An abundance of X-linked genes expressed in spermatogonia

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Spermatogonia are the self-renewing, mitotic germ cells of the testis from which sperm arise by means of the differentiation pathway known as spermatogenesis[1]. By contrast with hematopoietic and other mammalian stem-cell populations, which have been subjects of intense molecular genetic investigation, spermatogonia have remained largely unexplored at the molecular level. Here we describe a systematic search for genes expressed in mouse spermatogonia, but not in somatic tissues. We identified 25 genes (19 of which are novel) that are expressed in only male germ cells. Of the 25 genes, 3 are Y-linked and 10 are X-linked. If these genes had been distributed randomly in the genome, one would have expected zero to two of the genes to be X-linked. Our findings indicate that the X chromosome has a predominant role in pre-meiotic stages of mammalian spermatogenesis. We hypothesize that the X chromosome acquired this prominent role in male germ-cell development as it evolved from an ordinary, unspecialized autosome.

We identified genes specific to germ cells through 'cDNA subtraction'[2,3], whereby a pool of transcripts present in one cell type ('tracer') is depleted of transcripts shared with other cell types ('driver'). In our subtraction, tracer cDNA was generated from purified mouse spermatogonia[4], whereas driver cDNA was generated from a combination of 11 somatic tissues (heart, brain, lung, liver, skeletal muscle, kidney, spleen, stomach, thymus, skin and germ-cell-depleted *Kit*$^{W\text{-}v/W\text{-}v}$ testis[5]).

To validate our cDNA subtraction experiments, we tested whether we had recovered previously identified genes that were known to be expressed in spermatogonia but not in somatic tissues. Eight such genes (*Mage*, *Ube1y*, *Usp9y*, *Rbmy*, *Stra8*, *Ott*, *Ddx4* and *Dazl*; Table 1) had been identified during the past decade through the efforts of several laboratories. The extent to which we recovered the eight known genes would provide a measure of our protocol's adequacy in capturing spermatogonially expressed, germ-cell–specific genes. We determined the nucleotide sequence of 2,235 fragments chosen at random from the cDNA subtraction product. We expected that this collection of sequence fragments would constitute a redundant sampling of a much smaller set of genes. Nucleotide sequence analysis revealed that 409 fragments corresponded to 13 known germ-cell–specific genes, including all 8 genes shown to be expressed in spermatogonia in previous studies (Table 1). We recovered five other known germ-cell–specific genes (Table 1) that were not previously reported to be expressed in spermatogonia. We tested and confirmed their expression in purified spermatogonia by RT–PCR (data not shown; primitive type A and mature type A and B spermatogonia prepared from prepubertal testes). We recovered no known genes specific to meiotic or post-meiotic germ cells. These results indicated that our spermatogonial cDNA subtraction would provide an efficient and sensitive route to the identification of germ-cell–specific genes expressed before meiosis.

Through further analysis of the remaining subtraction product sequences, we identified 23 novel germ-cell–specific genes. We first identified sequence fragments that were present at least twice among the 2,235 subtraction product sequences and that did not correspond to known genes. By testing these sequence fragments for expression in diverse mouse tissues, we identified novel fragments that seemed to be expressed in germ cells, but not in somatic cells of the testis or other organs (Fig. 1). Nucleotide sequencing of cDNA clones, and rescreening of libraries as necessary, resulted in full-length cDNA sequences for 23 novel germ-cell–specific genes (Table 2).

Virtual translation of these novel cDNA sequences and comparison with the previously reported genes indicate that many spermatogonially expressed, germ-cell–specific proteins are involved in transcriptional or post-transcriptional regulation of gene expression. Similarities to well-characterized proteins suggest that these proteins include a component of RNA polymerase II transcription initiation complexes (the product of *Taf2q*; Table 2), a nuclear RNA export factor (*Nxf2*), a ribonuclease inhibitor (*Rnh2*), a ring-finger protein (*Rnf17*), an RNA helicase (*Mov10l1*), and four proteins with RNA-binding domains (RRM domains in the *Dazl* (refs. 6,7) and *Rbmy* products[8]; tudor domains in the *Stk31* and *Tdrd1* products). These findings, and particularly the large number of putative RNA regulators, are reminiscent of the large role played by post-transcriptional gene regulation in pre-meiotic germ-cell development in *Drosophila melanogaster* and *Caenorhabditis elegans*[9,10]. Our studies suggest that the same is true of pre-meiotic germ-cell development in mammals.

