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Original Citation: Availability: This version is available at: 11577/2458825 since: 2022-01-25T17:52:18Z Publisher: BIOINFORMATICS INST, UNIV AUCKLAND, PRIVATE BAG, AUCKLAND, 00000, NEW ZEALAND Published version: DOI: Terms of use: Open Access This article is made available under terms and conditions applicable to Open Access Guidelines, as described at http://www.unipd.it/download/file/fid/55401 (Italian only)

## Characterization and Evolution of the Cell Cycle-Associated Mob Domain-Containing Proteins in Eukaryotes

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Abstract: The MOB family includes a group of cell cycle-associated proteins highly conserved throughout eukaryotes, whose founding members are implicated in mitotic exit and co-ordination of cell cycle progression with cell polarity and morphogenesis. Here we report the characterization and evolution of the MOB domain-containing proteins as inferred from the 43 eukaryotic genomes so far sequenced. We show that genes for Mob-like proteins are present in at least 41 of these genomes, confirming the universal distribution of this protein family and suggesting its prominent biological function. The phylogenetic analysis reveals five distinct MOB domain classes, showing a progressive expansion of this family from unicellular to multicellular organisms, reaching the highest number in mammals. Plant Mob genes appear to have evolved from a single ancestor, most likely after the loss of one or more genes during the early stage of Viridiplantae evolutionary history. Three of the Mob classes are widespread among most of the analyzed organisms. The possible biological and molecular function of Mob proteins and their role in conserved signaling pathways related to cell proliferation, cell death and cell polarity are also presented and critically discussed.

Keywords: Mob genes, protein structure, phylogenesis, cytokinesis, apoptosis, morphogenesis

## Introduction

Normal development of multicellular organisms requires appropriate cell numbers and organ sizes, and it is determined by coordinated cell proliferation, cell growth and programmed cell death (reviewed by Danial and Korsmeyer, 2004; Murray, 2004; Sherr, 2004). Disruption or malfunction of these processes can cause diseases, such as cancer. Recent studies in yeasts and higher eukaryotes have led to the identification of a number of proteins and their interactors as key components of specific metabolic pathways that control the coordination between cell proliferation, morphogenesis and programmed cell death (Lai et al. 2005).

Members of the NDR (nuclear Dbf2-related) family, a subclass of AGC-type protein kinases, are essential components of pathways that control important cellular processes, such as mitotic exit, cytokinesis, cell proliferation and morphogenesis, and apoptosis (reviewed by Hergovich et al. 2006). Some recent progress in this field has shed light on the mechanisms that underlie the regulation and function of the NDR proteins by means of the co-activator Mob (Mps1-one binder) proteins. Combined data from yeast, worms, flies, mice and human cells have highlighted the conserved and important roles of MOB-domain containing proteins in the activation of NDR kinases (Manning et al. 2002; Hergovich et al. 2006). In particular, Mob proteins play a critical role in cell-cycle regulation chiefly by interacting with and activating the Dbf2-related protein kinases (Komarnitsky et al. 1998; Lee et al. 2001; Mah et al. 2001). This subfamily of serine/threonine kinases includes Dbf2, Dbf20 and Cbk1 in *Saccharomyces cerevisiae*, Ndr1, Ndr2, Lats1 and Lats2 in human, Warts (aka dLats) and Trc (aka dNdr) in *Drosophila melanogaster* and Sax1 (aka ceNdr) and a hypothetical Lats homolog in *Caenorhabditis elegans*. Like their Mob protein partners, this subfamily of protein kinases regulates cell growth, cell

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division and cell morphology (Justice et al. 1995; Xu et al. 1995; Zallen et al. 2000). In metazoans, members of the NDR family act as tumour suppressors (for example, LATS1) or potential protooncogenes (for example, NDR1). In the molecular regulation of the NDR family kinases, an important role is also played by protein kinases belonging to the sterile 20 (STE20)-like kinase group (for review see Hergovich et al. 2006). A summary of available information on Mob-domain containing proteins and its interacting NDR-type kinases in given in Table 1.

Mob proteins interact with NDR kinases by binding a conserved stretch of primary sequence at their N terminus, also known as NTR (N-terminal regulatory) domain. The interaction of Mob proteins with the NTR activation site is a conserved feature of all members of the NDR-kinase family that have been tested so far in yeasts, flies and human cells (Mrkobrada et al. 2006). Interestingly, Mob proteins do not function solely as co-activators of NDR kinases, but are also required for the localization of yeast NDR kinases. Recent evidence further indicates that the targeting of Mob proteins to the plasma membrane is sufficient to fully activate mammalian NDR1/2 (Hergovich et al. 2005; Stegert et al. 2005) and LATS1 (Hergovich et al. 2006). Taken together, these findings indicate that Mob binding to the N terminus of NDR family members allows efficient auto-phosphorylation on the activation segment, and at the same time recruits NDRs to activation sites, thereby bringing this protein into close proximity with its upstream activating kinase.

The MOB family includes a group of cell cycle-associated, non-catalytic proteins highly conserved in eukaryotes, whose founding members are implicated in mitotic exit and co-ordination of cell polarity with cell cycle progression (Luca et al. 2001; Stegmeier et al. 2002). Two distinct Mob proteins, Mob1 and Mob2, are known in fungi, while an expansion in metazoans gives rise to six in human, four in D. melanogaster, and four in C. elegans (Mrkobrada et al. 2006). Mob1 proteins have been demonstrated to be important for both mitosis completion and cell plate formation in yeast (Luca and Winey, 1998; Salimova et al. 2000). Moreover, the Mob1-related proteins Mob2 physically associates with specific kinases throughout the cell cycle, being required and periodically activated in yeast to promote polarized growth (Weiss et al. 2002; Nelson et al. 2003). Mob1-like proteins have been also found in animals (Stavridi et al. 2003; Ponchon et al. 2004; Devroe et al. 2004). Plant genomes such as alfalfa, rice and *Arabidopsis* contain uncharacterized Mob1-related genes (Van Damme et al. 2004; Citterio et al. 2005, 2006). Although there are data to suggest that Mob proteins act as kinase activating subunits in higher eukaryotes, their function remains to be proved.

This paper deals with the characterization and evolution of the cell cycle-associated and morphogenesis-related MOB domain-containing proteins belonging to 43 eukaryotic genomes. Results on the structural characteristics and phylogenesis of Mob proteins are reported, and adopted for the classification of family members using a novel nomenclature. The biological and molecular function of Mob proteins and their role in conserved signaling pathways related to cell proliferation, cell death and cell polarity are also presented and critically discussed.

## **Methods for Bioinformatic Analyses**

To perform a complete and exhaustive analysis on the Mob domain distribution and phylogenetic relationship among eukaria, the proteomes of 43 complete and ongoing eukaryotic genomes were downloaded from NCBI (ftp://ftp.ncbi.nih.gov/ genomes/), ENSEMBL (ftp://ftp.ensembl.org/pub) and DOE Joint Genome Institute (http://genome. jgi-psf.org/euk home.html) sites.

The hidden Markov model profile for the Mob domain (Pfam code: PF03637) was downloaded from the Pfam site (http://www.sanger. ac.uk/Software/Pfam/) (Sonnhammer et al. 1998) and was used to search for similarity against the proteome databases using HMMER software (Durbin et al. 1998).

Using a cut-off expectation value equal or lower than  $e^{-20}$ , a total of 202 MOB domain containing proteins were identified (see supplementary Table 1S). Among these, ten sequences were not considered in the subsequent analysis because of low quality problems. As many as 192 Mob domains were extracted from the original sequences and aligned using the progressive alignment algorithm implemented in CLUSTALW (Higgins et al. 1992), and the result was edited to remove any ambiguous region.

The ProtTest software (http://darwin.uvigo.es/) (Abascal et al. 2005) was used to select the most

lable 1. Summary	of available d	ata on Mob-don	nain containing proteins and its interacting NDI	R-type kina	ases (see tootnotes tor mai	in Keterences).
Organism	Protein name	Accession	Description/Function	Group	Subcellular localization	Interacting kinases
Saccharomyces cerevisiae <sup>1</sup>	Mob1p	NP_012160	Component of the MEN: regulates mitoric exit and cytokinesis	·	Spindle pole body and bud neck	Dbf2p-Dbf20p
	Mob2p	NP_116618	Component of the RAM signaling network: links cell morphology changes with cell cycle progression	ı	Nucleus, cytoplasm and cortex	Cbk1p
Schizosacchar- omvces pombe <sup>2</sup>	Mob1p	NP_595191	Component of the SIN: controls septum initiation and cvtokinesis	ı	Spindle pole body and mitotic septum	Sid2p
	Mob2p	NP_587851	Required for maintenance of cell polarity: coordinates cell morphogenesis with cell cycle progression	·	Mitotic septum	Orb6p
Caenorhabditis elegans <sup>3</sup>	1 1	NP_510184 NP_502248 ND_402700	F09A5.4c F38H4.10	ı . C		
		NP_501179	C30A3.3 T12B3.4	04		
Drosophila melanogaster <sup>4</sup>	dMob1	NP_729716	CG11711-PB. Mob1, isoform B	7		Trc (dNDR)/ Warts (Lats)
	Mats	NP_651041	CG13852-PA. Mob as tumor suppressor	~		Trc (dNDR)/ Warts (Lats)
	dMob3 dMob4	NP_609364 NP_610229	CG4946-PA CG3403-PA	4 0		
Homo sapiens <sup>5</sup>	hMOB1	NP_775739	MOB-KL1A, MOB kinase	<del></del>	Nucleus, cytoplasm	LATS1/2 (low affinity for NDR1/2)
	MATS1	NP_060691	MOB-KL1B, MOB kinase activator-like 1B (MOB1B)	<del></del>	Centrosome, poles of mitotic spindle	LATS1
	hMOB2	NP_443731	HCCA2 protein	7	Nucleus, perinuclear region and eventsem	NDR1/2
	hMOB3A	NP_955776	PREI3, preimplantation protein (Phocein)	ი	Perinuclear region, membrane	PP2A
	hMOB3B hMOB3C MOB-LAK	NP_079037 NP_958805 NP_570719	MOB-KL2B, MOB kinase activator-like 2B MOB-KL2C, MOB kinase activator-like 2C MOB-LAK, metal ion binding	4b 4b b	Intracellular	
Arabidopsis thaliana <sup>6</sup>	Mob1A Mob1B	NP_199368 NP_193640	Similar to yeast Mob1p Mob1-like domain containing protein	م د	Nucleus	
5	Mob2A Mob2B	NP_197544 NP_197543	Similar to yeast Mob2p Similar to yeast Mob2p		Nucleus Fragmoplast	

The thrifty phenotype: a review of evolutionary models

Organism	Protein name	Accession	Description/Function	Group	Subcellular localization	Interacting kinases
Medicago sativa <sup>7</sup>	Mob1A Mob1B	CAC41010 CAG25780	Similar to yeast Mob1p Similar to yeast Mob1p	م م	Cytoplasm and cell plate	
Trypanosoma hrucei <sup>8</sup>	Mob1A	AAL10512	Mob1-1 essential for cytokinesis	ı	Cytoplasm	tbPK50 (functional homolog of Orb6)
222	Mob1B	AAL10513	Cell cycle associated protein Mob1-2	ı		
<sup>1</sup> Luca et al. (1998); Luc <sup>2</sup> Verde et al. (1998); Sa <sup>3</sup> No references	a et al. (2001); K limova et al. (200	omarnitsky et al. (1: 00); Hou et al. (2005	998); Mah et al. (2001); Stegmeier et al. (2002); Weiss et al 3; 2004).	II. (2002); Ma	h et al. (2005); Stoepel et al. (2005	5).
<sup>4</sup> Geng et al. (2000); He <sup>5</sup> Moreno et al. (2001); E <sup>6</sup> VanDamme et al. (200 <sup>7</sup> Citterio et al. (200 <sup>8</sup> Hammatron et al. (2005); C	et al. (2005); Lai Sichsel et al. (200 4); Barcaccia et a itterio et al. (200	i et al. (2005). )4); Devroe et al. (2' al. (unpublished). 6).	004); Hergovich et al. (2005); Lai et al. (2005); Bothos et al.	. (2005); Her	govich et al. (2006).	
	<u>.</u>					

The phylogenetic analysis allowed the identification of different Mob groups. The proteins belonging to different branches of the phylogenetic tree were aligned using CLUSTALW software and a consensus sequence was extracted for each group. The consensus sequences reflect the most common sequences in the alignment. For a more detailed analysis and visualization of each aligned group, a web logo was created using the web version of WebLogo software (http://weblogo.berkeley.edu).

## Results: Structural Analysis of Mob Proteins

### Primary structure characteristics and classification of family members

Mob proteins are a small family of highly conserved proteins, found in all eukaryotes, approximately 210 to 240 amino acid residues in length. The evolution of MOB family genes is poorly understood and a classification and nomenclature of Mob genes is not fully established. Here we propose some insight into the evolutionary dynamics of this family and a system of classification based on a phylogenetic analysis of Mob genes in all complete and ongoing eukaryotic genome sequences.

Mrkobrada et al. (2006) proposed a classification based on the alignment of the core domain of Mob proteins from yeast to human, identifying three distinct groups defined by similarity between the conserved N-terminal region. On the basis of the distribution of ScMob1 and ScMob2 members within the clusters, they referred to the groups as Mob1-like, Mob2-like and Mob3-like. The Mob1-like group contains two subgroups (A and B): Mob1A contains the ortholog of ScMob1 in fungal species and single proteins from H. sapiens and D. melanogaster, whereas the Mob1B group contains one or more Mob proteins from *H. sapiens*, *D. melanogaster*, D. rerio, C. elegans and X. laevis. The Mob2-like cluster contains two groups, Mob2A, consisting of the fungal ortholog ScMob2 and a second group, Mob2B, containing metazoan genes.

Table 1 (Continued)

Finally, the Mob3-like group is the most divergent one and contains a single protein from each metazoan organism analyzed. Moreover, two mammalian homologs to yeast MOB genes have been described, the mammalian Mob homolog (MMh), that has high similarity with *S. cerevisiae* Mob2 genes, and phocein or mammalian Mob1 distantly related to MOB1 and MOB2 (Hennebold et al. 2000; Baillat et al. 2001; 2002; Moreno et al. 2001). Stavridi et al. (2003) proposed that MMh be referred to as Mob2 and that phocein/mMob1 be referred to only as phocein.

To classify the Mob domain into related groups of sequences, a phylogenetic analysis was performed, by searching Mob domain hidden Markov model profile on all complete or ongoing available eukaryotic genomes. Figure 1 shows the phylogenetic tree for 192 Mob genes (see also supplementary Figure 1S). The results highlight that Mob domain is clearly separated into five classes: Mob1, Mob2, Mob3, Mob4 and Mobp with high bootstrap support. Among the different classes, Mob3 is the most divergent clade.

The numbers of genes in class Mob1, Mob2, Mob3, Mob4 and Mobp are 47, 28, 31, 57 and 14 respectively. Some of the *C. elegans* and *C. briggsae*, and *S. cerevisiae*, *S. pombe* and Protist Mob related proteins clustered outside these groups and they will be treated separately. Mob4 class can be subdivided into two phylogenetic clades, corresponding to invertebrate (9 genes) and vertebrate Mob-like genes (48). Moreover, vertebrate Mob-like genes can be further subdivided into other two subgroups, Mob4a, containing 19 genes, and Mob4b with 29 Mob-like proteins.

The average amino acid identity within Mob classes is 92% (Mob1), 54% (Mob2), 86% (Mob3), 70% (Mob4), 86% (Mob4a), 84% (Mob4b) and 78% (Mobp).

The results partially support the previous classification by Mrkobrada et al. (2006). The main differences are probably due to the higher number of genes analyzed in this study and concern the Mob1 class which was previously subdivided into two groups, Mob1A and Mob1B. Our analysis allowed us to recognize a Mob1 class that corresponds to Mob1A group and a Mob4 class that contains the previously established Mob1B group (see Mrkobrada et al. 2006). Moreover, both Mob4a and Mob4b groups proved to contain Mob-like genes previously annotated as part of the Mob1B group (Mrkobrada et al. 2006).

### Phylogenesis: Distribution and Evolution of Mob Genes in Eukaryotic Genomes

The phylogenetic tree shown in Figure 1 has been generated from the available proteomes of 43 complete and draft genomes (see also supplementary Figure 1S). Only in two plant genomes, *Ostreococcus tauri* and *Zea mays*, it was not possible to identify Mob-like proteins. This could be due to the consensus sequence quality and to the genome assembly; both of them being quite important issues for producing a high quality alignment and a reliable counting of Mob genes.

Figure 2 shows the distribution of Mob-like proteins among the organisms used for the analysis. Vertebrates (mammals, birds, amphibian and fish) have the highest number of Mob genes, distributed in all the Mob classes. Interestingly, all the vertebrate genes of the Mob4 class are included in a single branch that is supported by a bootstrap value of 77%. This suggests that all Mob4-like vertebrate genes derived from a single ancestral gene at the basis of Mob4 chordata/hemichordata gene evolution. The two subclasses Mob4a and Mob4b found in vertebrates must have arisen from an early duplication, which further subdivided this class into two subgroups.

Among vertebrates, mammals reveal the highest number of Mob genes. *M. musculus* have the highest number of Mob4b genes (4), while *P. troglodytes* and *R. norvegicus* have the highest number of Mob1 genes (4). *L. africana*, *O. cuniculus* and *S. scrofa*, compared to the other mammals, present a smaller number of Mob genes, probably reflecting a still limited coverage of the entire gene space of these organisms.

Mrkobrada et al. (2006) reports that the *Homo* sapiens genome contains six Mob-like proteins whereas in our analysis we found seven Mob-like proteins. Nomenclature of Mob genes not only is poorly established but often can be quite misleading. Proteins identified by codes NP\_060691 and NP\_775739 are annotated as "Mob4B" and "MOB1, Mps One Binder kinase activator-like 1A" respectively, while in our phylogenetic tree they both fall in Mob1 group. NP\_443731 is a member of the Mob2 group but it is annotated as "HCCA2 protein". Moreover



**Figure 1.** Phylogenetic tree of the 192 Mob domain proteins. Mob groups identified with the phylogenetic analysis are shown and highlighted in different colors. The Panel **A** shows a maximum likelihood Mob protein phylogenetic tree (the scale represents the number of amino acid substitution per site). The Panel **B** shows a maximum likelihood cladogram without branch length for an easier visualization of the Mob groups (the colored dot on each organism name refers to the taxonomy classification). The red dot on each node of the tree represents a bootstrap value equal or higher than 50%, while the blue dot a bootstrap value equal or higher than 70%.

protein NP\_955776 in public databases is defined as "preimplantation protein 3 isoform 2" and in our analysis belongs to the Mob3 group. Finally, NP\_958805, NP\_079037, NP\_570719 proteins, annotated respectively as "MOB1, Mps One Binder kinase activator-like 2C isoform 2", "MOB1, Mps One Binder kinase activator-like 2B" and "MOB-LAK", are all members of the Mob4 group, with the first one belonging to Mob4a and the last two to Mob4b group.

All insects show four Mob genes belonging respectively to Mob1, Mob2, Mob3 and Mob4 classes, except *D. pseudoobscura*, in which only two Mob genes can be found, probably due to genome assembly quality. Finally, plants represent a monophyletic group defined as Mobp class.

