>
ECA24	<i>COR061</i>	24	10.7	29	
ECA24	<i>TKY394</i>		4.1	33	
ECA24	<i>LEX074</i>	24	1	34	
ECA24	<i>LEX032</i>	24	5.6	39.5	
ECA24	<i>PI</i>	24q15–q16	7.2	46.7	

Table 1 (Continued)

Chromosome <sup>2</sup>	Locus <sup>3</sup>	FISH/syteny <sup>4</sup>	cM <sup>5</sup>	cM cum <sup>6</sup>	Other loci <sup>7</sup> /orientation <sup>8</sup>
ECA24	UCDEQ467	24	4.1	50.8	
ECA24	COR024	24	7.5	58.3	/Y
ECA25	NVHEQ043	25	0		
ECA25	UCDEQ405	25	15.3	15.3	
ECA25	COR018	25	4.3	19.6	
ECA25	COR080	25	10.4	30	
ECA25	UCDEQ464	25q18–q19	5.2	35.3	/L
ECA26	COR071	26	0		UM031, NVHEQ070
ECA26	LEX044	26q13–q15	7.2	7.2	UM031
ECA26	A17	26q13–q14	7.8	15.1	UM031, NVHEQ070/N
ECA27	COR031	27	0		
ECA27	COR040	27	9.9	9.9	
ECA27	UCDEQ005	27	8.3	18.2	
ECA27	ASB38	27	0.8	19	
ECA27	TKY294		7.2	26.2	HMS45
ECA27	LEX005	27q16	14.7	40.8	VHL150
ECA27	COR017	27	7.6	48.4	/L
ECA28	IGF1	28	0		
ECA28	HTG30		6.1	6.1	NVHEQ054
ECA28	UM003	28	3	9.1	NVHEQ054
ECA28	TKY333		3.5	12.6	/N
ECA29	COR082	29	0		
ECA29	COR027	29	26	26	
ECA29	VIASH39	29	15.9	41.8	
ECA29	TKY325		7.3	49.2	
ECA29	LAMBDA12.2	29	2	51.1	
ECA29	ASB43	29q16	6.4	57.5	L
ECA30	LEX075	30	0		HMS18, LEX025, VHL20
ECA30	UCDEQ455	30	6.4	6.4	HMS18, LEX025, VHL20/N
ECA31a	AHT33	31q15–q16	0		COR038
ECA31a	VIASH21	31	0	0	COR038/N
ECA31b	AHT34	31q16	0		
ECA31b	TKY278		0	0	/N

<sup>1</sup>Includes 310 of 344 polymorphic loci that were tested. The remaining 34 loci could not be placed on the map.

<sup>2</sup>Location of the marker with subdesignation 'a' or 'b' identifying different linkage groups on that chromosome.

<sup>3</sup>Published acronym for each of the genes or markers tested.

<sup>4</sup>Assignments to chromosomes based on other studies using syteny or FISH.

<sup>5</sup>Distance between adjacent loci in a linkage group.

<sup>6</sup>Cumulative distance for a linkage group.

<sup>7</sup>Markers that appear in the linkage groups but which cannot be placed into a definitive linear arrangement with respect to other loci. They are shown in their most likely position.

<sup>8</sup>Orientation of the linkage group. '/Y' indicates orientation is known; '/L' denotes that orientation is assumed based on terminal location for one markers; '/N' indicates that orientation is unknown.

<sup>9</sup>Asterisk represents apparent mapping discrepancy (see text).

X-chromosome markers. None of the markers demonstrated Y-chromosome segregation.

Two loci were assigned to linkage groups in conflict with previously reported FISH mapping assignments. *AHT30* was linked to ECA13 markers despite reported FISH mapping to ECA22p13. *TKY028* was previously mapped by FISH to ECA10 while in this report it was linked with markers on ECA6. Evidence for linkage mapping of *AHT30* and *TKY028*, respectively, were sufficiently strong in this study

that they merited reporting despite the discrepancies. In addition, these interpretations were supported by family studies (J.E. Swinburne, personal communication) and syteny mapping (L.V. Millon, personal communication).

In summary, this second generation of the International Equine Genetic Map represents a significant improvement compared with the precedent version (Guérin *et al.* 1999). The genome map of the horse continues to grow through the continuing development of the linkage maps as well as

introduction of radiation hybrid maps (Kiguwa *et al.* 2000; Chowdhary *et al.* 2002).

### Acknowledgements

The workshop was conducted under the auspices and with support from the Dorothy Russell Havemeyer Foundation, Inc. We also acknowledge financial support from USDA-NRSP-8, the New Zealand Equine Research Foundation, the New South Wales Racing Research Fund in the University of Sydney, the Link Foundation Endowment for Equine Research at Texas A & M, The Morris Animal Foundation, the Harry M. Zweig Memorial Fund for Equine Research in New York State, the University of Queensland SPIRT Grant 99/ARCCOL003G, University of Kentucky Agricultural Experiment Station 02-14-127, Kaltenbor's fund of Norway INRA, Département de Génétique animale and Haras Nationaux and technical assistance from Patrick Gallagher, Rebecca Terry, Michelle Mousel, Catherine Wagner at the University of Kentucky, Shane Thomas and Glenda Goh from the University of Queensland and D. Honeycutt from Texas A & M University.

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