We examined the sex specificity of all 36 spermatogonially expressed, germ-cell–specific genes—in particular, whether they are expressed in the ovary, the site of female germ cells. Eleven genes (four novel genes and seven previously reported genes) are

Table 1 • Known mouse genes expressed in spermatogonia but not in somatic tissues

Gene symbol*	Expression	Chromosome
Mage	testis	X
Ube1y	testis	Y
Usp9y	testis	Y
Rbmy	testis	Y
Tuba3/Tuba7	testis	6
Stra8	testis	6
Ott	testis and ovary	X
Sycp2	testis and ovary	2
Sycp1	testis and ovary	3
Figla	testis and ovary	6
Sycp3	testis and ovary	10
Ddx4	testis and ovary	13
Dazl	testis and ovary	17

*For references, see Web Table A.

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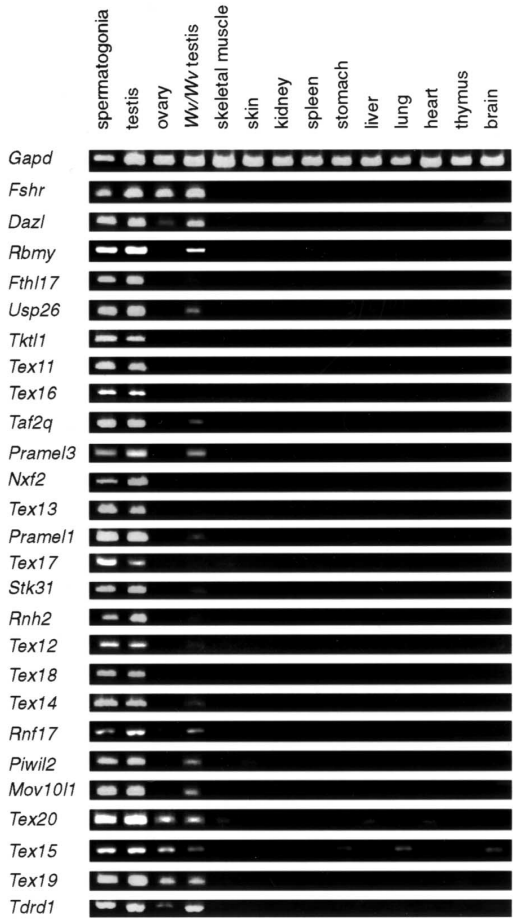

Fig. 1 Expression of 23 novel germ-cell-specific genes in mouse tissues assayed by RT–PCR. RNAs were prepared from spermatogonia of 8-d CD-1 males, testes of 8-d C57BL/6J males, ovaries of adult C57BL/6J females, germ-cell–depleted testes of adult $Kit^{W\text{-}v/W\text{-}v}$ C57BL/6J males[5], and other tissues of 8-d C57BL/6J males. The *Gapd*, *Fshr*, *Dazl* and *Rbmy* served as controls. *Gapd* is expressed ubiquitously. *Fshr* is expressed in somatic cells of testis and ovary. The *Fshr* signal in the spermatogonial lane likely reflects the fact that there is 15% contamination (with testicular somatic cells) in the spermatogonial preparation. *Rbmy* and *Dazl* are expressed only in germ cells, with *Rbmy* expressed only in testis[8] and *Dazl* expressed in both testis and ovary[6,7]. Germ cells are reduced in number, but are not entirely absent, in $Kit^{W\text{-}v/W\text{-}v}$ testes. For genes expressed in spermatogonia, one expects to see a reduced RT–PCR signal (or none) in $Kit^{W\text{-}v/W\text{-}v}$ testes as compared with wild-type testes. This was observed for all genes except the somatically expressed controls *Gapd* and *Fshr*. For *Tex20* and *Tex15*, faint RT–PCR signals are visible in some somatic tissues. Additional RT–PCR data not shown: all 36 germ-cell-specific genes under study (including the 13 genes listed in Table 1) were found to be expressed in primitive type A spermatogonia prepared from 6-d CD-1 mice.

expressed in ovary and in male germ cells (Tables 1 and 2, and Fig. 1). By contrast, the other 25 genes (19 of which are novel) seem to be male-specific (Table 1 and Fig. 1).