The phylogenetic tree shows that *S. cereviseae* (NP\_012160, NP\_116618), *S. pombe* (NP\_595191, NP\_587851), *C. elegans* (NP\_502248, NP\_510184), *C. briggsae* (CAE62136, CAE61392) and Protist proteins are listed as *incertae sedis*. Because of historical reasons, in the previous literature Mob yeast genes have been generally described as the founding members of the Mob family (Stavridi et al. 2003, Mrkobrada et al. 2006). However, the protein sequences analyzed in this work, mostly of multicellular organisms, do not allow a clear definition of the phylogenetic relationships existing

among the yeast and the other Mob genes. In this regard it is interesting to point out that NP\_116618 and NP\_587851 yeast proteins, described as Mob2A in Mrkobrada et al. (2006), did not cluster with any other protein, possibly due to an early divergence of these orthologs in the lineage that generated modern Fungi.

Even if it is quite difficult to reconstruct the evolution of the Mob family as a whole, some possible scenarios can be drawn by looking at the distribution of genes in the so far sequenced organisms. If plants are not considered, Figure 2 indicates a minimum of two genes in all the eukaryotic genomes analyzed. This in turn seems to suggest a duplication of the ancestral Mob gene at an early stage of the eukaryotic evolution.

Going from unicellular to multicellular organisms there is a progressive expansion of the Mob family, reaching the highest number in mammals. Moreover, plant Mob-like genes appear to have evolved from a single ancestor, most likely due to the loss of one or more genes during the early evolution of Viridiplantae. Compared to vertebrates, plants show a significant decrease in Moblike gene possibly due to the adaptation to a much more simple life style. The relationship observed among genes of the same organism and/or different organisms suggests that the Mob gene family



Figure 2. Mob protein distribution among organisms used in the analysis. Different Mob groups are represented with different color and the species grouped on the base of the taxonomy classification. The label "incertae sedis" refers to Mob proteins that have an undefined position on the phylogenetic tree.

evolved under a birth-and-death type of evolution. In this model new genes are created by duplication, and some duplicated genes are maintained in the genome for a long time whereas other are deleted or become nonfunctional through deleterious mutations (Nei and Rooney, 2005).

## Mob-like Protein Structure and Architecture of Mob-domain Containing Proteins

Three Mob1 protein structures have been described in literature. Human and *Xenopus laevis* structures correspond to the most conserved C-terminal core but lack the variable N-terminal region, whereas *Saccharomyces* Mob1 structure contains both the conserved C-terminal core and the variable N-terminal region (Stavridi et al. 2003; Ponchon et al. 2004; Mrkobrada et al. 2006).

In our phylogenetic tree, Human and *Xenopus* proteins used in structure analyses belong to the Mob1 group, while *Saccharomyces* Mob-like proteins have been assigned as *incertae sedis*.

To compare the different Mob classes, a consensus sequence for each identified group was constructed. Figure 3 shows the amino acid sequence conservation over all positions for each of the seven Mob groups: Mob1, Mob2, Mob3, Mob4, Mob4a, Mob4b and Mobp. These consensus sequences were then adopted to generate a new multiple protein alignment, using three additional Mob proteins, such as the S. cerevisiae Mob1 and Mob2 proteins (NP 116618 and NP 012160) and one H. sapiens Mob1 protein (NP 775739). The latter two proteins were added in the alignment since they have been structurally characterized (Stavridi et al. 2003; Mrkobrada et al. 2006). The final multiple alignment of Mob group consensus sequences in shown in Figure 4.

Mob proteins are approximately 210 to 240 amino acid residues in length, with the exception of *S. cerevisiae* Mob1, which has a further 78 residue N-terminal extension not conserved or even present in the closely related fungal proteins.

Mob1 adopts a globular structure consisting of seven  $\alpha$  helices, two 3<sub>10</sub>-helices and a  $\beta$  hairpin. The core of the structure consists of a helical bundle formed by four long  $\alpha$  helices (H2, H4, H5, and H7). This left-handed four-helix bundle, comprising the H2 and H5 helices running anti-parallel to H4 and H7 helices, is capped at one end by two short helices (H3 and H6) and the  $\beta$  hairpin, which are stabilized Mobi NAF SCHWINE STELANDELEN I VERBUELWINEN IL TETLAMAR EN NAMENA DUTVOLDELENT VERBUELWINEN INFORMATINE FREI NAME, Mobi2

LIRIPETRATE DEGETERSTANNI I AL RASSIAR LEPERCENTREL REDELITANA GARTSOTTENTE Lirikere (Pldtintonallink Prinskesnalcikik: Renderradegeteratiskinikol I Pleevinisera

Mob4

Mob3

aug Fleffen ik Kitfferkoft af Rislikoa af skudir alloft. <mark>Buldina hividfeir i Lukituse Asekterikoanse re</mark> Etiliade u Kiet Leven Juldine eilber offisien fereneffen f. Leven Lukier (Physiolistic eile Easterikoen flefve

#### Folis (HE) Mob4a

NULKOFSKATRENGER TRELINAALISLULEVISLEEDINAAMINEERILIITINEESE RANATOEEN Gerliefskake Naliotel informer sommonen villenen vienelen vienelen vienelenen Defler Modeld

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## NGLELAS FROM FRYNOAR SCHOLLANI DATUSSIN DE ANDER VELLEN I TVEFROMSLASTLÆFTE A DE JEMISTER FRA Adven konstant vellen de Alde, i Rom Gaffelf, enkender Fri Mini Haffer i velgehliff Roffelf, ef Lig Kalena

**Figure 3.** Sequence logos for each of the multiple alignment Mob groups: Mob1, Mob2, Mob3, Mob4, Mob4a, Mob4b and Mobp. Each logo consists of stacks of symbols, one stack for each position in the sequence: the overall height of the stack indicates the sequence conservation at that position, while the height of symbols within the stack indicates the relative frequency of each amino acid at that position. The yellow arrows represent the starting position adopted for the multiple alignment of Mob group consensus sequences.

On one side, the structure has a flat surface consisting of H1 and H2 and parts of H3, H4, H6, and H7. Stavridi et al. (2003) reports that most of the conserved residues of Mob family members map to parts of the flat surface formed by H2 and two loops, L1 and L2, adjacent to the N-terminus of H2. Loop L1 in human Mob protein goes from residues 46 to 51 and Leu47 and Pro48 are highly conserved since are needed to stabilize the structure of the loop. These results are confirmed in our analysis, with the exception of position 47 in Mob3 consensus sequence where a Pro is present. Moreover, Stavridi et al. (2003) reports that Glu51 is conserved only in Mob1 family. Figure 4 shows that Glu51 is conserved in Mob1 and Mob4 consensus sequences, while in Mob2 sequence is replaced by an isoleucine and in Mob3 by a glutamine.

The L2 loop, consisting of residue 128–142, presents several highly conserved amino acids involved in structural interaction, such as Pro133 and Pro141 and Phe132 and Phe140 that, together with Phe144 from H5, form hydrophobic interactions with each other and with Ala58 and Ile151 from H2 and H5, respectively. Figure 4 shows that all these positions are conserved, except for Mob3 where various non-conservative amino acid changes can be seen in the consensus sequence (Phe140 $\rightarrow$ Glu140, Phe144 $\rightarrow$ Val144, Ala58 $\rightarrow$  Tyr58). Moreover, the Mob3 consensus sequence is missing the amino acid in position 141.

Helix H2 has a large number of conserved residues, several of which have solvent exposed negatively charged side chains. While Stavridi et al. (2003) report that Asp52 is the only charged conserved residue in all Mob families, in our analysis we found that in Mob4 and Mob4a there is an amino acid conservative substitution Asp $\rightarrow$ Asn. Moreover, we observed that Glu55, that makes a hydrogen bond with Glu51, is conserved in Mob1, Mob2 and Mobp groups while Mob4 contains aspartate and the consensus sequence

	30	40 H]	L1 50	60 H2	70	H3 <sup>80</sup>	90	100	110 H4
HsNP_775739 Mob1 ScNP_116618	KHAEATLGSG KHAEATLGSG PFVRTALVKG	- NLRMAVML - NLRQAVML - SFKTIVQL	PEGEDLNEWV PEGEDLNEWI PKYVDLGEWI	AVNTVDEFN AVNTVDEFN ALNVFEFFT	Q INMLYGTITD Q INMLYGTITE N LNQFYGVVAE	FCTEESCPVM FCTEESCPVM YVTPDAYPTM	SAGPKYEYHW SAGPKYEYHW NAGPHTDYLW	ADGTNIKKPI ADGTNIKKPI LDA – NNRQV	KCSAPKYIDY KCSAPKYIDY SLPASQYIDL
ScNP_012160 Mobp (plants) Mob4	QIVEMTLGSE KHIDATLGSG KQAEASLQSG	G VLNQAVKL - NLREAVKL	PRGEDENEWL PPGEDINEWL PPGENLNDWL	AVHCVDFYN AVNTVDFFN AVHVVDFFN	Q INMLYGSITE Q VNLLYGTLTE R INLIYGTISD	FC SPQTCPRM FC TP SNC PTM YC TEQTCPTM	AG-PGTQYW IATNEYEYLW TAGPKYEYRW SGGSRYEYLW	AFQ-KGQPPV ADGVTIKKPI ADGEEYKKPT	KCTAPQYTDF SVSAPKYVEC EVSAPKYVEY PLPAPKYTEL
Mob4b Mob4a Mob3	KKAQAS LKSG QY I QQN I RAD		P P G E N I D D WI P E G Q D E G V W K	AVHVVDFFN	R INLIYGTMSE	FCSETSCPVM	AGGPRYEYRW	QDERQYKRPA AAHKTPK	KLSAPRYMAL ECPAIDYTRH
Consensus	KHAEATLGSG	NLREAVKL	PEGEDLNEWL	AVHTVDFFN	Q INLLYGTITE	FCTEETCPTM	SAGPKYEYLW	ADG-KIKKPI	KCSAPKYIDL
Conservation		nh_nl_l				Than Ta		da_lah	
	120 H4	<sup>130</sup> L2	140	150 H	5 160	H6 170	180	H7 190	200 H8
HsNP_775739 Mob1		DETLEPSKIG		VAKTILKEL	E RVYAHIYHQH		AHLNTSFKHF		
ScNP_116618 Mob2	ALTWINNKVN VMSSVOKLVT	DKNL FPTKNG DEDIFPTKYG		DVQR IMVQM	F RIFAHIYHHH	EDKIVHLSLE EKETLALELH	AHWNSFFSHF GHLNTLFAHF		KIIDRKEMAP NLLDPKETSI
ScNP_012160 Mobp (plants)		DESLEPSKVT DETIEPQKLG	GTEPEGEIQR APEPPNEKD	VIQPILRRL VVKTIFKRL	F RVYAHIYCHH F RVYAHIYHSH	FORILELNLQ	TVLNTSFRHF AHLNTCFKHF		ELLRPADFGP RLIDKKELAP
Mob4 Mob4b	LMDWIEAQIN	NEAL FPVSTD	VPEPKTEIQ- TPEPKNELQ-	ICKKILTRL	F RVFVHVYIHH F RVFVHVYIHH	FDRIVEIGAE FDRVIQMGAE	AHVNTCYKHF AHVNTCYKHF	YYFVTEF YYFVTEF	DLISAKELEP NLIDRKELEP
Mob4a	LMDWIEGLIN	DEDIFPTRVG	VPEPKNEQQ	VCTKILTRL	F RVFVHVYIHH	FDS I L SMGAE	AHVNTCYKHE	YY FIREF	SLVDHRELEP
Consensus	LMDWIEDQLN	DEDLFPSKVG	VPFPKNFIQ-	VCKKILKRL	F RVFAHIYHHH	FDEILELGAE	AHLNTCFKHF	ILFVQEF	NLIDRKELAP
Conservation			alla La			In-non-non			
	H9 210								
HsNP_775739	QELIEKLGS	K							
ScNP_116618	LPLIESFEK								
ScNP_012160	LELVMELRD	R							
Mobp (pidins) Mob4	LADMTSRICK								
Mob4b Mob4a	REMTERICH								
Mob3 Consensus		A							
Conservation		Tener							

**Figure 4.** Multiple alignment of Mob group consensus sequences. The alignment was performed taking into consideration two structural defined Mob proteins, Hs NP\_775739 and Sc NP\_012160 plus Sc NP\_116618. The helix (yellow lines) and loops (black lines) nomenclature and position on the alignment refer to Hs Mob protein as described by *Stavridi* et al. (2005). On each of the alignment columns, a colour scale going from red to blue represents high and low amino acid conservation, respectively.

of Mob3 contains a valine. Asp63 interacts with His185, that is conserved in all Mob consensus sequences except for Mob3 that contains a lysine. Interestingly, Asp63 is conserved in all Mob4, Mobp and Mob1 classes, but it is replaced by a threonine in Mob2 and by a glutamine in Mob3.

Towards the C-terminal of helix H2 there is Asn69, the only polar residue other than tyrosines, that is conserved in all members of the Mob family. H2 also has several hydrophobic residues that are conserved to varying degrees in members of the Mob protein family: notably, Trp56 and Phe64, which should have buried side chains and participate in hydrophobic interactions that stabilize the protein fold, are conserved in all Mob consensus sequences.

A Zn binding site appears to be conserved in all Mob classes, with a peculiar exception in fungi. Considering human Mob1 protein as a reference, the Zn binding site is composed by Cys79 and Cys84 from loop connecting H3 to the first strand of the  $\beta$ hairpin and His161 and His166 from H5 (Stavridi et al. 2003). The presence of the Zn atom contribute to the stability of the structure by anchoring H3 to the C terminus of H5. As reported in Mrkobrada et al. (2006) most of the yeast genes previously described as Mob2A apparently lack the Zn binding site, since the two cysteines are substituted with a valine and a tyrosine respectively, suggesting an alternative structural element for stability compensations. The consensus sequences alignment confirms these observations with the S. cerevisiae NP 116618 as the only Mob protein lacking the Zn binding site (Fig. 4). To make sure that this observation was not due to a consensus artefact, we analyzed the complete 192 Mob-like protein multiple alignment (see supplementary Figure 1S) and we found that essentially all the proteins analyzed contained a well conserved Zn binding site. The only exceptions, found in *M. musculus* XP 001000051, S. purpuratus XP 001185390 and M. mulatta XP 001108825, are probably due to bad quality sequences producing an unreliable alignment in the region that contains His161 and His166.

## **Biological Roles of Mob Proteins and Conserved Signaling Pathways**

Cell cycle progression and cytokinesis The involvement of Mob proteins in cell proliferation was first suggested by Luca and

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Winey in 1998. They demonstrated that Mob1 is an essential yeast gene required for the completion of mitosis and maintenance of ploidy, as yeast Mob1 mutations resulted in a late nuclear division arrest at restrictive temperature. Following studies better elucidated the biological role of this protein in budding and fission yeasts. In Saccharomyces cerevisiae Mob1p is an essential regulator of the localization and activity of Dbf2 protein kinase, a component of the mitotic exit network (MEN). MEN is a GTPase driven signaling network that co-ordinates exit from mitosis with cytokinesis (Fig. 5). It promotes the inactivation of the mitotic Cdk1-cyclin B complex and drives mitotic exit by leading to the release from the nucleolus and subsequent activation of the Cdc14p phosphatase during anaphase (Luca et al. 2001; Stegmeier and Amon, 2004). Although inactivation of Cdk1-cyclin B complex is required for cytokinesis, the MEN was shown to be essential for cytokinesis, and in particular for actomyosin ring contraction and septum deposition, also independently of its role in mitotic exit. In fact, when MEN function is abrogated in conditions where mitotic exit is allowed by artificial suppression of mitotic CDK activity cytokinesis does not take place (Shou et al. 1999; Lippincott et al. 2001; Park et al. 2003).

In S. pombe cytokinesis is regulated by a signaling cascade termed the septation initiation network (SIN). It is organized similarly to the MEN but is not involved in mitotic exit (reviewed by Simanis, 2003; Krapp et al. 2004; Wolfe and Gould, 2005). In S. pombe Mob1 is part of the SIN and interacts with Sid2, the ortholog of S. cereviasae Dbf2, regulating its localization and kinase activity. Nevertheless, how Mob proteins can regulate kinase activity is still under investigation. By analyzing the NMR or X-ray crystal structures of S. cerevisiae, X. laevis and human Mob1p, it has been proposed that Mob proteins may regulate their target kinases through electrostatic interaction mediated by conserved charged surfaces. It seems that the negatively charged surface on MOB proteins interacts directly with the positively charged basic-hydrophobic N terminus of their target kinases Dbf2/Sid2, inducing a conformational change which enable the upstream kinase Cdc15/Cdc7 to phosphorilate and thereby stimulate DBf2/Sid2 activity. In this regard, MOB proteins may functionally resemble cyclins (Stavridi et al. 2003; Ponchon et al. 2004; Mrkobrada et al. 2006). However, yeast Mob1 proteins do not function



Figure 5. Components of the mitotic exit network (MEN) and septation initiation network (SIN) in yeasts (Saccharomyces cerevisiae and Schizosaccharomyces pombe), and of the MEN-like network in human cells. Exit from mitosis and co-ordination with cytokinesis is driven through a GTPase signaling network, where Mob1p is an essential regulator of the localization and activity of Dbf2 and Dbf2-like (Sid2 and Lats1) protein kinase. The network promotes the inactivation of the mitotic Cdk1-cyclin B complex and drives mitotic exit by leading to the release of the Cdc14p phosphatase from the nucleolus and its subsequent activation during anaphase.

solely as activators of Dbf2/Sid2, but are also required for Dbf2/Sid2 localization to activation sites (Frenz et al. 2000; Lee et al. 2001). It has been extensively reported that, in agreement with their functions in mitosis exit and cytokinesis, Dbf2/ Sid2-Mob1 complexes localize to the spindle pole body (SPB) in anaphase and move to the division site in late mitosis (Stegmeier and Amon, 2004). Nevertheless, it must be underlined that the function of Dbf2/Sid2 in cytokinesis and how this complex ultimately leads to release of Cdc14 from the nucleolus during mitotic exit remain unclear. One reason is that, while the components of MEN that act upstream of Dbf2-Mob1 have been characterized, the molecular substrates for Dbf2-Mob1 have yet to be identified. At this regards Mah et al. (2005) determined that Dbf2-Mob1 preferentially phosphorylates serine over threonine and required an arginine three residues upstream of the phosphorilated serine in its substrate (RXXS motif).

Recent findings suggest also an involvement of MEN-Mob1p in coordinating chromosome segregation and/or spindle integrity with mitotic exit and cytokinesis via regulation of chromosome passenger proteins. Mob1p has been demonstrated to be essential for maintaining the localization of Aurora, INCENP, and Survivin chromosomal passenger proteins on anaphase spindles and for dissociating Aurora from the kinetochore region (Stoepel et al. 2005). Consistent with these functions, the MEN protein kinase complex Mob1p-Dbf2p localizes to mitotic nuclei and partially co-localizes with Cdc14p and kinetochore proteins.

Overall the available data in yeast indicates an essential role of Mob1p in cell cycle progression, through the interaction with Dbf2/Sid2 protein kinases and reveals an essential temporal and spatial regulation of Mob1 activity.

MEN components are conserved through evolution and in particular Mob1 and Dbf2-related proteins have been found in both animal (Stavridi et al. 2003; Ponchon et al. 2004; Devroe et al. 2004) and plant cells (Van Damme et al. 2004; Citterio et al. 2005, 2006), suggesting that their role in controlling cell cycle progression might be conserved in higher eukaryotes. The demonstration that animal Dbf2 homologous proteins NDR (nuclear Dbf2-related) genetically and physically interact with Mob1-related proteins (Bothos et al. 2005; Hammarton et al. 2005; He et al. 2005; Lay et al. 2005) and the determination of the yeast, human and X. laevis Mob protein structures, suggest that Mob proteins act as kinase activating subunits also in higher eukaryotes.