We then discovered a strong and unexpected correlation between the sex specificities of the genes and their genomic locations. We ascertained the chromosomal locations of all 36 spermatogonially expressed, germ-cell–specific genes, 8 of which had been mapped previously (Fig. 2). As expected, the 11 genes that are expressed in both testes and ovaries seemed to be scattered randomly throughout the genome, with 1 such gene mapping to the X chromosome and the other 10 genes distributed among 9 autosomes. By contrast, 13 of 25 male-specific genes mapped to sex chromosomes, with 3 genes (all as previously reported) localizing to the Y chromosome and 10 genes (9 of which are novel) mapping to the X chromosome. If the 22 non-Y-chromosomal, male-specific genes had been distributed randomly throughout the genome, one would have expected 0–2 such genes to be X-linked. Our finding of 10 X-linked genes is highly unlikely to have occurred by chance ($P<10^{-8}$), and it indicates a roughly 15-fold enrichment on the X chromosome for male germ-cell–specific, spermatogonially expressed genes. Our mapping and expression studies indicate that, in mammals, the X chromosome has a role in the mitotic stages of spermatogenesis.

Why should the mammalian X chromosome have such a specialization in spermatogonial function? The mammalian X and Y chromosomes evolved from an ordinary pair of autosomes, a gradual process that began approximately 240–320 million years ago with the emergence of *SRY*, the sex-determining factor, on one member of that ancestral autosomal pair[11,12]. Apart from *SRY*, the ancestral autosome was unlikely to have had an outsized role in testicular development or function. At issue then are the adaptive forces that caused the X chromosome, as it differentiated from the Y chromosome, to accumulate so many genes expressed in early stages of spermatogenesis. No explanation is provided by traditional, prevailing models of mammalian X-chromosome evolution, as these have focused on issues of gene dosage (the emergence of X inactivation) without envisioning or predicting any functional specialization[13,14]. We will outline two possible explanations, both previously debated in evolutionary biology: sex-chromosome meiotic drive[15–17] and sexual antagonism[18–20].

Meiotic drive, which has been documented in diverse species, including mice[21], refers to mechanisms that result in preferential transmission of certain chromosomes at the expense of their homologs. X and Y chromosomes are considered much more likely to become subject to meiotic drive during evolution than are autosomes[15,16]. Sex-chromosome meiotic drive skews the transmission of X versus Y chromosomes to gametes, and thus the critical drive genes should be expressed during spermatogenesis. Perhaps some of the X-linked genes that we report are drivers of X transmission or suppressors of Y transmission.

The theory of sexually antagonistic genes, which has been postulated to explain the wealth of spermatogenesis factors on mammalian Y chromosomes[18,22], might also account for our findings on the X chromosome. Studies in *Drosophila* and other systems have demonstrated the existence of sexually antagonistic genes, which enhance reproductive fitness in one sex but diminish fitness in the other sex[23]. Empirical and theoretical studies indicate that, during evolution, sexually antagonistic genes should accumulate preferentially on sex chromosomes[18,19]. Here, conditions favor the emergence of genes that benefit the heterogametic sex (for example, XY), even when those genes are detrimental to the homogametic sex (XX). Sexual antagonism provides a powerful explanation for the enrichment of dominant genes that benefit males on Y chromosomes, including male-ornamentation genes in guppies and spermatogenesis genes in mammals[18,22,24].

Focusing on recessive mutations that enhance reproductive fitness in males but diminish it in females, it has been argued that natural selection should favor the emergence of sexually antagonistic alleles on X chromosomes[19]. The evolutionary dynamics of such male-benefit mutations were considered when they first appear as rare alleles on X chromosomes as opposed to autosomes. When they are rare, autosomal recessive alleles would be of no advantage to (heterozygous) males and thus would be unlikely to spread widely in the population. By contrast, X-linked recessive alleles would immediately benefit hemizygous males, greatly increasing the alleles' likelihood of permeating the population. Eventually, as an allele's frequency increased in the population, female fitness would be diminished by the detrimental

Table 2 • New spermatogonially expressed, germ-cell–specific genes in mouse, and their human orthologs

Mouse genes Gene symbol	Gene name	Expression	Chr	Comments*	Human orthologs Gene symbol	Chr
Fthl17	ferritin heavy polypeptide-like 17	testis	X	ferritin, functioning in iron metabolism, consists of 24 heavy and light chains	*FTHL17*	X
Usp26	ubiquitin specific protease 26	testis	X	predicted protein contains His and Cys domains conserved among deubiquitinating enzymes	*USP26*	X
Tktl1	transketolase-like 1	testis	X	homologous to human transketolase	*TKTL1*	
Tex11	testis expressed gene 11	testis	X	novel 947-residue protein	*TEX11*	X
Tex16	testis expressed gene 16	testis	X	novel 1,139-residue protein; rich in serine		
Taf2q	TBP-associated factor, RNA polymerase II, Q	testis	X	human autosomal homolog *TAF2F* encodes a component of TFIID	*TAF2Q*	X
Pramel3	PRAME (human)-like 3	testis	X	homologous to human *PRAME*, encoding a melanoma antigen recognized by cytotoxic T cells		
Nxf2	nuclear RNA export factor 2	testis	X	homologous to Mex67p, *NXF1* and *NXF2*, encoding nuclear RNA export factors	*NXF2*	X
Tex13	testis expressed gene 13	testis	X	novel 186-residue protein; 2 closely related homologs on human X chromosome	*TEX13A* *TEX13B*	X X
Pramel1	PRAME (human)-like 1	testis	4	homologous to human *PRAME*		
Tex17	testis expressed gene 17	testis	4	novel 120-residue protein; calculated pI 9.9		
Stk31	serine/threonine kinase 31	testis	6	putative protein kinase with tudor domain (found in RNA-interacting proteins) and coiled coil region	*STK31*	7
Rnh2	ribonuclease inhibitor 2	testis	7	predicted protein contains 6 leucine-rich repeats		
Tex12	testis expressed gene 12	testis	9	novel 123-residue protein with coiled coil region	*TEX12*	11
Tex18	testis expressed gene 18	testis	10	novel 80-residue protein		
Tex14	testis expressed gene 14	testis	11	predicted protein contains 2 protein kinase domains	*TEX14*	17
Rnf17	ring finger protein 17	testis	14	a RING finger-containing protein	*RNF17*	13
Piwil2	piwi (*Drosophila*)-like 2	testis	14	homologous to *Drosophila piwi*, involved in germline stem cell renewal and meiotic drive		
Mov10l1	Mov10 (mouse)-like 1	testis	15	putative RNA helicase	*MOV10L1*	22
Tex20	testis expressed gene 20	testis and ovary	2	novel 188-residue protein; calculated pI 10.2		
Tex15	testis expressed gene 15	testis and ovary	8	novel 2785-residue protein	*TEX15*	8
Tex19	testis expressed gene 19	testis and ovary	11	novel 351-residue protein with coiled coil region		
Tdrd1	tudor domain protein 1	testis and ovary	19	predicted protein contains 4 tudor domains	*TDRD1*	10

*For references, see Web Table B.

effects of homozygosity. This would generate adaptive pressure to limit the gene's expression to males, through additional mutations. Based on this theoretical scenario, it was postulated that X chromosomes should evolve to carry a disproportionate share of male-specific genes functioning in male differentiation[19]. Our findings are in accord with this prediction.

Our hypothesis that mammalian X chromosomes have preeminent roles in early stages of spermatogenesis can now be tested through targeted disruption of the many X-linked mouse genes reported here, and through genetic studies in humans. To facilitate studies in humans, we identified orthologous, full-length human cDNA sequences for 13 of 23 novel mouse genes reported here (Table 2). In all 13 cases, the orthologous human genes are expressed exclusively in testes (or testes and ovaries), presumably in germ cells (Fig. 3), and map to chromosomal regions of known conserved synteny between the mouse and human genomes (Table 2). In particular, we have identified testis-specific, X-linked human orthologs of six of the novel testis-specific, X-linked mouse genes reported here. In the cDNA subtraction experiments reported here, we recovered the mouse homologs of *USP9Y*, *RBMY* and *DAZ*, the three human Y chromosomal genes that have been most strongly implicated in male infertility[25–27] (Table 1). Perhaps some of the novel X-linked genes will also prove to be sites of mutation in human spermatogenic failure. The stage is set for systematic examination, in both