Nevertheless the biological roles of MOB proteins are still to be understood. In higher eukaryotes multiple MOB members are involved in multiple pathways. To date two probably distinct signaling networks, namely MEN and HIPPO (Bothos et al. 2005; Edgar, 2006), controlling cell proliferation and involving Mob1-like proteins have been recently proposed in Drosophyla and mammalian cells (see Hergovich et al. 2006). HIPPO pathway has been described in flies where participates to the control of tissue growth. This network includes cell cycle and cell death regulators, such as Hippo (Hpo), Salvador (Sav), Lats/ Warts (dNDRs), Mats (Mob as tumor suppressor, dMob1) and Yorkie (Yki) factors (reviewed by Edgar, 2006). All components of the HIPPO pathway are well conserved in mammals and researchers have hypothesized that they share a similar function in humans. The complex Lats-Mob1A was also indicated as a component of the uncharacterized MEN network in higher eukaryotes. Bothos et al. (2005) have demonstrated that, similarly to ScMob1, hMob1A interacts and co-localizes with Lats1 at the centrosomes and midbody and that the suppression of Lats1 or hMob1A extends telophase but not other phases of mitosis. On the basis of the identification of evolutionary conserved MEN components the authors suggested the presence of a MEN conserved pathway in higher eukaryotes (Fig. 5). Given the complexity of the interactions it is possible that different isoforms of hMob1A and Lats belong to specific network and/or that the activation of different pathways is organism, tissue and/or cellular context dependent. Also the subcellular localization of the hMob1A-Lats1 complex is likely determinant for Lats1 activation and function. Hergovich et al. (2006) demonstrated that the membrane-targeting of hMob1A results in a significant increase of Lats1 activity in mammalian cells, while the simple co-expression of Lats1 with hMob1A does not elevate Lats1 kinase activity. On the other hand, the presence of a MEN pathway in higher eukaryotes is also suggested by the study of Mob1 proteins in plants (Citterio et al. 2006). Medicago sativa Mob1 proteins are mostly expressed in actively proliferating tissues and their localization pattern shares many features with that of yeast, despite the differences in mitotic entry and progression between the two organisms. The subcellular localization of MsMob1-like proteins is cell cycle-regulated. In alfalfa cells, Mob1 proteins forms grains in the cytoplasm from which fibrillar structures radiate in all directions, preferentially toward the cell mid-plane. These grains could likely correspond to sites in which microtubules are reorganized during cell cycle progression, the yeast SPBs, and barely detectable in  $G_1$  and S cells, whereas become evident in G<sub>2</sub>, forming clusters around the nucleus. In mitosis, they preferentially localize at the two opposite cellular poles. Differently from yeast, in alfalfa cells undefined Mob1 fibrillar structures are formed. In addition, during pre-prophase Mob1-like proteins mark the inner border of the cell wall in correspondence with the outer parts of the pre-prophase band, and in cytokinesis besides the progressive labeling of the septum, forms fibrillar structures, that partially co-localize with phragmoplast microtubules and partially form an aster, radiating from the growing septum poles.

Overall the results collected so far in plants indicate that Mob1-like proteins are involved in cell proliferation, are expressed in a cell cycle-dependent manner and are localized to the cell division midplane during cytokinesis, marking the progressive formation of the phragmoplast, as shown in Figure 6.

An interesting possibility is that Mob1-like proteins participate to the orientation of cell plate during cytokinesis, interacting with cytoskeletal structures and conjugating the determination of division site, marked by pre-prophase band before the onset of mitosis, with the septum formation (Citterio et al. 2006). Nevertheless the expression of MsMob1 could not rescue the lethality of the yeast mob1 mutant. This inability can be attributed to several reasons and does not rule out that the two genes do encode functional homologs. It is possible that MsMob1 does not bind efficiently to budding yeast Dbf2, thus explaining the lack of cross-complementation. Importantly, amino acid residues of ScMob1, such as Thr105, Leu196 and Cys221, that are changed in mob1 mutant alleles and presumably crucial for Mob1 function (Luca and Winey, 1998; Stavridi et al. 2003), are replaced



**Figure 6.** Results of the simultaneous immunolocalization of Mob1like proteins (green fluorescence, Panels **A**, **D** and **G**) and alpha tubulin (red fluorescence, Panels **C**, **F** and **I**) in alfalfa cells during three successive stages of cytokinesis (the yellow fluorescence represents tubulin and Mob1-like protein co-localization). DNA was also stained with DAPI (gray signal, **B**, **E** and **H**). Mob1-like proteins are localized to the cell division midplane during cytokinesis, marking the progressive formation of the phragmoplast (for additional information, see Citterio et al. 2006).

in a non-conservative way in the MsMob1 primary sequence, suggesting that in spite of their high degree of similarity the two proteins might have substantially diverged and that the interaction of Mob1 proteins with their effectors may be speciesspecific.

On the whole, the available data strongly suggest that in higher eukaryotes as in yeast Mob1 members of MOB family play a role in the control of cell proliferation, through the regulation of NDR activity and localization. However further experiments are needed to better understand the roles of the single Mob1-like genes in each type of organism and tissue.

# Apoptosis and Programmed Cell Death

In a multicellular organism, the maintenance and surveillance of organ size is essential. Any imbalance in the relationship between cell size, cell proliferation and cell death must be prevented to allow proper organ development and to maintain the integrity of organ tissue over time. Failure to coordinate the creation of new cells (proliferation) and the elimination of excess ones (by apoptosis) can lead to diseases (Green and Evan, 2002). Mob proteins are involved in the control of cell death and its coordination with cell proliferation, being direct co-activators of NDR (nuclear Dbf2-related) kinases.

Recent advances using D. melanogaster lead to the identification of a pathway that participates in the control of tissue growth (Harvey et al. 2003; Jia et al. 2003; Pantalacci et al. 2003; He et al. 2005; Huang et al. 2005). The control of cell death and proliferation by the Hippo (hpo)-Large tumor suppressor (Lats) pathway was demonstrated and a similar pathway was also postulated in mammals (Fig. 7). In Drosophila, four factors that induce tissue overgrowth without affecting pattern formation were identified: Sav, Hpo, Lats and dMob1/Mats (reviewed by Hergovich et al. 2006). Loss of any of these factors results in tissue overgrowth which is associated with increased cell proliferation and decreased cell death, indicating that Sav, Hpo, Lats and dMob1 all function as tumour suppressors. Genetic and biochemical independent studies indicate that Hpo interacts with Say, which acts as a scaffold protein, and phosphorilates Warts-Mats. The association of Mats with Warts is essential in this regulatory process, as flies that carry mutation in Mats are



**Figure 7.** The HIPPO (hpo)-Large tumor suppressor (Lats) pathway validated in *Drosophila melanogaster* and its similar pathway recently postulated in mammals. The network involves Hippo (Hpo), Salvador (Sav), Lats1/Warts (dNDRs), Mats (Mob as tumor suppressor, dMob1) and Yorkie (Yki) factors, and participates to the control of tissue growth by regulating cell cycle arrest and cell death. In *Drosophila* Hpo interacts with Sav, which acts as a scaffold protein, and phosphorilates Warts-Mats. Activated Warts can negatively regulate the transcription of cell cycle and cell death regulators such as cyclin E and the apoptosis inhibitor DIAP1, through the phosphorilation of the non-DNA binding transcriptional co-activator Yorkie. All components of the HIPPO pathway are well conserved in mammals and they have a similar function in humans since Lats1 (Warts), Mob1A (Mats), MST2 (Hippo) and Yap (Yorkie) genes can all functionally rescue their correspondent *Drosophila* mutants.

unable to control tissue growth, despite having a functional Warts. Activated Warts has been proposed to negatively regulate the transcription of cell cycle and cell death regulators. Interestingly, the tissue overgrowth phenotype in *Drosophila* is accompanied by elevated levels of an important regulator of S-phase entry (i.e. cyclin E) and Diap1 (*Drosophila* inhibitor of apoptosis protein-1), an inhibitor of apoptosis. Moreover, *Drosophila* Salvador (Sav) interacts biochemically with Hpo, thereby facilitating the activation of Lats by phosphorylation (Harvey et al. 2003; Pantalacci et al. 2003; Wu et al. 2003). The activated Lats-dMob1 (*Drosophila* Mps1-one binder-1) complex then inactivates Yorkie (Yki) by phosphorylation (Huang et al. 2005). Phosphorylated Yki can not stimulate the expression of cyclin E and Diap1, which results in decreased cell proliferation (low cyclin E) and increased cell death (low Diap1). It is worth noting that the association of dMob1 with Lats is essential in this regulatory process, as flies that carry mutations in dMob1 are unable to control tissue growth, despite having a functional Lats (Lai et al. 2005). Therefore, Lats that is phosphorylated by Hpo needs to bind to its co-activator dMob1 to properly coordinate cell death and proliferation (Fig. 7). As a matter of fact, cells that carry mutations in Hpo, Sav, Lats and dMob1 show an accelerated proliferation, but maintain a normal size. As a consequence, loss of these genes must stimulate cell growth and reduce cell death.

In mammals, a similar pathway was postulated (Fig. 7). Several human orthologs of the Hpo–Sav– Lats-dMob1-Yki pathway have emerged as putative tumour suppressors (Tapon et al. 2002; Lai et al. 2005; Takahashi et al. 2005; Jimenez-Velasco et al. 2005). Human mammalian sterile 20-like kinase (MST1/2) associates with hWW45 (the human ortholog of Sav) and activates LATS1/2 by phosphorylation (Chan et al. 2005). The LATS-hMOB1 complex then potentially activates specific gene expression programs through YES-associated protein (YAP). Similar to large tumour suppressor (Lats) in invertebrates, several findings point to LATS functioning as a tumour suppressor in mammals (St John et al. 1999; Hisaoka et al. 2002; Takahashi et al. 2005; Jimenez-Velasco et al. 2005). The significance of functional conservation is further strengthened by the fact that human MST2, hMOB1A and LATS1 can rescue the tissue-overgrowth phenotype of Hpo, dMob1/Mats and Lats mutants in D. melanogaster (Wu et al. 2003; Lai et al. 2005). Moreover HIPPO components, including Mob1A are mutated in mammalian tumors.

Overall, LATS seems to be a tumour-suppressor protein that is conserved in flies and humans, whereas the roles of mammalian NDR1/2 and their co-activators MOBs are yet to be fully established. Existing findings indicate that mammalian NDR1/2 could function as proto-oncogenes (Hergovich et al. 2006).

Like in animals, also in plants specific cell types undergo programmed cell death (PCD) as part of their developmental and differentiation program (Vaux and Korsmeyer, 1999). From embryogenesis to fertilization, cell and tissue death is an integral part of plant development and morphogenesis as well as a response to the environment (Barlow, 1982; Buckner et al. 1988). Even though the cellular deterioration patterns described in plant tissues are in some cases similar to those observed in animal tissues, little is known of the mechanisms that control PCD in plants (Pennell and Lamb, 1997; Allen et al. 1998; Vaux and Korsmeyer, 1999). In angiosperms, PCD occurs late in the degenerative stage of the reproductive phase in both anther and pistil (Wu and Cheung, 2000). Production of functional male gametes depends largely on the deterioration and death of the anther tapetum, whose main functions appear to be the nurturing of microspores with cortical surface molecules and allowing pollen dispersion at maturity. The pathway of female gametogenesis frequently begins with the death of all but one reduced megaspores, while surrounding nucellar cells degenerate in concert with embryo sac expansion (Reiser and Fisher, 1993; McCormick, 1993; Barcaccia et al. 2003).

Mob1 may be a component of a complex of proteins with multiple functions, not only involved in cytokinesis, cell proliferation and morphogenesis, but also operatively associated with cell death. Database searches revealed that MOB domain (pfam03637) can be combined in complex proteins with elements of the NB-ARC domain (pfam00931), a signaling motif shared by animal cell death gene regulators. Proteins containing a highly conserved Mob1 domain include also receptors for ubiquitination targets (F-Box), Ser/Thr and Tyr kinases as well as CBL (Calcineurin B-Like)-interacting kinases which may be implicated in either cell proliferation or cell death. The possible involvement of Mob1 proteins in PCD is also supported by our recent analysis of Mob1-like expression in alfalfa reproductive tissues (Fig. 8). In the ovules during gametogenesis, both transcripts and proteins were mainly visualized in the reduced megaspores undergoing PCD or in the remnants of degenerated megaspores, whereas in the anthers, Mob1-like gene products were specifically found at the end of gametogenesis in tapetum cells naturally undergoing PCD to allow pollen grain dispersal (Citterio et al. 2005). Moreover, localization of MOBdomain containing proteins was also documented in alfalfa meristematic tissues of the plant roots. It is known that the root cap consists in living parenchyma cells derived continuously from the apical meristem and programmed to die: as new cells are produced in the interior, those on the root periphery are shed in an orderly manner. Hybridization signals were detected in a thin cell layer of the root apex where meristematic root tip cells divide and differentiate in root cap. Such finding



**Figure 8.** Mob1-like expression patterns in plant reproductive tissues, with particular reference to alfalfa (*Medicago sativa* L.). The cartoons show spores and cells that most prominently undergo programmed cell death (PCD) in ovules and anthers (adapted from Wu and Cheung, 2000). In ovules at the end of sporogenesis, proteins are mainly visualized in the reduced megaspores undergoing PCD or the remnants of degenerated megaspores (**A**, **B**), whereas in anthers, proteins are specifically found at the end of gametogenesis in tapetum cells naturally undergoing PCD to allow pollen grain dispersal (**C**). Bar: 20 µm (for experimental details, see Citterio et al. 2005).

further supports the concept that Mob proteins are related to the onset of programmed cell death in plants (Citterio et al. 2006).

Further experiments will help clarifying the function of Mob1-like proteins in both cell proliferation and PCD. The challenge will be to dissect the roles of each Mob1-like gene in different tissues. The production and exploitation of specific antibodies against each of the Mob1-like gene products encoded by a specific member of the MOB family should aid in determining whether a multi-domain protein component with distinct functions is operative during cell proliferation and PCD.

### **Cell Polarity and Morphogenesis**

The MOB2-NDR proteins are central factors of the RAM (Regulation of Ace2 Activity and Morphogenesis) network in cell separation and polarity establishment. In this section we will briefly review on the progress made so far on the elucidation of the role played by MOB proteins and NDR kinases in regulating cell morphology in co-ordination with the mitotic exit. Co-ordinating asymmetric cell division, and establishment and maintenance of cell polarity are essential processes in growth and differentiation. Polarized morphogenesis is necessary for proper functioning of specific cell types such as neurons, epithelial cells, plant root hairs and pollen tubes and fungal hyphae and its core elements are substantially conserved across eukarvotes. Cell intrinsic polarity is established early during cell division and factors governing cell separation and cell polarity are tightly controlled and co-ordinated.

Studies carried out on yeast, have led to the identification of the so-called RAM network of proteins as a central element involved in the early phases of polar morphogenesis during cell separation (Nelson et al. 2003). The core components of the yeast RAM network are the LATS/NDR kinase CBK1p and its upstream regulator MOB2p, which play a dual role in controlling mother-daughter cell separation and establishment of cell polarity. Cell separation in yeast relies on the daughter cell specific expression of genes necessary for septum degradation, shown to be dependent on the specific localization and activation of the ACE2 transcription factor in the daughter cell nucleus together with MOB2p and CBK1p at the end of mitosis (Colman-Lerner et al. 2001; Weiss et al. 2002).

Loss of function strains mob $2p\Delta$  and cbk $1p\Delta$  as well as ace $2p\Delta$  show defects in the cell separation process resulting in clumps of cells. However, interestingly, the mob $2p\Delta$  and cbk $1p\Delta$  cells, but not the ace $2p\Delta$ , display loss of polar growth suggesting that the MOB2p-CBK1p complex regulates cell morphology through a specific pathway that is independent from Ace2 activity (Weiss et al. 2002; Nelson et al. 2003). Cells deleted for either CBK1 or MOB2 or expressing a catalytically inactive form of Cbk1p in S. cerevisiae (Racki et al. 2000; Bidlingmaier et al. 2001; Colman-Lerner et al. 2001; Weiss et al. 2002) or lacking CBK1 and MOB2 orthologs in S. pombe (Verde et al. 1998; Hou et al. 2003) are round and lack axial polarization, proper bud selection and mating projections. In addition cells lacking a functional MOB2p-CBK1p machinery display multiple sites of bud selection and growth suggesting a general role for these proteins in determining early events for cell polarity establishment (Nelson et al. 2003). A schematic representation of the S. cerevisiae RAM network is reported in Figure 9.

Based on genetic and biochemical studies in yeast, MOB2p-CBK1p activity is placed downstream of and dependent on the functional presence of the other RAM proteins KIC1p, HIM1p, TAO3p and SOG2p with KIC1p, HIM1p and SOG2p forming a functional complex required for MOB2p–CBK1p phosphorylation and activation (Nelson et al. 2003). The KIC1p kinase, the second kinase of the RAM signaling network together with CBK1, displays significant sequence similarity to the MEN kinase Cdc15p, involved in the activation of the MEN MOB1p-DBF2p kinase complex directly (Mah et al. 2001), and it has been shown to activate Mob2p-Cbk1p for regulating Ace2p and cellular morphogenesis (Nelson et al. 2003). These data suggest the conservation of the core interaction of MOB and NDR proteins in both MEN and RAM networks and of their mode of regulation by immediate upstream factors. Furthermore, the role of the MOB2-NDR complex in establishing cell polarity seems to be conserved throughout eukaryotes, since loss of function of CBK1/ORB6-related NDR kinases leads to defects in cell axialization and cell spreading and/or branching also in Drosophila, C. elegans and in mammalian cells. However, while loss of CBK1 function in yeast leads to a failure in axialization and bud selection of cells (Racki et al. 2000; Bidlingmaier et al. 2001;



**Figure 9.** Schematic representation of the *S. cerevisiae* RAM network. The protein kinase Kic1 associates with the proteins Sog2 and Hym1 to form a complex necessary for proper localization and function of Cbk1. In analogy to its counterpart Cdc15 in the MEN network, Kic1 likely activates Cbk1 directly. Pag1 interacts with Kic1 and Cbk1 facilitating its activation. Cbk1 requires the interaction with Mob2 for activation and to regulate the transcription factor Ace2, essential for cell separation to occur, through the transcription of genes involved in cell wall synthesis in a daughter cell specific way. The Cbk-Mob2 complex also regulates polarized growth of cells, proper bud site selection and formation of mating projections through a largely uncharacterized Ace2 independent pathway.