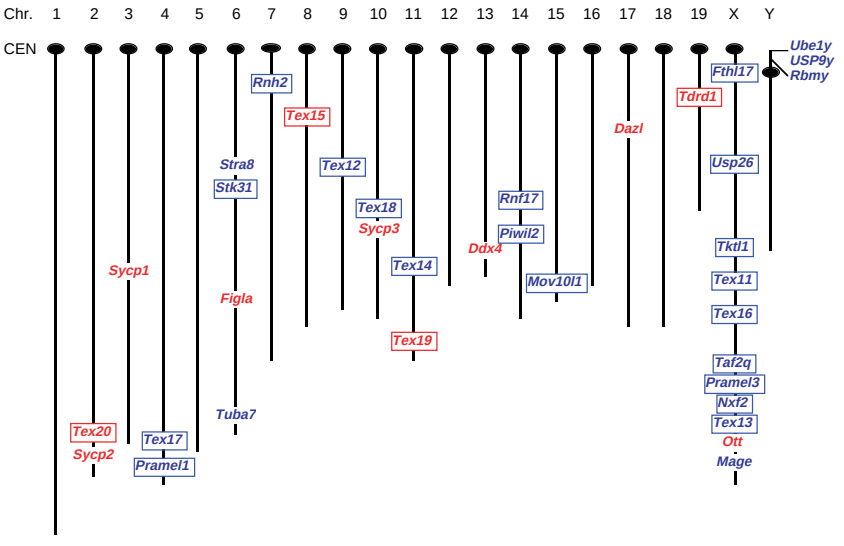

Fig. 2 Chromosomal locations of 36 spermatogonially expressed, germ-cell–specific genes in mouse. Genes that seem to be expressed only in testis are shown in blue; genes expressed in both testis and ovary are shown in red; novel genes are boxed. Eight of the genes (*Sycp3*, *Sycp1*, *Dazl*, *Rbmy*, *Ube1y*, *Usp9y*, *Mage*, *Ott*) were mapped previously (Table 1); all other genes were mapped by radiation hybrid analysis. In the case of gene families residing on a single chromosome, only one family member is shown (for example, *Magea5* is a representative of the X-linked *Mage* family). The Y chromosome is shown in proportion to its estimated physical length[30]; all other chromosomes are drawn on a centiray scale[29].

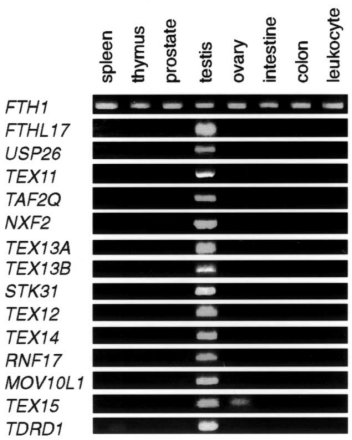

Fig. 3 Expression of orthologous human genes assayed by RT–PCR. *FTH1* (encoding ferritin heavy chain) is expressed ubiquitously and served as a control. All of the novel human genes seem to be expressed in testis; *TEX15* is also expressed in ovary, as is its mouse ortholog (Fig. 1). We also tested eight additional tissues (heart, brain, placenta, lung, liver, skeletal muscle, kidney, pancreas) and detected no expression of the novel genes there (data not shown).

mouse and human, of the postulated role of the X chromosome in early stages of spermatogenesis.

Note: supplementary information is available on the Nature Genetics web site (http://genetics.nature.com/supplementary_info/).

Methods

Isolation of mouse spermatogonia. We isolated spermatogonia by the Staput method of sedimentation velocity at unit gravity[4]. Primitive type A spermatogonia were prepared from testes of 6-d CD-1 mice (Charles River Laboratories). Mature type A and type B spermatogonia were isolated from 8-d CD-1 mice. By microscopic examination, at least 85% of the cells in the resulting preparations were spermatogonia, with no more than 15% somatic cell (Sertoli cell) contamination. The spermatogonial preparations contained no spermatocytes, as spermatocytes are not present in the testes of 6-d or 8-d CD-1 mice[4].

cDNA subtraction. We carried out three independent subtraction experiments, using cDNAs from primitive type A, type A or type B spermatogonia as tracer. In all cases, tracer and driver cDNAs were derived from oligo(dT)-selected RNAs. Germ-cell-depleted testes were from $Kit^{W\text{-}v/W\text{-}v}$ animals. Before subtraction, tracer and driver cDNAs were digested to completion with *Rsa*I. In each of the three experiments, we carried out one round of subtraction using the PCR-select protocol[2] (Clontech). To more thoroughly subtract ubiquitous cDNAs, four additional rounds of subtraction were performed using a modified procedure (D. Menke, pers. comm.) as described[3]. Between rounds of subtraction, we monitored enrichment of *Dazl* cDNA (germ-cell-specific) and disappearance of *Gapd* cDNA (ubiquitous). Three plasmid libraries (one for each of the three independent experiments) were prepared from the resulting pools of subtracted cDNA fragments. We sequenced (one read only) 800 randomly selected clones from each of the three libraries. Of the 2,400 sequences generated, 165 were of poor quality or derived from the cloning vector, leaving 2,235 sequences for further analysis.

Sequence analysis. Of the 2,235 sequence fragments, 409 corresponded to 13 previously reported germ-cell-specific genes (142 to *Mage*, 11 to *Ube1y*, 2 to *Usp9y*, 44 to *Rbmy*, 10 to *Tuba3/Tuba7*, 2 to *Stra8*, 45 to *Ott*, 16 to *Sycp2*, 3 to *Sycp1*, 3 to *Figla*, 8 to *Sycp3*, 21 to *Ddx4* and 102 to *Dazl*). Among the remaining 1,826 sequence fragments, we searched electronically for redundancies and identities to known genes. We found 98 unique, novel sequence fragments that were each recovered at least twice. We tested each of these 98 sequences for germ-cell specificity by RT–PCR on the 14 tissues shown in Fig. 1. Of the 98 sequences, 45 were found to be expressed in spermatogonia and wild-type testis, but not in somatic tissues including $Kit^{W\text{-}v/W\text{-}v}$ testis, indicating that they are germ-cell specific. After full-length cDNA sequences were assembled, these 45 sequence fragments were found to derive from a total of 23 different genes. Of the original set of 2,235 sequence fragments, 546 corresponded to these 23 novel genes (8 to *Fthl17*; 29 to *Usp26*; 38 to *Tktl1*; 66 to *Tex11*; 2 to *Tex16*; 132 to *Taf2q*; 57 to *Pramel3*; 13 to *Nxf2*; 5 to *Tex13*; 4 to *Pramel1*; 3 to *Tex17*; 2 to *Stk31*; 6 to *Rnh2*; 29 to *Tex12*; 4 to *Tex18*; 2 to *Tex14*; 8 to *Rnf17*; 16 to *Piwil2*; 36 to *Mov10l1*; 7 to *Tex20*; 71 to *Tex15*; 6 to *Tex19*; 2 to *Tdrd1*).

cDNA cloning. Full-length mouse cDNA sequences were composites derived from subtracted cDNA clones, 5′- and 3′-RACE products, and clones isolated from conventional cDNA libraries that were prepared from adult testes (Clontech, Stratagene and one library of our own construction). We identified orthologous human sequences by searching GenBank using mouse cDNA sequences. We obtained full-length human cDNA sequences by screening a cDNA library prepared from adult testes (Clontech).

Radiation hybrid mapping. Using PCR, we tested genomic DNAs from the 93 cell lines of the mouse T31 radiation hybrid panel (Research Genetics) for the presence of each gene[28]. PCR conditions and primer sequences have been deposited at GenBank. Analysis of the results positioned the genes with respect to the radiation hybrid map of the mouse genome constructed at the Whitehead/MIT Center for Genome Research[29] (http://www-genome.wi.mit.edu/mouse_rh/index.html). Chromosomal mapping data of human genes were retrieved from GenBank and confirmed by RH mapping using the GeneBridge 4 panel (Research Genetics; data not shown).