Colman-Lerner et al. 2001; Du and Novick, 2002; Weiss et al. 2002; Nelson et al. 2003) the inactivation of the *Drosophila* NDR encoding gene tricornered (trc) leads to split epidermal hairs and bristles (Geng et al. 2000) and augmented dendritic branching (Emoto et al. 2004). Similar defects in dendritic branching are observed in the presence of mutations of the *C. elegans* NDR encoding gene Sax1 (Zallen et al. 2000) suggesting a negative role exerted by NDR kinases in the control of cell axialization and branching in higher eukaryotes opposite to the positive role played by the MOB2p-CBK1p complex of yeast. Hyperpolarization instead of loss of polarization has also been shown following systematic mutagenesis of components of the RAM network in the pathogenic fungus Cryptococcus neoformans (Walton et al. 2006). This was observed in the presence of substantial conservation of subcellular localization and protein-protein interactions between MOB2p and CBK1 homologs and upstream components (Walton et al. 2006), further suggesting a general conservation of the central role of the MOB2p-CBK1p/NDR complex in directing cell polarity in eukaryotes, but pointing to a probable divergence of downstream components leading to opposite cell polarity phenotypes. This may reflect different mechanisms of cell shape control via the re-organization of the cytoskeleton through assembly of actin cables, controlled for example in yeast by formin (Burns et al. 1994; Evangelista et al. 2002), or via alternative systems. Interestingly, the MOB2p-CBK1p complex seems to regulate cell polarity through a mechanism that is at least partly independent from the actin cables assembly since in RAM mutants actin organization has been reported to be not substantially affected (Weiss et al. 2002; Nelson et al. 2003). In addition MOB2 or CBK1 mutations result in additive phenotypes when combined with mutations affecting the formin encoding gene Bni1 (Du and Novick, 2002; Weiss et al. 2002; Nelson et al. 2003). This together with the finding that Cbk1p has been shown to bind Sec2p, a guanine nucleotide exchange factor involved in vesicle transport and exocytosis (Racki et al. 2000), have lead to the hypothesis that the RAM network may act in cell polarity through regulation of vesicle transport (Terbush et al. 1996; Lipschutz and Mostov, 2002). On the contrary, the Drosophila Trc gene functions altering actin and microtubule organization (He et al. 2005) and has been placed on the same genetic pathway of RhoA GTPase since loss of Trc function and expression of a dominant negative form of RhoA result in similar non additive phenotypes (He et al. 2005). Rho GTPases are well known players in cell polarity establishment through the regulation of actin dynamics, however even though it has been suggested that they may be downstream components of NDR kinases in Drosophila (He et al. 2005) and in C. elegans (Zallen et al. 2000), definitive biochemical evidence is needed to fully clarify their exact hierarchical relationships. In fact, it cannot be excluded that the MOB-NDR

complex may be a downstream component of Rho GTPases, also considering the similarity of NDR kinases with Rho kinases, the immediate downstream components of Rho signaling.

# General Discussion and Concluding Remarks

The MOB family includes a group of cell cycleassociated, non-catalytic proteins highly conserved in eukaryotes, whose founding members are implicated in mitotic exit and co-ordination of cell cycle progression with cell polarity and morphogenesis (Luca et al. 2001; Stegmeier et al. 2002; Nelson et al. 2003).

An HMM search for Mob-like domain containing proteins in 43 completed and ongoing eukaryotic genomes highlights the universal distribution of this protein family in the so-far sequenced organisms, suggesting its prominent biological function. The phylogenetic analysis reveals five distinct classes of the MOB domain, resulting in the necessity of a reassessment of the relationship existing among the proteins found in different taxa. As an example, in our analysis the founding member ScMob1 does not cluster within the Mob1 group, as previously reported in various papers (Stavridi et al. 2003; Mrkobrada et al. 2006).

Analysis on Mob domain distribution reveals a progressive expansion of this family from unicellular to multicellular organisms, reaching the highest number in mammals. Moreover, phylogenetic analysis shows that the Mob4 genes form a peculiar class of the invertebrata taxa, that underwent an expansion in vertebrata giving origin to Mob4a and Mob4b classes. Plant Mob genes appear to have evolved from a single ancestor, most likely due to the loss of one or more genes during the early stage of Viridiplantae evolutionary history. Finally Mob1, Mob2 and Mob3 classes are widespread among almost all analyzed organisms. Mob3 class is the most divergent one, suggesting a possible different function for the genes belonging to this class. Mob2 class, compared to the other Mob classes, presents a lower gene identity percentage homogeneity, revealing the possible presence of other subgroups belonging to this class.

Different distribution and phylogenetic relationship among genes of the same organism and/or different organisms suggest that the Mob gene family evolves under a birth-and-death evolution model (Nei and Rooney, 2005).

Two distinct Mob proteins, Mob1 and Mob2, are known in fungi, while an expansion in metazoans gives rise to six (seven) in human, four in D. melanogaster, and four in C. elegans (Mrkobrada et al. 2006). Mob1 proteins have been demonstrated to be important for both mitosis completion and cell plate formation in yeast (Luca and Winey, 1998). Moreover, the Mob1-related proteins Mob2 physically associate with specific kinases throughout the cell cycle, being required and periodically activated in yeast to promote polarized growth (Weiss et al. 2002). Mob1-like proteins have been also found in animals (Stavridi et al. 2003; Ponchon et al. 2004; Devroe et al. 2004). Plant genomes such as alfalfa, rice and Arabidopsis contain uncharacterized Mob1-related genes (Van Damme et al. 2004; Citterio et al. 2005; 2006). Although there are data to suggest that Mob1 proteins act as kinase activating subunits in higher eukaryotes, their function remains to be proved. Present findings suggest that animal and yeast Mob1 may have similar functions.

That Mob1 proteins play a crucial role in cytokinesis has been demonstrated in yeast (Luca and Winey, 1998). The study of a spontaneous lethal mutation in a Drosophila Mob1 gene has recently implicated the MOB-domain containing proteins in the control of animal cell proliferation and apoptosis (Lai et al. 2005). Moreover, the identification of the animal Dbf2 homologous proteins NDR (Nuclear Dbf2-Related) interacting with Mob1-related proteins, and the determination of the human and *Henopus laevis* Mob protein tridimensional structures, may mean that Mob proteins act as kinase activating subunits even in higher eukaryotes. The functional co-dependence and cell cycle regulation of the Mob and Dbf2-like proteins is reminiscent of how cyclins bind and regulate cyclin-dependant kinases (Morgan, 1996; Mah et al. 2001).

MOB-domain containing proteins represent essential regulators of the localization and activity of nuclear Dbf2-related (NDR) protein kinases, components of the mitotic exit network (MEN) in yeast and MEN-like in human. Several lines of research in mammals are now in progress to define the precise roles of NDR interactors, particularly the regulation of Mob activators and MST kinases. A general regulation scheme at the molecular level, probably valid for all NDR family members, has recently been established (see Hergovich et al. 2006). The binding of the co-activator MOB-domain containing proteins to the N terminus of NDR kinases seems crucial for activation and function. It is known that Mob proteins interact with NDR-type kinases by binding a conserved stretch of primary sequence at their N-terminal regulatory domain. The interaction of Mob proteins with the NTR activation site is a conserved feature of all members of the NDR kinase family that have been tested so far in yeasts, flies and humans. Interestingly, Mob proteins do not function solely as co-activators of NDR kinases, but are also required for the localization of yeast NDR kinases. As a matter of fact, members of the NDR family are essential genes in both uni- and multicellular organisms. Dbf2p and Sid2p regulate mitotic exit and cytokinesis in yeasts, and their counterparts in mammals and plants could also have a similar role.

Recent advances lead to the identification of the Hippo signaling pathway that controls the coordination of apoptosis and cell proliferation, and tissue growth in D. melanogaster (see Hergovich et al. 2006). The association of Mob1p with Lats (Large tumor suppressor) is essential in this regulatory process since flies that carry mutations in dMob1 are unable to control tissue growth, despite having a functional Lats (Lai et al. 2005). Therefore, Lats that is phosphorylated by Hpo needs to bind to its co-activator dMob1 to properly coordinate cell death and proliferation. Interestingly, conserved key components of this pathway have been found to be mutated in human cancer samples, which indicates that a kinase network is probably conserved from flies to humans.

In plants, signaling mechanisms co-ordinate mitosis spatially and temporarily with cytokinesis to ensure integrity of genetic transfer during the cell cycle (Guertin et al. 2002), and important genes required for cytokinesis have recently been discovered. The involvement of plant Mob genes in cell cycle control is supported by recent data collected in Arabidopsis and Medicago sativa (Van Damme et al. 2004; Citterio et al. 2006). For instance, in Arabidopsis several putative cell cycle associated components (e.g. Mob1-like proteins) were targeted to the cell division plane and to the nucleus, suggesting that this organelle operates as a coordinating hub for cytokinesis (Van Damme et al. 2004). Moreover, in M. sativa Mob1-like proteins were proven to appear during late telophase and to localize across the entire cell division midplane, thus marking the progressive formation of the phragmoplast (Citterio et al. 2006). Nevertheless, the key role of MOB-domain containing proteins in plants is still poorly understood.

The greater amount of Mob1-like proteins in proliferating than in non-proliferating tissues, together with their cell cycle-regulated subcellular localization and their presence at the cleavage site suggest that these proteins may have a function in cell division similar to that of yeast Mob1 essential for mitotic exit and septum formation. In yeast, the spindle pole body operates as a signaling center during cytokinesis (Simanis, 2003). MEN/SIN regulators such as Sid kinases and Dbf2/Mob1 temporarily associate with the spindle pole body at some point in the cell cycle. For instance, in S. cerevisiae, Mob1 mobilizes to the spindle pole body (SPB) at anaphase and localizes to the bud neck, the future site for cell division, during cytokinesis (Hou et al. 2003). In analogy to this function, centrosomes have been implicated in completing cytokinesis in animals and human cells (Doxsey, 2001). In higher plant cells, microtubules (MTs) show dynamic structural changes during cell cycle progression and play significant roles in cell morphogenesis (Hasezawa and Kumagai, 2002). In addition to the cortical microtubules that control the cell shape, the preprophase band (PPB) and the phragmoplast are other plant-specific structures which can be observed from late interphase to prophase, and from anaphase to telophase, respectively. How plant MT arrays reorganize during the cell cycle is an unanswered question. Plants lack conventional animal centrosomes and yeast SPBs seem to possess flexible centrosomes from which nucleating material disperses at different cell cycle stages (Chan et al. 2003).

Despite differences between plant and yeast in mitotic entry and progression, the localization pattern in plant cells of Mob proteins shares many features with yeast (Van Damme et al. 2004; Citterio et al. 2006). In plant cells, Mob1-like proteins form grains in the cytoplasm from which fibrillar structures radiate in all directions, preferentially toward the cell midplane. These grains likely correspond to sites in which microtubules are reorganized during cell cycle progression. Proteins, barely visible in  $G_1$  and S, are clearly seen in  $G_2$  forming a ring around the nucleus,

whereas during mitosis they preferentially localize as punctuate clusters at the two opposite cellular poles. Differently from yeast, in plants cells undefined fibrillar structures are formed. In cytokinesis besides the progressive labeling of the septum, Mob1-like proteins form fibrillar structures that partially co-localize with phragmoplast microtubules and partially form an aster, radiating from the growing septum poles. An interesting possibility is that Mob1-like proteins participate in cell plate orientation during cytokinesis, interacting with cytoskeletal structures and coupling the establishment of the division site, marked by PPB before the onset of mitosis, with septum formation. The interaction between MTs and Mob1p is emphasized by the characterization of haploid mob1 yeast mutants, which display a complete increase in ploidy at permissive temperature, caused by cytokinetic defects (Luca et al. 2001). However, although it is well demonstrated that yeast Mob1 is essential for the exit from mitosis and for septum formation, its exact function is still to be known even in this simple organism. Mob1 has been proposed to activate the mitotic exit network acting as an activating subunit of the Dbf2 protein kinase (Stavridi et al. 2003). In animal cells Dbf2 homologs interacting with Mob1-like proteins have been discovered (Ponchon et al. 2004; Devroe et al. 2004), suggesting a conserved function between yeast and higher eukaryotes. Nevertheless, Dbf2 homologs have not yet been characterized in plants.

The control of cell proliferation and cell death are central points of ongoing research programs in cell cycle control of all eukaryotes and, particularly, in human diseases by using model organisms. Basic studies addressed to the understanding of the mitotic events and its alterations will be crucial for practical applications in cell biology and medicine.

#### Acknowledgements

This work was supported by grant from the Italian Minister of the University, Research, Science and Technology (MURST). The fellowship of Giulio Galla was funded by the PRIN project titled: Genomic and functional analysis of candidate genes for apomeiosis (Research Unit responsible person: Gianni Barcaccia). The fellowship of Monica Zermiani was funded by the European Space Agency (ESA), MAP project number: 14341/00/NL/SH (Research Unit responsible person: Benedetto Ruperti).

#### References

- Abascal, F., Zardoya, R. and Posada, D. 2005. ProtTest: Selection of best-fit models of protein evolution. *Bioinformatics*, 21:2104–2105.
- Allen, R.T., Cluck, M.W. and Agrawal, D.K. 1998. Mechanisms controlling cellular suicide: role of Bc11 and caspases. *Cell Mol. Life Sci.*, 54:427–445.
- Baillat, G., Moqrich, A., Castets, F., Baude, A., Bailly, Y., Benmerah, A., and Monneron, A. 2001. Molecular cloning and characterization of phocein, a protein found from the Golgi complex to dendritic spines. *Mol. Biol. Cell*, 12:663–673.
- Baillat, G., Gaillard, S., Castets, F., and Monneron, A. 2002. Interactions of phocein with nucleoside-diphosphate kinase, Eps15, and Dynamin, I. *J. Biol. Chem.*, 277:18961–18966.
- Barcaccia, G., Tavoletti, S., Mariani, A., and Veronesi, F. 2003. Occurrence, inheritance and use of reproductive mutants of alfalfa (*Medicago* spp). *Euphytica*, 133:37–56.
- Barlow, P.W. 1982. Cell death: an integral part of plant development. In: M.B. Jackson, B. Grout, I.A. Mackenzie (Eds.), Growth regulators in plant senescence. Oxon British Plant Growth Regulator Group, Wantage. pp., 27–45.
- Bichsel, S.J., Tamaskovic, R., Stegert, M.R. and Hemmings, B.A. 2004. Mechanism of activation of NDR (Nuclear Dbf2-related) protein kinase by the hMOB1 protein. J. Biol. Chem., 279:35228–35235.
- Bidlingmaier, S., Weiss, E.L., Seidel, C., Drubin, D.G. and Snyder, M. 2001. The Cbk1p pathway is important for polarized cell growth and cell separation in *Saccharomyces cerevisiae*. *Mol. Cell. Biol.*, 21:2449–2462.
- Bothos, J., Tuttle, R.L., Ottey, M., Luca, F.C. and Halazonetis, T.D. 2005. Human LATS1 is a mitotic exit network kinase. *Cancer Res.*, 65:6568–6575.
- Buckner, B., Janick-Buckner, D., Gray, J. and Johal, G.S. 1998. Cell death mechanisms in maize. *Trends Plant Sci.*, 3:218–223.
- Burns, N., Grimwade, B., Ross-Macdonald, P.B., Choi, E.Y., Finberg, K., Roeder, G.S. and Snyder, M. 1994. Large-scale analysis of gene expression, protein localization, and gene disruption in *Saccharomyces cerevisiae. Genes. Dev.*, 8:1087–1105.
- Citterio, S., Varotto, S., Albertini, E., Feltrin, E., Soattin, M., Marconi, G., Sgorbati, S., Lucchin, M. and Barcaccia, G. 2005. Alfalfa Mob1-like proteins are expressed in reproductive organs during meiosis and gametogenesis. *Plant Mol. Biol.*, 58:789–808.
- Citterio, S., Piatti, S., Albertini, E., Aina, R., Varotto, S. and Barcaccia, G. 2006. Alfalfa Mob1-like proteins are involved in cell proliferation and localize in the cell division midplane during cytokinesis. Exp. *Cell. Res.*, 312:1050–1064.
- Chan, J., Calder, G.M., Doonan, J.H. and Lloyd C.W. 2003. EB1 reveals mobile microtubule sites in *Arabidopsis. Nat. Cell. Biol.*, 5:967–971.
- Chan, E.H., Nousiainen, M., Chalamalasetty, R.B., Schafer, A., Nigg, EA. and Sillje, H.H. 2005. The Ste20-like kinase Mst2 activates the human large tumour suppressor kinase Lats1. *Oncogene*, 24:2076–2086.
- Colman-Lerner, A., Chin, T.E. and Brent, R. 2001. Yeast Cbk1 and Mob2 activate daughter-specific genetic programs to induce asymmetric cell fates. *Cell*, 107:739–750.
- Danial, N.N. and Korsmeyer, S.J. 2004. Cell death: critical control points. Cell, 116:205–219.
- Devroe, E., Erdjument-Bromage, H., Tempst, P. and Silver, P.A. 2004. Human Mob proteins regulate the NDR1 and NDR2 serine-threonine kinases. J. Biol. Chem., 279:24444–24451.
- Doxey, S. 2001. Re-evaluating centrosome function. *Nat. Rev. Mol. Cell. Biol.*, 2:688–698.
- Du, L.L. and Novick, P. 2002. Pag1p, a novel protein associated with protein kinase Cbk1p, is required for cell morphogenesis and proliferation in *Saccharomyces cerevisiae*. *Mol. Biol. Cell*, 13:503–514.
- Durbin, R., Eddy, S., Krogh, A. and Mitchison, G. 1998. Biological sequence analysis: probabilistic models of proteins and nucleic acids. Cambridge University Press, UK.

- Edgar B.A. 2006. From cell structure to transcription: Hippo forges a new path. Cell, 124(2):267–73.
- Emoto, K., He, Y., Ye, B., Grueber, W.B., Adler, P.N., Jan, L.Y. and Jan, Y.N. 2004. Control of dendritic branching and tiling by the Tricornered-kinase/Furry signaling pathway in *Drosophila* sensory neurons. *Cell*, 119:245–256.
- Evangelista, M., Pruyne, D., Amberg, D.C., Boone, C. and Bretscher, A. 2002. Formins direct Arp2/3-independent actin filament assembly to polarize cell growth in yeast. *Nat. Cell. Biol.*, 4:260–269.
- Frenz, L.M., Lee, S.E., Fesquet, D. and Johnston, L.H. 2000. The budding yeast Dbf2 protein kinase localises to the centrosome and moves to the bud neck in late mitosis. J. Cell. Sci., 113:3399–3408.
- Geng, W., He, B., Wang, M. and Adler, P.N. 2000. The tricornered gene, which is required for the integrity of epidermal cell extensions, encodes the *Drosophila* nuclear DBF2-related kinase. *Genetics*, 156:1817–1828.
- Green, D.R. and Evan, G.I. 2002. A matter of life and death. *Cancer Cell*, 1:19–30.
- Guertin, D.A., Trautmann, S. and McCollum, D. 2002. Cytokinesis in eukaryotes. Microbiol. *Mol. Biol. Rev.*, 66:155–178.
- Guindon, S. and Gascuel, O. 2003. A simple, fast, and accurate algorithm to estimate large phylogenies by maximum likelihood. *Syst. Biol.*, 52:696–704.
- Hammarton, T.C., Lillico, S.G., Welburn, S.C. and Mottram, J.C. 2005. *Trypanosoma brucei* MOB1 is required for accurate and efficient cytokinesis but not for exit from mitosis. *Mol. Microbiol.*, 56:104–116.
- Hasezawa, S. and Kumagai, F. 2002. Dynamic changes and the role of the cytoskeleton during the cell cycle in higher plant cells. Academic Press, Tokio, Japan. pp., 161–191.
- Harvey, K.F., Pfleger, C.M. and Hariharan, I.K. 2003. The *Drosophila* Mst ortholog, hippo, restricts growth and cell proliferation and promotes apoptosis. *Cell*, 114:457–467.
- He, Y., Emoto, K., Fang, X., Ren, N., Tian, X., Jan, Y.N. and Adler, P.N. 2005. *Drosophila* Mob family proteins interact with the related tricornered (Trc) and warts (Wts) kinases. *Mol. Biol. Cell*, 16:4139–4152.
- Hennebold, J.D., Tanaka, M., Saito, J., Hanson, B.R. and Adashi, E.Y. 2000. Ovary-selective genes I: the generation and characterization of an ovary-selective complementary deoxyribonucleic acid library. *Endocrinology*, 141:2725–2734.
- Hergovich, A., Bichsel, S.J. and Hemmings, B.A. 2005. Human NDR kinases are rapidly activated by MOB proteins through recruitment to the plasma membrane and phosphorylation. *Mol. Cell. Biol.*, 25:8259–8272.
- Hergovich, A., Schmitz, D., and Hemmings, B. A. 2006. The human tumour suppressor LATS1 is activated by human MOB1 at the membrane. *Biochem. Biophys. Res. Comm.*, 345:50–58.
- Hergovich, A., Stegert, M.R., Schmitz, D. and Hemmings, B.A. 2006. NDR kinases regulate essential cell processes from yeast to humans. *Nature Reviews*, 7:253–264.
- Higgins, D.G., Bleasby, A.J. and Fuchs, R. 1992. CLUSTAL: a package for performing multiple sequence alignments on a micro-computer. Comput. Appl. *Biosci.*, 8:189–191.
- Hisaoka, M., Tanaka, A., and Hashimoto, H. 2002. Molecular alterations of h-warts/LATS1 tumor suppressor in human soft tissue sarcoma. *Lab Invest.*, 82:1427–1435.
- Hou, M.C., Wiley, D.J., Verde, F. and McCollum, D. 2003. Mob2p interacts with the protein kinase Orb6p to promote coordination of cell polarity with cell cycle progression. J. Cell Sci., 116:125–135.
- Hou, M.C., Guertin, D.A. and McCollum, D. 2004. Initiation of cytokinesis is controlled through multiple modes of regulation of the Sid2p-Mob1p kinase complex. *Mol. Cell Biol.*, 24:3262–3276.
- Huang, J., Wu, S., Barrera, J., Matthews, K. and Pan, D. 2005. The Hippo signaling pathway coordinately regulates cell proliferation and apoptosis by inactivating Yorkie, the *Drosophila* homolog of YAP. *Cell*, 122:421–434.
- Jia, J., Zhang, W., Wang, B., Trinko, R. and Jiang, J. 2003. The Drosophila Ste20 family kinase dMST functions as a tumor suppressor by restricting cell proliferation and promoting apoptosis. *Genes. Dev.*, 17:2514–2519.