Expression analysis. Total RNAs were prepared using TRIzol reagent (Gibco BRL); poly(A)$^+$ RNAs were subsequently isolated using a QuickPrep Micro mRNA purification kit (Amersham Pharmacia Biotech). For each of the 14 tissues shown in Fig. 1, reverse transcription primed with either random hexamers or oligo $(dT)_{18}$ was carried out in bulk, using poly(A)$^+$ RNA (70 ng) from spermatogonia and poly(A)$^+$ RNA (200 ng) from each of the other tissues. RT products were diluted to a final volume of 200 µl, 5 µl of which was used in each PCR amplification. PCR conditions and primer sequences have been deposited at GenBank.

GenBank accession numbers. cDNA sequences for mouse genes: *Fthl17*, AF285569; *Mov10l1*, AF285587; *Nxf2*, AF285575; *Piwil2*, AF285586; *Pramel1*, AF285578; *Pramel3*, AY004873; *Rnf17*, AF285585; *Rnh2*, AF285581; *Stk31*, AF285580; *Taf2q*, AF285574; *Tdrd1*, AF285591; *Tex11*, AF285572; *Tex12*, AF285582; *Tex13*, AF285576; *Tex14*, AF285584; *Tex15*, AF285589; *Tex16*, AF285573; *Tex17*, AF285579; *Tex18*, AF285583; *Tex19*, AF285590; *Tex20*, AF285588; *Tktl1*, AF285571; and *Usp26*, AF285570.

cDNA sequences for human genes: *FTHL17*, AF285592; *MOV10L1*, AF285604; *NXF2*, AF285596; *RNF17*, AF285602 and AF285603; *STK31*, AF285599; *TAF2Q*, AF285595; *TDRD1*, AF285606; *TEX11*, AF285594; *TEX12*, AF285600; *TEX13A*, AF285597; *TEX13B*, AF285598; *TEX14*, AF285601; *TEX15*, AF285605; and *USP26*, AF285593.

Primer sequences and PCR conditions for mouse RH mapping: *Figla*, G65193; *Magea5*, G65194; *Ddx4*, G65195; *Ott*, G65196; *Sycp2*, G65197; *Sycp3*,G65198; *Stra8*,G65199; *Tuba3*, G65200; *Tuba7*, G65201; *Fthl17*, G65202; *Mov10l1*,G65203; *Nxf2*,G65204; *Piwil2*, G65205; *Pramel1*, G65206; *Pramel3*, G65331; *Rnf17*, G65207; *Rnh2*, G65208; *Stk31*, G65210; *Taf2q*, G65211; *Tdrd1*, G65212; *Tex11*, G65213; *Tex12*, G65214; *Tex13*, G65215; *Tex14*, G65216; *Tex15*, G65217; *Tex16*, G65218; *Tex17*, G65219; *Tex18*, G65220; *Tex19*, G65221; *Tex20*, G65222; *Tktl1*, G65223; and *Usp26*, G65224.

Primer sequences and RT–PCR conditions for mouse genes: *Gapd*, G65758; *Fshr*, G65759; *Dazl*, G65760; *Rbmy*, G65761; *Fthl17*, G65778; *Mov10l1*, G65779; *Nxf2*, G65780; *Piwil2*, G65781; *Pramel1*, G65762; *Pramel3*, G65782; *Rnf17*, G65763; *Rnh2*, G65783; *Stk31*, G65784; *Taf2q*, G65785; *Tdrd1*, G65786; *Tex11*, G65787; *Tex12*, G65788; *Tex13*, G65789; *Tex14*, G65790; *Tex15*, G65791; *Tex16*, G65792; *Tex17*, G65793; *Tex18*, G65794; *Tex19*, G65795; *Tex20*, G65796; *Tktl1*, G65797; *Usp26*, G65798.

Primer sequences and RT–PCR conditions for human genes: *FTH1*, G65764; *FTHL17*, G65765; *MOV10L1*, G65766; *NXF2*, G65767; *RNF17*, G65799; *STK31*, G65768; *TAF2Q*, G65769; *TDRD1*, G65770; *TEX11*, G65771; *TEX12*, G65772; *TEX13A*, G65773; *TEX13B*, G65774; *TEX14*, G65775; *TEX15*, G65776; *USP26*, G65777.

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