- Jimenez-Velasco, A. Roman-Gomez, J., Agirre, X., Barrios, M., Navarro, G., Vazquez, I., Prosper, F., Torres, A. and Heiniger, A. 2005. Downregulation of the large tumor suppressor 2 (LATS2/KPM) gene is associated with poor prognosis in acute lymphoblastic leukemia. *Leukemia*, 19:2347–2350.
- Justice, R.W., Zilian, O., Woods, D.F., Noll, M. and Bryant, P.J. 1995. The Drosophila tumor suppressor gene warts encodes a homolog of human myotonic dystrophy kinase and is required for the control of cell shape and proliferation. Genes. Dev., 9:534–546.
- Komarnitsky, S.I., Chiang, Y.C., Luca, F.C., Chen, J., Toyn, J.H., Winey, M., Johnston, L.H. and Denis, C.L. 1998. DBF2 protein kinase binds to and acts through the cell cycle-regulated MOB1 protein. *Mol. Cell Biol.*, 18:2100–2107.
- Krapp, A., Gulli, M.P. and Simanis, V. 2004. SIN and the art of splitting the fission yeast cell. *Curr. Biol.*, 14:R722–R730.
- Lai, Z.C., Wei, X., Shimizu, T., Ramos, E., Rohrbaugh, M., Nikolaidis, N., Ho, L.L. and Li, Y. 2005. Control of cell proliferation and apoptosis by mob as tumor suppressor, mats. *Cell*, 120:675–685.
- Lee, S.E., Frenz, L.M., Wells, N.J., Johnson. and A.L. and Johnston, L.H. 2001. Order of function of the budding-yeast mitotic exit-network proteins Tem1, Cdc15, Mob1, Dbf2, and Cdc5. *Curr. Biol.*, 11:784–788.
- Lippincott, J., Shannon, K.B., Shou, W.Y., Deshaies, J. and Li, R. 2001. The Tem1 small GTPase controls actomyosin and septin dynamics during cytokinesis. J. Cell Sci., 114:1379–1386.
- Lipschutz, J.H. and Mostov, K.E. 2002. Exocytosis: the many masters of the exocyst. *Curr. Biol.*, 12:R212–R214.
- Luca, F.C. and Winey, M 1998. MOB1, an essential yeast gene required for completion of mitosis and maintenance of ploidy. *Mol. Biol. Cell*, 9:29–46.
- Luca, F.C., Mody, M., Kurischko, C., Roof, D.M., Giddings, T.H. and Winey, M. 2001. Saccharomyces cerevisiae Mob1p is required for cytokinesis and mitotic exit. Mol. Cell Biol., 21:6972–6983.
- Mah, A.S., Jang, J. and Deshaies, R.J. 2001. Protein kinase Cdc15 activates the Dbf2-Mob1 kinase complex. *Proc. Natl. Acad. Sci. U.S.A.*, 98:7325–7330.
- Mah, A.S., Elia, A., E.H., Devgan, G., Ptacek, J., Schutkowski, M., Snyder, M., Yaffe, M.B. and Deshaies, R.J. 2005. Substrate specificity analysis of protein kinase complex Dbf2-Mob1 by peptide library and proteome array screening. *BMC Biochem.*, 6:22.
- Manning, G., Whyte, D.B., Martinez, R., Hunter, T. and Sudarsanam, S. 2002. The protein kinase complement of the human genome. *Science*, 298:1912–1934.
- McCormick. S. 1993. Male gametophyte development. *Plant Cell*, 5:1265–1275.
- McPherson, J.P., Tamblyn, L., Elia, A., Migon, E., Shehabeldin, A., Matysiak-Zablocki, E., Lemmers, B., Salmena, L., Hakem, A., Fish, J., Kassam, F., Squire, J., Bruneau, B.G., Hande, M.P., Hakem, R. 2004. Lats2/Kpm is required for embryonic development, proliferation control and genomic integrity. *EMBO J.*, 23:3677–3688.
- Morgan, D.O. 1996. The dynamics of cyclin dependent kinase structure. *Curr. Opin. Cell. Biol.*, 8:767–772.
- Moreno, C.S., Lane, W.S. and Pallas, D.C. 2001. A mammalian homolog of yeast MOB1 is both a member and a putative substrate of striatin family-protein phosphatase 2A complexes. J. Biol. Chem., 276:24253–24260.
- Mrkobrada, S., Boucher, L., Ceccarelli, D.F.J., Tyers, M. and Sicheri, F. 2006. Structural and functional analysis of *Saccharomyces cerevisiae* Mob1. J. Mol. Biol., 362:430–440.
- Murray, A.W. 2004. Recycling the cell cycle: cyclins revised. *Cell*, 116:221-234.
- Nei, M. and Rooney, A. P. 2005. Concerted and birth-and-death evolution of multigene families. *Annu. Rev. Genet.*, 39:121–52.
- Nelson, B., Kurischko, C., Horecka, J., Mody, M., Nair, P., Pratt, L., Zougman, A., McBroom, L., Hughes, T., Boone, C. and Luca, F. 2003. RAM: a conserved signaling network that regulates Ace2p transcriptional activity and polarized morphogenesis. *Mol. Biol. Cell*, 14:3782–3803.

Pantalacci, S., Tapon, N. and Leopold, P. 2003. The Salvador partner Hippo promotes apoptosis and cell-cycle exit in *Drosophila*. *Nature*. *Cell. Biol.*, 5:921–927.

- Park, C.J., Song, S.G., Lee, P.R., Shou, W.Y., Deshaies, R.J. and Lee, K.S. 2003. Loss of CDC5 function in *Saccharomyces cerevisiae* leads to defects in Swe1p regulation and Bfa1p/Bub2p-independent cytokinesis. *Genetics*, 163:21–33.
- Pennell, R.I. and Lamb, C. 1997. Programmed cell death in plants. *Plant Cell*, 9:1157–1168.
- Ponchon, L., Dumas, C., Kajava, A.V., Fesquet, D. and Padilla, A. 2004. NMR solution structure of Mob1, amitotic exit network protein and its interaction with an NDR kinase peptide, J. Mol. Biol., 337:167–182.
- Racki, W.J., Becam, A.M., Nasr, F. and Herbert, C.J. 2000. Cbk1p, a protein similar to the human myotonic dystrophy kinase, is essential for normal morphogenesis in *Saccharomyces cerevisiae*. *EMBO J.*, 19:4524–4532.
- Reiser, L. and Fisher, R.L. 1993. The ovule and the embryo sac. *The Plant Cell*, 5:1291–1301.
- Salimova, E., Sohrmann, M., Fournier, N. and Simanis, V. 2000. The *S. pombe* orthologue of the *S. cerevisiae* MOB1 gene is essential and functions in signaling the onset of septum formation. *J. Cell Sci.*, 113:1695–1704.
- Sherr, C.J. 2004. Principles of tumor suppression. Cell, 116:235-246.
- Shou, W.Y., Seol, J.H., Shevchenko, A., Baskerville, C., Moazed, D., Chen, Z.W.S, Jang, J., Shevchenko, A., Charbonneau, H. and Deshaies, R.J. 1999. Exit from mitosis is triggered by Tem1-dependent release of the protein phosphatase Cdc14 from nucleolar RENT complex. *Cell*, 97:233–244.
- Simanis, V. 2003. Events at the end of mitosis in the budding and fission yeasts. J. Cell Sci., 116:4263–4275.
- Stavridi, E.S., Harris, K.G., Huyen, Y., Bothos, J., Verwoerd, P.M., Stayrook, S.E., Pavletich, N.P., Jeffrey, P.D. and Luca, F.C. 2003. Crystal structure of a human Mob1 protein: toward understanding Mob-regulated cell cycle pathways. *Structure*, 11:1163–1170.
- Stegert, M.R., Hergovich, A., Tamaskovic, R., Bichsel, S.J. and Hemmings, B.A. 2005. Regulation of NDR protein kinase by hydrophobic motif phosphorylation mediated by the mammalian Ste20-like kinase MST3. *Mol. Cell. Biol.*, 25:11019–11029.
- Stegmeier, F., Visintin, R. and Amon, A. 2002. Separase, polo kinase, the kinetochore protein Slk19, and Spo12 function in a network that controls Cdc14 localization during early anaphase. *Cell*, 108:207–220.
- Stegmeier, F., and Amon, A. 2004. Closing mitosis: the functions of the Cdc14 phosphatase and its regulation. *Annu. Rev. Genet.*, 38:203–232.
- Stoepel, J., Ottey, M.A., Kurischko, C., Hieter, P. and Luca, F.C. 2005. The mitotic exit network Mob1p-Dbf2p kinase complex localizes to the nucleus and regulates passenger protein localization. *Mol. Biol. Cell*, 16:5465–5479.
- Sonnhammer, E.L.L., Eddy, S.R., Birney, E., Bateman, A. and Durbin, R. 1998. Pfam: multiple sequence alignments and HMM-profiles of protein domains. *Nucleic Acids Res.*, 26:320–322.

- St John, M.A., Tao, W., Fei, X., Fukumoto, R., Carcangiu, M.L., Brownstein, D. G., Parlow, A. F., McGrath, J. and Xu, T. 1999. Mice deficient of Lats1 develop soft-tissue sarcomas, ovarian tumours and pituitary dysfunction. *Nature Genet.*, 21:182–186.
- Takahashi, Y., Miyoshi, Y., Takahata, C., Irahara, N., Taguchi, T., Tamaki, Y. and Noguchi, S. 2005. Down-regulation of LATS1 and LATS2 mRNA expression by promoter hypermethylation and its association with biologically aggressive phenotype in human breast cancers. *Clin. Cancer Res.*, 11:1380–1385.
- Tapon, N., Harvey, K.F., Bell, D.W., Wahrer, D.C., Schiripo, T.A., Haber, D.A. and Hariharan, I.K. 2002. Salvador promotes both cell cycle exit and apoptosis in *Drosophila* and is mutated in human cancer cell lines. *Cell*, 110:467–478.
- Terbush, D.R., Maurice, T., Roth, D. and Novick, P. 1996. The Exocyst is a multiprotein complex required for exocytosis in *Saccharomyces cerevisiae*. *EMBO J.*, 15:6483–6494.
- Van Damme, D., Bouget, F.Y., Van Poucke, K., Inzé, D. and Geelen, D. 2004. Molecular dissection of plant cytokinesis and phragmoplast structure: a survey of GFP-tagged proteins. *Plant J.*, 40:386–398.
- Vaux, D.L. and Korsmeyer, S.J. 1999. Cell death in development. *Cell*, 96:245–254.
- Verde, F., Wiley, D.J. and Nurse, P. 1998. Fission yeast Orb6, a ser/thr protein kinase related to mammalian rho kinase and myotonic dystrophy kinase, is required for maintenance of cell polarity and coordinates cell morphogenesis with the cell cycle. *Proc. Natl. Acad. Sci. U.S.A.*, 95:7526–7531.
- Walton, F., Heitman, J. and Idnurm, A. 2006. Conserved elements of the RAM signaling pathway establish cell polarity in the Basidiomycete *Cryptococcus neoformans* in a divergent fashion from other fungi. *Mol. Biol. Cell Vol.*, 17:3768–3780.
- Weiss, E.L., Kurischko, C., Zhang, C., Shokat, K., Drubin, D.G. and Luca, F.C. 2002. The *Saccharomyces cerevisiae* Mob2p-Cbk1p kinase complex promotes polarized growth and acts with the mitotic exit network to facilitate daughter cell-specific localization of Ace2p transcription factor. J. Cell Biol., 158:885–900.
- Wolfe, B.A. and Gould, K.L. 2005. Split decisions: coordinating cytokinesis in yeast. *Trends Cell Biol.*, 15(1):10–18.
- Wu, H.M. and Cheung, A.Y. 2000. Programmed cell death in plant reproduction. *Plant. Mol. Biol.*, 44:267–281.
- Wu, S., Huang, J., Dong, J. and Pan, D. 2003. Hippo encodes a Ste-20 family protein kinase that restricts cell proliferation and promotes apoptosis in conjunction with salvador and warts. *Cell*, 114:445–456.
- Xu, T., Wang, W., Zhang, S., Stewart, R.A. and Yu, W. 1995. Identifying tumor suppressors in genetic mosaics: the *Drosophila* lats gene encodes a putative protein kinase. *Development*, 121:1053–1063.
- Zallen, J.A., Peckol, E.L., Tobin, D.M. and Bargmann, C.I. 2000. Neuronal cell shape and neurite initiation are regulated by the Ndr kinase SAX-1, a member of the Orb6/COT-1/warts serine/threonine kinase family. *Mol. Biol. Cell*, 11:3177–3190.

## **Supplementary Information**

**Table 1S.** The ?rst column reports the organism name, the second one the gene code whereas the third column the Mob class. Finally the fourth column shows the code number used in the multiple alignment in Figure 1S.

Organism	Protein ID	Mob aroup	Multiple Alignment Numbe
Schizosaccharomyces pombe 972h-	NP 595191		130
Schizosaccharomyces pombe 972h-	NP 587851	-	131
Caenorhabditis elegans	NP 510184	-	84
Caenorhabditis elegans	NP 502248	-	86
Saccharomyces cerevisiae	NP_116618	-	148
Saccharomyces cerevisiae	NP_012160	-	147
Caenorhabditis briggsae	CAE62136	-	16
Caenorhabditis briggsae	CAE61392	-	15
Tribolium castaneum	XP_971775	Mob1	132
Bos taurus	XP_871266	Mob1	164
Canis familiaris	XP_866427	Mob1	36
Canis familiaris	XP_852858	Mob1	27
Strongylocentrotus purpuratus	XP_788775	Mob1	96
Bos taurus	XP_593426	Mob1	166
Canis familiaris	XP_539306	Mob1	29
Pan troglodytes	XP_515735	Mob1	194
Gallus gallus	XP_427212	Mob1	141
Gallus gallus	XP_420601	Mob1	136
Apis mellifera	XP_393046	Mob1	149
Pan troglodytes	XP_001159136	Mob1	196
Pan troglodytes	XP_001153960	Mob1	197
Pan troglodytes	XP_001153899	Mob1	201
Macaca mulatta	XP_001110736	Mob1	182
Macaca mulatta	XP_001110694	Mob1	184
Macaca mulatta	XP_001107567	Mob1	180
Rattus norvegicus	XP_001075592	Mob1	88
Rattus norvegicus	XP_001073264	Mob1	90
Rattus norvegicus	XP_001068056	Mob1	87
Rattus norvegicus	XP_001068001	Mob1	92
Takifugu rubripes	SINFRUP00000155283	Mob1	107
Takifugu rubripes	SINFRUP00000150734	Mob1	106
Danio rerio	NP_999948	Mob1	38
Danio rerio	NP_956494	Mobl	37
Danio rerio	NP_956208	Mob1	39
Homo sapiens	NP_//5/39	Mobl	1/5
Mus musculus	NP_663546	Mobl	1
Drosophila melanogaster	NP_651041	Mob1	11
Mus musculus	NP_081011	Mob1	3
Homo sapiens	NP_060691	Mob1	173
Xenopus tropicalis	NP_001072572	MoD1	76
Nemotostella vestensia	NP_001017026	Mob1	/5
	jgillieinveil244739	Mob1	03
		Mob1	169
Monodolphis domostica	ENSOREF00000007848	Mob1	186
Gastorostous, aculaatus	ENSCACP00000013120	Mob1	180
Gasterosteus aculeatus	ENSGACF00000020945	Mob1	40
Anonheles gambiae	ENSANGP0000011008	Mob1	18
Andes acounti	ENSANGI 00000019090	Mob1	113
Tetraodon nigroviridis	CAG08455	Mob1	100
Tetraodon nigroviridis	CAE97101	Mob1	101
Schistosoma japonicum	AAX26825	Mob1	153
	ΔΔΤ66503	Mob1	123
	AAH74352	Mob1	123
Xenopus laevis	1R3BA	Mob1	124
Tribolium castaneum	XP 968602	Mob2	134
Canis familiaris	XP 854258	Mob2	35
Strongylocentrotus purpuratus	XP 782014	Mob2	99
Danio rerio	XP 694168	Mob2	44
Gallus gallus	XP 421030	Mob2	140
Apis mellifera	XP 392406	Mob2	151
Strongylocentrotus purpuratus	XP 001196170	Mob2	98
Pan troglodytes	XP 001152532	Mob2	200

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Rattus norvegicus	XP_001063786	Mob2	95
Takifugu rubripes	SINFRUP00000148580	Mob2	111
Takifugu rubripes	SINFRUP00000129967	Mob2	112
Xenopus tropicalis	NP_989013	Mob2	81
Homo sapions	NP_729716	Mob2	170
	NP_082584	Mob2	7
Xenopus tropicalis	NP_001008166	Mob2	82
Danio rerio	NP 001002364	Mob2	43
Nematostella vectensis	jgi Nemve1 227021	Mob2	64
Ciona intestinalis	jgi Cioin2 254445	Mob2	172
Oryzias latipes	ENSORLP00000011487	Mob2	163
Gasterosteus aculeatus	ENSGACP00000017036	Mob2	51
Gasterosteus aculeatus	ENSGACP00000014609	Mob2	50
Anopheles gambiae	ENSANGP00000018173	Mob2	20
Aedes aegypti	EAT40339	Mob2	115
Drosophila pseudoobscura	EAL29650	Mob2	117
letraodon nigroviridis	CAG01535	MoD2	105
Xenopus laevis	AAHO1102	Mob2	129
Tribolium castaneum	XP 972981	Mob3	135
Canis familiaris	XP 857401	Mob3	34
Canis familiaris	XP 536018	Mob3	33
Pan troglodytes	XP_516009	Mob3	199
Gallus gallus	XP_428779	Mob3	142
Gallus gallus	XP_426564	Mob3	139
Apis mellifera	XP_394425	Mob3	152
Strongylocentrotus purpuratus	XP_001185390	Mob3	97
Rattus norvegicus	XP_001062295	Mob3	94
Takifugu rubripes	SINFRUP00000143894	Mob3	110
Homo sapiens	NP_955776	MoD3	1/8
Caeporbabditis elegans	NP_010229	Mob3	12
Mus musculus	NP 079559	Mob3	6
Sus scrofa	NP 001027551	Mob3	67
Xenopus tropicalis	NP 001017210	Mob3	80
Danio rerio	NP_001003439	Mob3	42
Nematostella vectensis	jgi Nemve1 116750	Mob3	59
Ciona intestinalis	jgi Cioin2 265674	Mob3	171
Paramecium tetraurelia	GSPATP00005136001	Mob3	71
Oryzias latipes	ENSORLP00000025857	Mob3	162
Oryctolagus cuniculus	ENSOCUP0000004946	Mob3	24
Monodelphis domestica	ENSMODP0000015412	MoD3	192
		Mob3	191
Anonheles gambiae	ENSANGP0000023372	Mob3	21
Aedes aegypti	EAT45255	Mob3	116
Drosophila pseudoobscura	EAL24719	Mob3	118
Tetraodon nigroviridis	CAG02595	Mob3	104
Caenorhabditis briggsae	CAE70187	Mob3	17
Sus scrofa	AAX68443	Mob3	68
Schistosoma japonicum	AAW25644	Mob3	155
Xenopus laevis	AAH88913	Mob3	127
Tribolium castaneum	XP_969194	Mob4	133
Apis mellifera	XP_396081	MoD4	10
Caeporbabditis elegans	NP 501179	Mob4	10
Naegleria gruberi	igilNaegr1180300	Mob4	58
Naegleria gruberi	igilNaegr1170019	Mob4	57
Naegleria gruberi	jgi Naegr1 34457	Mob4	62
Paramecium tetraurelia	GSPATP00019838001	Mob4	70
Paramecium tetraurelia	GSPATP00009352001	Mob4	69
Anopheles gambiae	ENSANGP00000025093	Mob4	19
Aedes aegypti	EAT40974	Mob4	114
Caenorhabditis briggsae	CAE61872	Mob4	14
Schistosoma japonicum	AAX30250	Mob4	156
Schistosoma japonicum	AAWZ4/93	Mob4a	154
Canis familiaris	XP 539625	Mob4a	700
Gallus gallus	XP 422452	Mob4a	138
Pan troglodytes	XP 001162561	Mob4a	198
Macaca mulatta	XP_001108354	Mob4a	183
Rattus norvegicus	XP_001064295	Mob4a	93
Takifugu rubripes	SINFRUP00000171937	Mob4a	109

Homo sapiens	NP_958805	Mob4a	177
Mus musculus	NP_780517	Mob4a	5
Danio rerio	NP_001002191	Mob4a	41
Xenopus tropicalis	jgi Xentr4 296225	Mob4a	79
Ciona intestinalis	jgi Cioin2 206559	Mob4a	170
Oryzias latipes	ENSORLP00000020718	Mob4a	160
Monodelphis domestica	ENSMODP0000002353	Mob4a	190
Loxodonta africana	ENSLAFP00000002217	Mob4a	55
Gasterosteus aculeatus	ENSGACP00000015732	Mob4a	48
Gasterosteus aculeatus	ENSGACP00000015729	Mob4a	47
Tetraodon nigroviridis	CAF95835	Mob4a	103
Xenopus laevis	AAH73205	Mob4a	126
Mus musculus	XP_987898	Mob4b	9
Canis familiaris	XP_855034	Mob4b	31
Bos taurus	XP_613282	Mob4b	165
Bos taurus	XP_581226	Mob4b	167
Canis familiaris	XP_531966	Mob4b	28
Gallus gallus	XP_429197	Mob4b	137
Rattus norvegicus	XP_343162	Mob4b	91
Pan troglodytes	XP_001154635	Mob4b	195
Macaca mulatta	XP_001108825	Mob4b	185
Macaca mulatta	XP_001104813	Mob4b	181
Rattus norvegicus	XP_001065615	Mob4b	89
Mus musculus	XP_001000067	Mob4b	4
Mus musculus	XP_001000051	Mob4b	8
Takifugu rubripes	SINFRUP00000157959	Mob4b	108
Danio rerio	NP_956010	Mob4b	40
Mus musculus	NP_835162	Mob4b	2
Homo sapiens	NP_570719	Mob4b	176
Homo sapiens	NP_079037	Mob4b	174
Xenopus tropicalis	NP_001011080	Mob4b	78
Xenopus tropicalis	jgi Xentr4 464458	Mob4b	77
Oryzias latipes	ENSORLP00000011358	Mob4b	159
Oryzias latipes	ENSORLP00000011352	Mob4b	158
Oryctolagus cuniculus	ENSOCUP00000015183	Mob4b	22
Monodelphis domestica	ENSMODP0000005633	Mob4b	188
Monodelphis domestica	ENSMODP0000005289	Mob4b	187
Loxodonta africana	ENSLAFP00000007392	Mob4b	52
Loxodonta africana	ENSLAFP0000000390	Mob4b	53
Tetraodon nigroviridis	CAG00735	Mob4b	102
Xenopus laevis	AAI03736	Mob4b	125
Arabidopsis thaliana	NP_199368	Mobp	143
Arabidopsis thaliana	NP_197544	Mobp	145
Arabidopsis thaliana	NP_197543	Mobp	146
Arabidopsis thaliana	NP_193640	Mobp	144
Oryza sativa (japonica cultivar-group)	NP_001064531	Mobp	120
Oryza sativa (japonica cultivar-group)	NP_001050541	Mobp	119
Oryza sativa (japonica cultivar-group)	NP_001050340	Mobp	121
Populus trichocarpa	jgi Poptr1_1 836365	Mobp	65
Populus trichocarpa	jgi Poptr1_1 589279	Mobp	61
Chlamydomonas reinhardtii	jgi Chlre3 133994	Mobp	66
Medicago sativa subsp. falcata	CAJ44124	Mobp	73
Medicago sativa subsp. falcata	CAG25780	Mobp	74
Medicago sativa subsp. falcata	CAC41010	Mobp	72
Medicago truncatula	AP006361_12.1	Mobp	202

		20		40		60	E.	80		
175		MSFLFG- SRS	SKTEKPKKNI	PEGSHOYELL	KHAEATLGSG	- N- LBMAVML	P- EGEDENEW	VAVNTVDEEN	QINMLYGTIT 76	5
2	• • • • • • • • • • • • • • • • • • •	ALKOVENK	DKTERPKRKE	EPGTORFELH	KRAQASLNSG	VD- LBAAVQL	P-NGEDONDW	VAVHVVDEEN	RINLIYGTIC 79	3
89	MSI	ALKOVENK	DKTERPKRKE	EPGTORFELH	KRAQASLNSG	VD- LRAAVQL	P- SGEDQNDW	VAVHVVDEEN	RINLINGTIC 79	3
28	······································	ALKOVENK	DKTERPKRKE	EPGTORFELH	KRAQASLNSG	VD- LKAAVQL	P- SGEDONDW	VAVHVVDFFN	RINLINGTIC 79	3
174	MSI	ALKOVEN	DKTERPKRKE	EPGTOREELH	KRAQASLNSG	VD- LKAAVOL	P. SGEDONDW	VAVHVVDEEN	BINLINGTIC 79	2
181	MSI	ALKOVENK	DKTERPKRKE	EPGTORFELH	KRAQASLNSG	VD- LKAAVQL	P- SGEDONDW	VAVHVVDEEN	RINLINGTIC 79	9
195	MSI	ALKOVEN K	DKTERPKRKE	EPGTORFELH	KRAQASLNSG	VD- LKAAVQL	P- SGEDONDW	VAVHVVDEEN	RINLINGTIC 79	3
22	MSI	ALKOVENK	DKTERPKRKE	EPGTORFELH	KRAQA SLN SG	VD- LKAAVQL	P- SGEDONDW	VAVHVVDEEN	RINLIYGTIC 79	3
187	MSI	ALKOVEN K	DKTERPKRKE	EPGTORFELH	KRAQASLNSG	VD- LKAAVQL	P- SGEDONDW	VAVHVVDEEN	RINLINGTIC 79	3
127	UCI		ERPKRKE	EPGTOREELH	KRAQASLNSG	VD-LKAAVQL	P- SGEDONDW	VAVHVVDEEN	RINLINGTIC 65	2
77	MSI	GLKOVEN	DKTERPKRKE	DPGTOREELH	KRAQASITSG	VD- LKATVOL	P. TGEDINDW	VAVHVVDEEN	BINLINGTVC 79	5
4	SNP	FLKOVENK	DKTERPKRKE	EPGTORFELH	KRAQASLNAG	LD- LRLAVOL	P- PGEDLNDW	VAVHVVDEEN	RINLINGTIS 79	9
91	SNP	FLKQVENK	DKTERPKRKE	EPGTORFELH	KRAQASENAG	LD- LRLAVQL	P- PGEDLNDW	VAVHVVDEEN	RVNLIYGTIS 79	
8	••••• SN P	FLKQVFNK	DKTERPKRKE	EPGTORFELH	KRAQASLNAG	LD- LRLAVQL	P- PGEDLNDW	VAVHVVDFFN	RINLIYGTIS 79	3
185	····· SNP	FLKOVENK	DKTERPKRKE	EPGTORFELH	KKAQASLNAG	LD- LRLAVOL	P- PGEDLNDW	VAVHVVDEEN	RVNLINGTIS 79	3
176	SNP	ELKOVENK	DKTERPKRKE	FPGTOREELH	KKAQASENAG	LD- LBLAVOL	P- PGEDLNDW	VAVHVVDEEN	RVNLLVGTLS 79	2
167	SNP	FLKOVENK	DKTERPKRKE	EPGTORFELH	KKAQASENAG	LD- LKLAVOL	P-AGEELNDW	VAVHVVDEEN	RVNLIYGTIS 79	9
53	SNP	FLKQVEN K	DKTERPKRKE	EPGTORFELH	KRAQASLNAG	LD- LRLAVQL	P- PGEDLHDW	VAVHVVDEEN	RVNLIYGTVS 79	3
78	MSNP	- LKQVEN K	DRTERPKRKE	EPGTORFELH	KKAQA SENAG	LD- LKLAVQL	P-HGEDLNDW	VAVHVVDEEN	RINLIYGTVS 79	3
125	····· MSNP	- LKOVEN K	DRTERPKRKE	EPGTOREELH	KKAQASLNAG	LD- LKLAVQL	P-HGEDLNDW	VAVHVVDEEN	RINLINGTIS 79	3
102	MSN	ALKOVENK	DRTERPKEKE	EPGTOREELH	KKAQASENAG	LD- LKOAVOL	P. HGEDINDW	VAVHVVDEEN	RINLINGTIS 79	2
108	MSN	ALKOVENK	DRTERPKRKE	EPGTORFELH	KKAQASLNAG	LD- LKQAVQL	P-HGEDENDW	VAVHVVDEEN	BINLINGTIS 79	à
158	MSM	ALKOVENK	DRTERPKRKE	EPGTORFELH	KKAQASLNAG	LD- LKHAVQL	P-HGEDLNDW	VAVHVVDEEN	RINLIYGTIS 79	
159	MSM	ALKOVENK	DRTERPKRKE	EPGTORFELH	KKAQASLNAG	LD- LKHAVQL	P- HGEDLNDW	VAVHVVDFFN	RINLINGTIS 79	3
40	• • • • • • • • <mark>M SM</mark>	ALKOVENK	DRTERPKRKE	EPGTORFELH	KKAQASLNAG	LD- LKQAVQL	P-HGEDLNDW	VAVHVVDFFN	RINLIYGTIS 79	3
170		NLKSVEN K	EKTERPKKHE	EPGTIKEDLH	KKAQASLRSG	LD-LKAIVVL	P- SGEDENDW	AVHVVDEEN	RINLINGTVS 79	
93	MAL	CI KOVEAK	DKTERPRKRE	EPGTOREELY	KKAQASLKSG	LD-LRSVVRL	P. PGESIDDW	LAVHYVDEEN	RINLINGTMA 79	5
177	MAL	CLKOVEAK	DKTERPRKRE	EPGTORFELY	KKAQASLKSG	LD- LRSVVRL	P- PGENIDDW	IAVHVVDEEN	BINLIYGTMA 79	3
198	MAL	CLKQVFAK	DKTERPRKRE	EPGTORFELY	KKAQA SLKSG	LD- LRSVVRL	P- PGENIDDW	IAVHVVDFFN	RINLIYGTMA 79	)
183	MAL	CLKOVFAK	DKTERPRKRE	EPGTORFELY	KKAQASLKSG	LD- LRSVVRL	P- PGENIDDW	IAVHVVDFFN	RINLINGTMA 79	3
168	MAL	CLKOVES K	DKTERPRKRE	EPGTORFELY	KKAQASLKSG	LD- LRSVVRL	P- PGENIDDW	IAVHVVDEEN	RINLINGTMA 79	1
190	MAL	CLKOVENK	DKTERPRKRE	EPGTERFELY	KKAQASLKSG	LD- LBAVVEL	P- PGESINDW	LAVHYVDEEN	BINLIYGTMG 79	2
79	MAL	CLNOVEN K	DKTERPRKKE	EPGTORFELY	KKAQASLKSG	LD- LKTVVOL	P- PGENINDW	IAVHVVDFFN	RINLIYGTMS 79	9
126	MAL	CLNQVEN K	DRTERPRKKE	EPGTORFELY	KKAQA SLKSG	LD- LKTVVQL	P- PGENINDW	IAVHVVDFFN	RINLIYGTMS 79	3
138	MAL	CLKOVENK	DKTERPRKKE	EPGTORFELY	KKAQASLKSG	LD- LKAVVQL	P- PGESINDW	IAVHVVDFFN	RINLIYGTMS 79	3
47	MAL	CLGOVESK	DKTERPRKRE	EPGTORFELY	KKAQASLKSG	LD-LRKVVQL	P-EGENINDW	LAVHYVDEEN	RINLINGTVS 79	3
103	MAL	CLGOVES	OKTERPRERE	EPGTOREELY	KKAQASLKSG	LD- LBKVVOL	P. EGENISOW	LAVHYVDEEN	BINLIYGTMS 79	5
109		CLGOVES K	DKTERPRKRE	EPGTORFELY	KKAQASLKSG	LD- LRKVVQL	P- EGENI SDW	IAVHVVDEEN	RINLIYGTMS 79	9
160	MAL	CLGQVFSK	DKTERPKKRE	EPGTORFELY	KKAQASLKSG	LD- LRKVVQL	P- EGESENDW	IAVHVVDFFN	RINLIYGTMS 79	3
41	MAL	CLGOVES K	DKTERPRKRE	EPGTORFELY	KRAQASLKSG	LD- LRKVVQL	P-EGESINDW	IAVHVVDEEN	RINLIYGTVS 79	3
55	ALN	CLKOVESK	DKTERPRKRE	EPGTORFELY	KKAQASLKSG	LD-LRSVVRL	P- PGESIDDW		RINLINGTIS 79	1
114		GELEFFO R	EKTERPKKKE	TOGTIRYSLH	KOAHASLOSG	IN- LREVVKL	P- PGENMNDW	LAVHYVDEEN	BINLINGTIS 79	à
10	ALN	GFLEFFQ K	GKTERPKKPE	ASGTIRYSLH	KQAQASLOSG	IN- LROVVRL	P- QGENLNDW	LAVHVVDFFN	RINLIYGTVS 79	9
133	ALN	GFFDFFQK	GKTERPKKKE	THGTIRYSLH	KQACASLNSG	IN- LRSAVKL	P- EGEDLNDW	IAVHVVDEEN	RINLIYGTIS 79	3
150	ALS	GEMEFEQ K	GKTERPKKKE	AHGTLRYSLH	KQAQASENSG	IN- LRSVVKL	P- PGEDLNDW	IAVHVVDEEN	RINLINGTVS 79	3
83		SELDELOVNK	HKTERPKKKE	POGTLEYSLH	KOAEATLHSG	VD- LRHAVKL	P- PSENEDDW	LAVHTVDEEN	RINIMYGTIS 79	6
154	AEN	GEKELEVK	QKTERPKKKE	APDTIBYHLH	KHAEASLSAG	ID- LREAVKK	P-DEEELNDW	LAVHVVDEYN	BINLINGTIC 79	à
156						LREAVKK	P- DEEELNDW	TAVHVVDFYN	RINLIYGTIC 36	5
58	IVN	NLKNALD R	NKTERPKHKE	IKGSNOHTLH	KYKKET LG	SGOLTEAVKL	P- QDENLNEW	LAINTVDFYN	TTNLLYGSLG 78	3
59		GLMGVSD K	SNTERDER	SHOCKBERNEN	AMOKSTSSLG	SCREENTVKL	P- PGEKKNEW	LAVHVUDEVN	GINILYGSLE 78	5
92		MSELES- SRS	SKTEKPKKNI	PEGSHOYELL	KHAEATLGSG	- N- LBOAVML	P- EGEDLNEW	LAVNTVDEEN	OINMLYGTIT 76	5
197		MSFLFS- SRS	SKTEKPKKNI	PEGSHOYELL	KHAEATLGSG	- N- LRQAVML	P- EGEDLNEW	LAVNTVDEEN	QINMLYGTIT 76	5
184		MSFLFS- SCS	SKTEKLKKNI	PEGSHQYELL	KHAEATLGSE	- N- LRQAVML	P- EGEDENEW	IAVNTVDEEN	QINMLYGTIT 76	5
201		MSELES- SRS	SKTEKPKKNI	PEGSHOYELL	KHAEATLGSG	- N- LRQAVML	P-EGEDLNEW	IAVNTVDEEN	QINMLYGTIT 76	ŝ
182		MSELES. SPS	SKTEKPKKNI	PEGSHOVELL	KHAEATLGSE	- N- LHQAVML	P-EGEDINEW	LAVNTVDEEN	OINMLYGTIT 70	5
173		MSELES- SRS	SKTEKPKKNI	PEGSHOYELL	KHAEATLGSG	- N- LBOAVML	P-EGEDLNEW	LAVNTVDEEN	QINMLYGTIT 76	5
194		MSFLFS- SRS	SKTEKPKKNI	PEGSHQYELL	KHAEATLGSG	- N- LRQAVML	P- EGEDLNEW	IAVNTVDEEN	QINMLYGTIT 70	5
164		MSFLFS- SRS	SKTEKPKKNI	PEGSHQYELL	KHAEAT LGSG	- N- LRQAVML	P- EGEDLNEW	IAVNTVDEEN	QINMLYGTIT 76	5
87	• • • • • • • • • • • •	MSELES- SRS	SKTEKPKKNI	PEGSHQYELL	KHAEATLGSG	- N- LRQAVML	P-EGEDENEW	AVNTVDFFN	QINMLYGTIT 76	ć
2/		MSELES- SHS	SKTEKPKKNI	PEGSHOVELL	KHAFATLGSG	N. LPOAVML	P. EGEDENEW	LAVNTVDEEN	OLNMLYGTIT 76	2
180		ESCPES- SES	SKTEKPKKNI	PEGSHOYELL	KHAEATLGSG	- N- LBOAVML	P- EGEDENEW	LAVNTVDEEN	QINMLYGTIT 78	8
186		CS- S- SRS	SKTEKPKKNI	PEGSHOYELL	KHAEATLGSG	- N- LRQAVML	P- EGEDENEW	TAVNTVDEEN	QINMLYGTIT 7	3
100		S- SRS	SKTEKPKKNI	PEGSHQYELL	KHAEATLGSG	- N- LRQAVML	P- EGEDLNEW	IAVNTVDEEN	QINMLYGTIT 71	L
106	· · · · · · · · · · · · · · · · · · ·	MSELEG- SHS	SKTEKPKKNI	PEGSHQYELL	KHAEATLGSG	- N- LRQAVML	P-EGEDENEW	TAVNTVDEEN	QINMLYGTIT 78	5
30		MSELES. SPS	SKTEKPKKNI	PEGSHOVELL	KHAFATLOSO	N- LROAVML	P. EGEDENEW	LAVNTYDEEN	OLNMINGTIT 74	5
157		MSFLFG- SRS	SKTEKPKKNI	PEGSHOYELL	KHAEATLGSG	- N- LROAVML	P- EGEDENEW	LAVNTVDEEN	QINMLYGTIT 76	5
45		MSFLFG- NRS	SKTEKPKKNI	PEGSHQYELL	KHAEATLGSG	- N- LRQAVML	P- EGEDLNEW	TAVNTVDEEN	QINMLYGTIT 70	5
124		MGS	SHHHH	HHSSGLV	PRGSATLGSG	- N- LRQAVML	P- EGEDLNEW	TAVNTVDEEN	QINMLYGTIT 62	2
141	•••••		SKTEKOKKNE	PECSHOVELL	KHA BATEORO		P. FORDENER		OLNMI VOTIT 7	
30		MSELEG. NPS	SKTEKPKKNI	PEGSHOVELL	KHAEATLOSG	- N- LBAAVML	P-EGEDINGW	LAVNTYDEEN	QINMLYGTIT 76	5
3		MSFLFG- SRS	SKTEKPKKNI	PEGSHOYELL	KHAEATLGSG	- N- LBMAVML	P- EGEDLNEW	VAVNTVDEEN	QINMLYGTIT 76	5

90										
~~		MSELEG. SRS	SKTEKPKKNI	PEGSHOVELL	KHAFATEGSG	- N- ERMAVME	P. EGEDINEW	VAUNTUDEEN	OLNMENGT IT	76
166	watere alle energy	MSELEG. SRS	SKTEKPKKNI	REGSHOVELL	KHAFATIGSG	N. I BMAVMI	P. EGEDINEW	VAVNTVDEEN	OLNMINGTIT	76
136		MSELVG. SRS	SKTEKPKKNI	PEGSHOVELL	KHAFATLGSG	- N. I BMAVMI	P. EGEDINEW	VAVNTVDEEN	OINMLYGTIT	76
29	GT	LEVIEG. SES	SKTEKPKKNI	PEGSHOVELL	KHAFATLGSG	N. LRMAVMI	P. EGEDINEW	VAVNTVDEEN	OLNMLYGT IT	78
106		DVNESC. SPS	SKTEKPKKNI	PECSHOVELL	KHAFATLOSO		P. EGEDINEW	VAVNTVDEEN	OLNME VOTIT	78
101		MSELEG. NEG	NETEKOKKNI	PECSHOVELL	KHAEATLOSG	N. I PMAYME	P. DGEDLNEW	VAVNTVDEEN	OLNML VOT IT	76
107		MCELEA, NPC	NETEKOKKNI	PECSHOVELL	KHAEATLOSG		P. DCEDINEW	VAVNTVDEEN	OLNML VOT LA	76
107		MOFLEA- NHG	OKTEKOKKUL	PEGSHOVELL	KHALATLOSG		P. DGEDENEW	VAVINTUDEEN	O INMETOTTA	70
40		MSFLFG-NHS	SKIFKPKKNI	PEGSHQYELL	KHAEATEGSG	- N- LHMAVML	P-EGEDENEW	VAVNIVDEEN	GINMLYGITT	10
37		MSFLFG- SHS	SKTEKPKKNI	PEGSHQYELL	KHAEATLGSG	- N- LHMAVML	P-DGEDENEW	VAVNTVDEEN	QINMLYGTIT	76
76		MSFLFG- SHS	SKTEKPKKSL	PEGSHQYELL	KHAEATLGSG	- N- LRMAVML	P-EGEDENEW	VAVNTVDEEN	QINMLYGTIT	76
123		MSFLFG-NRS	SKTEKPKKSL	PEGSHQYELL	KHAEATLGSG	- N- LRMAVML	P-EGEDLNEW	VAVNTVDEEN	QINMLYGTIT	76
38	•••••	MSFLFG-NRS	SKTFKPKKNI	PEGSHQYELL	KHAEATLGSG	- N- LRMAVML	P- EGEDLNEW	VAVNTVDEEN	QINMLYGTIT	76
18		FHFPFPGSRS	SKTEKPKKNI	PEGTHQYDLM	KHAAATLGSG	- N- LRNAVQL	P-DGEDLNEW	VAVNTVDEEN	QINMLYGTIT	77
113		MSFLF RS	SKTEKPKKNI	PEGTHQYDLM	KHAAATLGSG	- N- LRNAVQL	P-DGEDLNEW	VAVNTVDFFN	QINMLYGTIT	74
11		MDFLFG- SRS	SKTEKPKKNI	PEGTHQYDLM	KHAAATLGSG	- N- LRNAVAL	P-DGEDLNEW	VAVNTVDFFN	QINMLYGTIT	76
132		MSFLFG- SRS	SKTFKPKKNI	PEGTHQYELM	KHAAATLGSG	- N- LRLAVML	P- EGEDLNEW	VAVNTVDEEN	QINMLYGTIT	76
149		MSFLFG- SRS	SKTEKPKKNI	PEGTHQYDLM	KHAAATLGSG	- N- LRLAVML	P- EGEDLNEW	VAVNTVDFFN	QINMLYGTIT	76
61		MSFLFG- SRS	TKTEKPKKNI	PEGTHOYDLM	RHAAATLGSG	- N- LRLAVML	P- EGEDLNEW	VAVNTVDEEN	QINMLYGTIT	76
96		MNFFSS RG	AKTERPKKNI	PEGEHQYELM	KHAEATLGSG	- N- LRQAVSL	P-DGEDINEW	VAVNTVDEEN	QINMLYGTIT	75
169		MSFFFQ-NRH	NKTFKPHKSI	PEGSHQHELI	RHAAATLGSG	- N- LQLAVAL	P- EGEDLNEW	IAVNTVDFFN	QINMLYGTIS	76
153	E	TNNNTSATTT	<b>TNNNNNT SNH</b>	DGSNKQHDIL	PETAATLGSG	- D- LRLAVRL	P- EGEDLHEW	<b>IAINTVDFFN</b>	QINMLYGTLL	78
69		LKFKMQ- PTD	SKTEKPLKQI	DKNORGYGLR	QIAQMTLGSG	- N- MLLAVEL	P-KGEDLNEW	LAVNTIEFYN	EISILYGTLV	78
70		MQ- TAD	PKTYKPLKQI	DKNORGYGLK	QLAQMTLGSG	- N- MLLAVEL	P-NGEDLNEW	LAVNTIEFYN	EISILYGTLV	72
64	MS	SLEGLG BN	ORTERPKKSA	PSGSKGAOLR	KHIDATLGSG	- N- LREAVEL	P- PGEDLNEW	LAVNTVDEEN	OVNELEGTET	77
119	M	SLEGLG EN	QKTERPKKSA	PSGSKGAQLE	KHIDATLGSG	- N- LBEAVEL	P- PGEDINEW	LAVNTVDEEN	QVNLLYGTLA	76
143	M	SLEGLG BN	QKTERPKKSA	PSGSKGAOLE	KHIDATLGSG	- N- LBEAVEL	P- PGEDANEW	LAVNTVDEEN	QUNELYGTET	76
73		SLEGLGS- BN	QKTERPKKSA	PTGSKGAQLO	KHIDATLGSG	- N- LBEAVEL	P- PGEDINEW	LAVNTVDEEN	QUNTMEGTET	77
74		SLEGLGS- BN	OKTERPKKSA	PTGSKGAQLO	KHIDATLGSG	- N- LBEAVEL	P- PGEDINEW	LAVNTVDEEN	QUNTMEGTET	77
72		SLEGLOS. PN	OKTERPKKSA	PTGSKGAOLO	KHIDATIGSG	- N- LBEAVEL	P- PGEDINEW	LAVNTVDEEN	OVNTMEGTET	77
202		SLEGLOS. PN	OKTERPKKSA	PSGSKGAOLO	KHIDATIGSG	- N- LBEAVEL	P- PGEDINEW	LAVNTVDEEN	OVNILEGTET	77
65		SI EGLOS, PN	OKTERPKKNA	PSGSKGAOLO	RHIDATICSC	N. I PEAVEL	P. PGEDINEW	LAVNTVDEEN	OVNILVOTIT	77
144	II.	DWNIMED. IN	OKTEPPKKSA	PSGTKGAELD	KHIDATIGSG	N. I PEAVEL	P. PGEDINEW	LAVNTVDEEN	OVNILLEGTET	78
120	KA1	STECLOS, KN	OKTERRKKNA	PSCNKCVOLK	KHIDATLOSO	N. IPDAVRI	P. PCEDINEW	LAVNTYDEEN	OVNELVCTEM	78
120			OLTERRYKES	PECENCIPIN	KHIDATLOSG	N. L DEAVEL	P. FGEDENEW		OVNELVCTIN	77
121	· · · · · · · · · · · · · · · · · · ·	MECL CATEN	AKTERORKNIT	PSGSKGLPLK	PHIDATLOSG	N. LHEAVEL	P. DCEDLNEW		AVELLYATE	76
120		- MEGESATAN	AKTERPHANI	PVGSKGLULK	ATLOSG	- N- IMEAVEL	P-PGEDENEW	LAVINTVDETIN	AVSILIAILE	70
145		- MEGES- NKI	ANTERVENT-	CAGINATULA	GTAEATEGSG	- 5- EMEAVEL	P- KGEDENEW		OI CHEVATE	14
145								MNIVDEEN	QISLLYAILE	18
140				TTUTTUOTTU						-
14/	N	VIDENTIPSH	OKPELOPOAG	TIVITHOUTK	GIVEMILGSE	- GVENGAVKE	P- HGEDENEW	LAVHCVDFYN	QINMLYGSIT	19
16					- MAVSTLGSG	- N- LHEALKL	P- PGEDKNEW	LAVNIIDLVN	QVHMVFGVLC	46
86				MSEPSSSWCN	PMAVSTLGSG	- N- LRDALKL	P- PGEDKNEW	LAVNIIDLVN	QVRMVFGVLC	57
131	···· S	SGSESKKSST	SQLVATGSPS	VEPTALYLQQ	PEVRTHLVKG	NESTIVSL	P- REVOLDEW	VALNVYELFT	YLNHEYDVEA	78
148	· · · · · · · · · Q	SQUETSTIPO	SQQQEASERS	ESQQIMELSE	PEVRTALVKG	SEKTIVOL	P- KYVDLGEW	IALNVEEFET	NENGFYGVVA	78
7		MDWLMG-KSK	AKPNGKK P	AAEEKKVYLE	PEHTKSRITD	- FEFKELVVL	P- REIDLNEW	LASNTTTEEH	HINLQYSTIS	75
95	LQA	VSKVLR-KSK	AKPNGKK P	AAEEKKVYLE	PEHTKSRITD	- FEFKELVVL	P- REIDLNEW	LASNTTTFFH	HINLQYSTIS	78
179		MDWLMG-KSK	AKPNGKK P	AAEERKAYLE	PEHTKARITD	- FOFKELVVL	P- REIDLNEW	LASNTTTFFH	HINLOYSTIS	75
200	LQA	VSKVLR- KSK	AKPNGKK P	AAEERKAYLE	PEHTKARITO	- EOEKELVVI	D DELDINEW			
35	a bit motore both a				THE REPORT OF THE PARTY OF THE		P-REIDLINEW	LASNIIIEEH	HINLOYSTIS	78
	AV SWPKLA	FYNAKV- KSK	AKPNGKKP	ATEEKKMYLE	PEYTKSRITD	- VGFKELVVL	P- REIDLNEW	LASNTTTEEH	HVNLQYSTIS	78 83
140	AV SWPKLA	FYNAKV- KSK Vgkvlr- ksk	AKPNGKKP GKPNGKKP	ATEEKKMYLE Apeekklyle	PEYTKSRITD	- VGFKELVVL - FEFKELVML	P-REIDLNEW	LASNTTTEEH	HINLOYSTIS HUNLOYSTIS HINLOYSTIS	78 83 78
140 81	AV SWPKLA	FYNAKV- KSK VGKVLR- KSK SYTVOK- KSK	AKPNGKKP GKPNGKKP GKPNGKKP	ATEEKKMYLE Apeekklyle Aseekklyle	PEYTKSRITD PEYTKSRITD PEYTRVRVTD	- VGFKELVVL - FEFKELVML - VEFKQLVTL	P-REIDLNEW P-REIDLNEW P-QEIDLNEW	LASNTTTFFH LASNTTTFFH LASNVTTFFN	HINLOYSTIS HINLOYSTIS HINLOYSTIS	78 83 78 79
140 81 128	AV SWPK LA	FYNAKV- KSK VGKVLR- KSK SYTVQK- KSK SYTVQK- KSK	AKPNGKKP GKPNGKKP GKPNGKKP GKPNGKKP	ATEEKKMYLE Apeekklyle Aseekklyle Aseekklyle	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD	- VGFKELVVL - FEFKELVML - VEFKQLVTL - VEFKQLVTL	P- REIDLNEW P- REIDLNEW P- QEIDLNEW P- QEIDLNEW	LASNTTTEFH LASNTTTEFH LASNTTTEFN LASNTTEFN	HINLOYSTIS HUNLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS	78 83 78 79 79
140 81 128 129	- AVSWPKLA	FYNAKV- KSK VGKVLR- KSK SYTVQK- KSK SYTVQK- KSK MEWLMG- KSK	AKPNGKK - P GKPNGKK - P GKPNGKK - P GKPNGKK - P GKPNGKK - P	ATEEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE ASEEKKLYLE	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD PEYTRVRVTD	- VGFKELVVL - FEFKELVML - VEFKOLVTL - VEFKOLVTL - VEFKOLVTL	P-REIDLNEW P-REIDLNEW P-QEIDLNEW P-QEIDLNEW P-QEIDLNEW	LASNTTTFFH LASNTTTFFH LASNTTTFFN LASNITTFFN LASNITTFFN	HINLOYSTIS HINLQYSTIS HINLQYSTIS HINLQYSTIS HINLQYSTIS	78 83 78 79 79 79 75
140 81 128 129 50	KAMGORN	EYNAKV- KSK VGKVLR- KSK SYTVQK- KSK SYTVQK- KSK MEWLMG- KSK TGMMEKRKSK	AKPNGKK P GKPNGKK P GKPNGKK P GKPNGKK P GKPNGKK P	ATEEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKQYLE	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD	- VGFKELVVL - FEFKELVML - VEFKQLVTL - VEFKQLVTL - VEIKQLVTL - FDLKELVVL	P-REIDLNEW P-REIDLNEW P-QEIDLNEW P-QEIDLNEW P-QEIDLNEW P-QEIDLNEW P-REIDLNEW	LASNTTTFFH LASNTTTFFH LASNVTTFFN LASNITTFFN LASSVTTFFN LASNTTTFFN	HINLOYSTIS HUNLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS	78 83 78 79 79 75 83
140 81 128 129 50 111		EYNAKV-KSK VGKVLR-KSK SYTVQK-KSK MEWLMG-KSK TGMMEKRKSK VGKVLR-KSK	AKPNGKKP GKPNGKKP GKPNGKKP GKPNGKKP TKPNGKKP TKPNGKKP	AT EEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKOYLE PAEEKKOYLE	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD LEYTKIRVVD	- VGFKELVVL - FEFKELVML - VEFKQLVTL - VEFKQLVTL - VEFKQLVTL - FOLKELVVL - FOLKELVVL	P - REIDLNEW P - REIDLNEW P - QEIDLNEW P - QEIDLNEW P - QEIDLNEW P - REIDLNEW P - REIDLNEW	LASNTTTEFH LASNTTTEFH LASNTTEFN LASNTTEFN LASSTTEFN LASNTTEFN LASNTTEFN	HINLOYSTIS HVNLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS LINLOYSTIS	78 83 78 79 79 79 75 83 80
140 81 128 129 50 111 43		FYNAKV- KSK VGKVLR- KSK SYTVQK- KSK MEWLMG- KSK TGMMEKRKSK VGKVLR- KSK	AKPNGKKP GKPNGKKP GKPNGKKP GKPNGKKP TKPNGKKP TKPNGKKP TKPNGKKA	ATEEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKQYLE PAEEKKQYLE PPEEKKHYLE	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD PEYTKVRVAD	- VGFKELVVL - FEFKELVML - VEFKQLVTL - VEFKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL	P- RE I D LN EW P- RE I D LN EW P- QE I D LN EW P- QE I D LN EW P- QE I D LN EW P- RE I D LN EW P- RE I D LN EW P- RE I D LN EW	LASNTTTFFH LASNTTTFFH LASNTTFFH LASNTTFFN LASNTTFFN LASNTTFFN LASNTTFFN LASNTTFFN	HINLOYSTIS HUNLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS	78 83 78 79 79 75 83 80 80
140 81 128 129 50 111 43 44	A W SWPK LA 	EYNAKV- KSK VGKVLR- KSK SYTVOK- KSK SYTVOK- KSK MEWLMG- KSK TGMMEKRKSK VGKVLR- KSK VGKVLR- KSK HHKALR- KGK	AKPNGKKP GKPNGKKP GKPNGKKP GKPNGKKP TKPNGKKP TKPNGKKA GKPNGKKA	ATEEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKOYLE PPEEKKOYLE PPEEKKHYLE PTEEKKHYLD	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD LEYTKIRVVD PEYTKVRVAD AEYTKVRVVD	- VGFKELVVL - EEFKELVML - VEFKQLVTL - VEFKQLVTL - VEFKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FELKELVVL	P-REIDLNEW P-QEIDLNEW P-QEIDLNEW P-QEIDLNEW P-QEIDLNEW P-REIDLNEW P-REIDLNEW P-REIDLNEW P-REIDLNEW	LASNTTTEFH LASNTTTEFH LASNTTTEFH LASNTTEFN LASNTTEFN LASNTTTEFN LASNTTTEFN LASNTTTEFN	HINLOYSTIS HUNLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS	78 83 78 79 79 75 83 80 80 78
140 81 128 129 50 111 43 44 172	- AV SWPK LA - LOA - RRSG - RRSG - RRSG - KAMGORN - MYLQA - YLQA - YLQA	EYNAKV- KSK VGKVLR- KSK SYTVQK- KSK MEWLMG- KSK TGMME KRKSK VGKVLR- KSK HHKALR- KGK	A K PNG KK P G K PNG KK P G K PNG KK P G K PNG KK P T K PNG KK P T K PNG KK P T K PNG KK P G K PNG KK P	ATEEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKQYLE PAEEKKQYLE PTEEKKHYLD IVEEKKPYLD	PEYTKSRITD PEYTKVRVTD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD LEYTKIRVVD AEYTKVRVAD AEYTKVRVAD PELFKQLPGH	- VGFKELVVL - FEFKELVML - VEFKQLVTL - VEFKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FELKELVVL - VDIRKVVTK	P- RE I D L N EW P- RE I D L N EW P- QE I D L N EW P- QE I D L N EW P- RE I D L N EW	LASNTTTEFH LASNTTTEFH LASNTTTEFN LASNTTTEFN LASNTTTEFN LASNTTTEFN LASNTTTEFN LASNTTTEFN LASNTTTEFN LASNTTTEFN	HINLOYSTIS HVNLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS NINOLYSTIS	78 83 78 79 79 75 83 80 80 78 67
140 81 128 129 50 111 43 44 172 12	- KAMGORN - MVLQA - WSG	EYNAKV- KSK VGKVLR- KSK SYTVOK- KSK SYTVOK- KSK TGMMEKEKSK VGKVLR- KSK VGKVLR- KSK HHKALR- KGK	A K PNGKK P GK PNGKK P GK PNGKK P GK PNGKK P TK PNGKK P TK PNGKK P TK PNGKK A GK PNGKK A GK PNGKK A GK PNGKK P FK FNG GG Q	ATEEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKQYLE PPEEKKYLE PTEEKKHYLE PTEEKKHYLE UYEEKKPYLO NSTDTKLYLE	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD LEYTKIRVVD PEYTKVRVAD AEYTKVRVAD AEYTKVRVAD PELFKQLPGH ESVLERKLPE	- VGFKELVVL - FEFKELVVL - VEFKQLVTL - VEFKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKDLVAL - FELKELVVL - ADLKALVDL	P - RE I D L N EW P - RE I D L N EW P - QE I D L N EW P - QE I D L N EW P - QE I D L N EW P - RE I D L N EW P - RN D ER EW P - AG D YN EW	LASNITTEEH LASNITTEEH LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASHITAELEH LASHITAELEH	HINLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS NINQUYGTIS	78 83 78 79 79 75 83 80 80 80 78 67 71
140 81 128 129 50 111 43 44 172 12 117	- KAMGOEN - WYEQA	EYNAKV-KSK VGKVLR-KSK SYTVOK-KSK SYTVOK-KSK GKVLR-KSK VGKVLR-KSK VGKVLR-KSK HKALR-KGK TFLCVAGKAR	AK PNGKK - P   GK PNGKK - P   GK PNGKK - P   GK PNGKK - P   GK PNGKK - P   TK PNGKK - P   TK PNGKK - P   GK PNGK P   GK PNGK P   GK PNGK P   GK PNGK P   F   F   F   F   F	AT EEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKQYLE PPEEKKHYLE PTEEKKHYLD IVEEKKPYLD NSTDTKLYLE	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD PEYTKVRVAD AEYTKVRVAD AEYTKVRVAD PELFKQLPGH ESVLERKLPE	- VGFKELVVL - FEFKELVML - VEFKQLVTL - VEFKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FELKELVVL - VDIRKVVTK - ADLKALVDL	P- RE I D L N EW P- RE I D L N EW P- QE I D L N EW P- QE I D L N EW P- RE I D L	LASNITTEEH LASNITTEEH LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASNITTEEN LASHILALEE LASHILALEE	HINLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS NINOLYGTIS HVNLVYGTIS	78 83 78 79 79 75 83 80 80 78 67 71 78
140 81 128 129 50 111 43 44 172 12 117 20	KAMGQEN MVLQA MVLQA VLQA VLQA VLQA VLQA VLQA VLQA VLQA	FYNAKV- KSK VGKVLR- KSK SYTVOK- KSK SYTVOK- KSK GKVLR- KSK VGKVLR- KSK VGKVLR- KSK HKALR- KGK TFLCVAGKAR TFLCVAGKAR	AK PNGKK - P   GK PNGKK - P   GK PNGKK - P   GK PNGKK - P   GK PNGKK - P   TK PNGKK - P   TK PNGKK - P   TK PNGKK - P   GK PNGKK - P   TK PNGKK - P   GK PNGKK - P   TK PNGKK - P   TK PNGKK - P   GK PNGKK - P   GK PNGKK - P   GK PNGK - P	ATEEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKQYLE PPEEKKQYLE PTEEKKHYLD NSTDTKLYLE NSTDTKLYLE NGDTKLYLE	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD LEYTKIRVVD LEYTKVRVD AEYTKVRVD AEYTKVRVD PELFKQLPGH ESVLERKLPE EGLLERKLPD	- VGFKELVVL - FEFKELVVL - VEFKQLVTL - VEFKQLVTL - FLKELVVL - FLKELVVL - FLKELVVL - FLKELVVL - ALKALVDL - ALKALVDL - ALKALVDL	P - RE I D L N EW P - RE I D L N EW P - QE I D L N EW P - QE I D L N EW P - QE I D L N EW P - RE I D L N EW P - AGLD YN EW P - AGLD YN EW	LASNTITETH LASNTITETH LASNTITETN LASNTITETN LASNTITETN LASNTITETN LASNTITETN LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASHTLALE LASHTLALE	H I NLQYSTIS H I NLQYSTIS H I NLQYSTIS H I NLQYSTIS H I NLQYSTIS L I NLQYSTIS L I NLQYSTIS L I NLQYSTIS L I NLQYSTIS H V NLYGTIS H V NLYGTIS H V NLYGTIS	78 83 79 79 75 83 80 80 78 67 71 78 78 78
140 81 128 129 50 111 43 44 172 12 117 20 115	- AVSWPKLA RRSG - RRSG - MYEQA - WYEQA - WSG - VEQA - SLN - SLN	FYNAKV- KSK VGKVLR- KSK SYTVOK- KSK SYTVOK- KSK TGMMEKEKSK VGKVLR- KSK VGKVLR- KSK VGKVLR- KSK VGKVLR- KSK HKALR- KGK 	A K PNGKK - P GK PNGKK - P GK PNGKK - P GK PNGKK - P T K PNGKK - P T K PNGKK - P T K PNGKK - A GK PNGKK - A GK PNGKK - A GK PNGKK - A GK FNGGD - Q RK E FD GD - S	AT EEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKQYLE PAEEKKQYLE PTEEKKHYLD IVEEKKYLD NSTDTKLYLE NGCDTKLYLE	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD PEYTRVRVTD DEYTKIRVVD LEYTKIRVVD PEYTKVRVAO AEYTKVRVAO AEYTKVRVAO PELFKOLPGH ESVLERKLPE EGLERKLPO	- YGFKELVYL - FEFKELVYL - VEFKQLVTL - VEFKQLVTL - FDLKELVYL - FDLKELVYL - FDLKELVYL - FDLKELVYL - FLKELVYL - ADLKALVDL - ADLKALVDL - ADLKLVDL	P - RE I D L N EW P - RE I D L N EW P - QE I D L N EW P - QE I D L N EW P - QE I D L N EW P - RE I D L N EW P - RG L D YN EW P - AG L D YN EW P - AG L D YN EW	LASNITTEFH LASNITTEFH LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASHITALAFF LASHTLALFE LASHTLALFE	H I N LQ Y ST I S H I N LQ Y ST I S H I N LQ Y ST I S H I N LQ Y ST I S L I N LQ Y ST I S N I N L Y ST I S N I N L Y G T I S H V N L Y G T I S H V N L Y G T I S	78 83 79 79 75 83 80 80 80 78 67 71 78 78 78 78
140 81 128 129 50 111 43 44 172 12 117 20 115 151	LQA RRSG RRSG KAMGORN VLQA VLQA VD SLN SLN	EYNAKV-KSK VGKVLR-KSK SYTVOK-KSK SYTVOK-KSK GKVLR-KSK VGKVLR-KSK VGKVLR-KSK HKALR-KGK TFLCVAGKAR IFFCPT-KAR CSKCGSRKAR	AK PNGKK - P   GK PNGKK - P   GK PNGKK - P   GK PNGKK - P   GK PNGKK - P   TK PNGKK - P   TK PNGKK - P   GK PNGK - P	AT EEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PEEKKYLE PEEKKYLE PEEKKYLE LYEEKKYLD LYEEKKYLD NSTDTKLYLE NGGDTKLYLE NGGDTKLYLE	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD LEYTKIRVVD PELFKQLPGH ESVLERKLPE ESVLERKLPE EGULERKLPE EGULERKLPE	- VGFKELVVL - FEFKELVML - VEFKQLVTL - VEFKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKELVVL - ADLKALVDL - ADLKALVDL - ADLKLVDL - ADLKLVDL	P- RE I D L N EW P- RE I D L N EW P- QE I D L N EW P- QE I D L N EW P- QE I D L N EW P- RE I D L N EW P- AG L D Y N EW		HINLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS HINLOYGTIS HVNLVYGTIS HVNLVYGTIS	78 83 79 79 75 83 80 80 78 67 71 78 78 78 78 78
140 81 128 129 50 111 43 44 172 12 117 20 115 151 134	- KAMGQEN - KAMGQEN - WYLQA - YEQA -	EYNAKV-KSK VGKVLR-KSK SYTVOK-KSK SYTVOK-KSK GKVLR-KSK VGKVLR-KSK VGKVLR-KSK HKALR-KGK HKALR-KGK FECVAGKAR FECVT-KAR VFECPT-KAR VFECPT-KAR	AK PNGKK - P   GK PNGKGK - P   GK PNGKG - T   GK PNGG - T   FK EKD GD - S   FK EKD GG - T   FK EKG GN - T   FK EKG GN - T	ATEEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKQYLE PPEEKKQYLE PTEEKKHYLD NSTDTKLYLE NGGDTKLYLE NGGDTKLYLE SSDSKLYL	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD LEYTKIRVVD AEYTKVRVD PELFKQLPGH ESVLERKLPE ESVLERKLPE EGLLERKLPD EGALERCLPE EAALEROLPE	- YGFKELVYL - FEFKELVYL - YEFKQLVTL - YEFKQLVTL - FDLKELVYL - FDLKELVYL - FDLKELVYL - FLKELVYL - FLKELVYL - ADLKALVYL - ADLKALVYL - ADLKLVYL - ADLRLVYL - ADLRVYL - ADLRVYL	P - RE I D L N EW P - RE I D L N EW P - QE I D L N EW P - QE I D L N EW P - QE I D L N EW P - RE I D L N EW P - RG L Y N EW P - AG L Y N EW	LASNTITETH LASNTITETH LASNTITETN LASNTITETN LASNTITETN LASNTITETN LASNTITETN LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASHTLALE LASHTLALE LASHTLALE LASHTLALE LASHTLALE	H I NLQYST IS H I NLQYST IS H I NLQYST IS H I NLQYST IS H I NLQYST IS L I NLQYST IS L I NLQYST IS L I NLQYST IS L I NLQYST IS H V NLYGT IS	78 83 78 79 79 75 83 80 80 80 78 67 71 78 78 78 78 78 78 78
140 81 128 129 50 111 43 44 172 12 117 20 115 151 134 105	- AVSWPKLA RRSG - RRSG - KAMGORN - MVLQA - VLQA - VSG - SLN - SLN - ELA	FYNAKV- KSK VGKVLR- KSK SYTVOK- KSK SYTVOK- KSK TGMMEKEKSK VGKVLR- KSK VGKVLR- KSK VGKVLR- KSK VGKVLR- KSK VGKVLR- KSK TFLCVAGKAR TFLCVAGKAR VFFCPT- KAR VFFCPT- KAR CSKCGSEKAR ILLCFR- KAR	A K PNGKK - P GK PNGKK - P GK PNGKK - P GK PNGKK - P T K PNGKK - P T K PNGKK - A GK PNGK - A GK PNGK - A GK - A	AT EEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKQYLE PAEEKKQYLE PTEEKKHYLD NSTDTKLYLE NSTDTKLYLE NGGDTKLYLE TE-DPKLYLE SSSDSKLYLE ERPYLQ	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD PEYTRVRVTD PEYTKVRVTD DEYTKIRVVD LEYTKIRVVD PEYTKVRVAD AEYTKVRVAD AEYTKVRVAD AEYTKVRVE BESVLERKLPE EGULERKLPD EGALERKLPD EGALERKLPD EGALERKLPD GOLVSORITH	- VGFKELVVL - FEFKELVVL - VEFKQLVTL - VEFKQLVTL - VEIKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKALVVL - ADLKALVDL - ADLKALVDL - ADLKALVDL - ADLKLVDL - ADLRLVDL - TDVGALAA	P- RE ID L N EW P- RE ID L N EW P- QE ID L N EW P- QE ID L N EW P- QE ID L N EW P- RE ID L N EW P- RG LD YN EW P- AG LD YN EW	LASNITTEFH LASNITTEFH LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASHITALFF LASHITALFF LASHITALFF LASHITALFF LASHITALFF LASHITALFF	H I N LQ Y ST I S H I N LQ Y ST I S H I N LQ Y ST I S H I N LQ Y ST I S L N LQ Y ST I S N N N L Y GT I S H V N LY GT I S	78 83 78 79 79 75 83 80 80 78 67 71 78 78 78 78 78 78 55
140 81 128 129 50 111 43 44 172 12 117 20 115 151 134 105 112	- KAMGGEN - KAMGGEN - WYLQA - YLQA - YLQA - SLN - SLN - ELA	EYNAKV-KSK VGKVLR-KSK SYTVOK-KSK SYTVOK-KSK GKVLR-KSK VGKVLR-KSK VGKVLR-KSK HKALE-KGK TFLCVAGKAR IFFCPT-KAR CSKCGSKAR ILLCER-KAR	AK PNGKK P GK PNGKK P GK PNGKK P GK PNGKK P TK PNGKK P T KPNGKK P T KPNGKK P GK PNGKK P GK NDKK P GK NDKK P GK NDKK P GK ST GK NDKK P GK ST GK ST ST ST ST ST ST ST ST ST ST ST ST ST S	AT EEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PEEKKYLE PEEKKYLE PEEKKYLE IVEEKKYLE NGGDTKLYLE NGGDTKLYLE SSSDSKLYLE SSSDSKLYLE EERPYLO	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD LEYTKIRVVD AEYTKVRVD PELFKQLPGH ESVLERKLPE EGLERKLPE EGLERKLPE EGLERKLPD EGVLERKLPD CGVVSQRITH	- YGFKELVYL - FEFKELVYL - YEFKQLVTL - YEFKQLVTL - FDLKELVYL - FDLKELVYL - FDLKELVYL - FDLKELVYL - YDIRKVYTK - ADLKALVYL - ADLKALVYL - ADLKALVYL - ADLKALVYL - ADLRLVYL - ADLRLVYL - TDYGALAAL	P- RE I D L N EW P- RE I D L N EW P- QE I D L N EW P- QE I D L N EW P- QE I D L N EW P- RE I D L N EW P- AG L Y N EW P- RG L Y N EW P- PG V R AE W		HINLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS HVNLOYSTIS HVNLOYGTIS HVNLOYGTIS HVNLOYGTIS HVNLOYGTIS NINLFFSALS	78 83 78 79 79 75 83 80 80 78 67 71 78 78 78 78 78 78 78 78 78 55 55
140 81 128 129 50 111 43 44 172 12 117 20 115 151 134 105 112 51	- KAMGQEN - KAMGQEN - WYLQA - YLQA - YLAA - YLQA - YLAA -	EYNAKV-KSK VGKVLR-KSK SYTVOK-KSK SYTVOK-KSK GKVLR-KSK VGKVLR-KSK VGKVLR-KSK HKALR-KGK HKALR-KGK FECVAGKAR FECVT-KAR VFFCPT-KAR VFFCPT-KAR VFFCPT-KAR	AK PNGKK - P GK PNGKK - P GK PNGKK - P GK PNGKK - P TK PNGKK - P TK PNGKK - P TK PNGKK - P TK PNGKK - P GK KNDKK - P GK KNDK - P GK KNDKK - P GK KNDK - P GK K	AT EEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PFEKKQYLE PFEKKQYLE PTEEKKHYLD UYEEKKHYLD NSTDTKLYLE NGGTKLYLE NGGTKLYLE SSDSKLYLE SSDSKLYLE ERPYLQ NNGERPYLQ	PEYTKSRITD PEYTKSRITD PEYTRVRVTD PEYTRVRVTD PEYTRVRVTD LEYTKIRVVD LEYTKIRVVD AEYTKVRVD PELFKQLPGH ESVLERKLPE EGLLERKLPD EGLLERKLPD EGLERKLPD CALERCLPE AALEROLPE ATVLERKLPD COYVSORITH COYVSORITH COYLCKOIPH	- VGFKELVVL - FEFKELVVL - VEFKQLVTL - VEFKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKLVVL - FDLKLVVL - ADLKALVDL - ADLKALVDL - ADLKLVDL - ADLKLVDL - DLKMLVDL - TDVGALAAL - TDVTPLAAL	P - RE I D L N EW P - RE I D L N EW P - QE I D L N EW P - QE I D L N EW P - QE I D L N EW P - RE I D L N EW P - RG D Y N EW P - AG L D Y N EW P - PG V R AE W		H I NLQ Y ST I S H I NLQ Y ST I S L I NLQ Y ST I S H V NL Y G T I S N I NL F SALS N I NL F SALS	78 83 78 79 79 75 83 80 80 80 78 67 71 78 78 78 78 78 78 78 55 55 78
140 81 128 129 50 111 43 44 172 12 117 20 115 151 134 105 112 51 163	LQA RRSG RRSG KAMGORN MVLQA VLQA VLQA SLN SLN SLN SLN FLA SLN TK	EYNAKV-KSK VGKVLR-KSK SYTVOK-KSK SYTVOK-KSK GKVLR-KSK VGKVLR-KSK VGKVLR-KSK HKALR-KGK TFLCVAGKAR TFLCVAGKAR IFFCPT-KAR CSKCGSRKAR ILLCER-KAR SGSLQPSGIR TESKAACDIS	AK PNGKK - P GK PNGKK - P GK PNGKK - P GK PNGKK - P GK PNGKK - P TK PNGKK - P TK PNGKK - P TK PNGKK - P GK K PNGKK - P GK K PNGKK - P GK K PNGK - A GK PNGK -	AT EEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE PPEEKKQYLE PAEEKKQYLE PTEEKKHYLD NSTDTKLYLE NGGDTKLYLE NGGDTKLYLE SSSDSKLYLE SSSDSKLYLE NGGDTKLYLE TE-DPKLYLE SSSDSKLYLE NGGERPYLQ	PEYTKSRITD PEYTKSRITD PEYTKVRVTD PEYTRVRVTD PEYTRVRVTD PEYTKVRVTD PEYTKVRVD LEYTKIRVVD PEYTKVRVAD AEYTKVRVAD AEYTKVRVAD PELFKQLPGH ESVLERKLPE EGULERKLPD EAALEROLPE EGULERKLPD EAALEROLPE GAUERKLPD EAALEROLPE CAYSORITH QOYVSORITH QOYVSORITH PHVRHRTD	- VGFKELVVL - FEFKELVVL - VEFKQLVTL - VEFKQLVTL - VEIKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKELVVL - ADLKALVDL - ADLKALVDL - ADLKALVDL - ADLKALVDL - ADLKALVDL - DLRLLVDL - TDVGALAAL - TDVTPLAAL - TDVTPLAAL - ADMALSAL	P- RE ID L N EW P- RE ID L N EW P- QE ID L N EW P- QE ID L N EW P- QE ID L N EW P- RE ID L N EW P- RG LD YN EW P- AG LD YN EW P- PG VI A EW P- PG VI A EW P- PG VI A EW	LASNITTEFH LASNITTEFH LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITTEFN LASNITALFE LASHILALFE LASHILALFE LASHILALFE LASHILALFE LASHILALFE LASHILALFE LASHILALFE LASHITALFE LASHITALFE LASHITALFE LASHITALFE LASHITALFE LASHITALFE LASHITALFE LASHITALFE	HINLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS HINLOYSTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS HVNLVYGTIS	78 83 78 79 79 75 83 80 80 78 771 78 78 78 78 78 78 78 78 78 78 78 78 78
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140 81 128 129 50 111 43 44 47 12 12 117 20 115 151 151 151 151 163 82 98 63 9 95 63 9 95 63 97 96 84 63 197 168 168 178 198 109 100 117 105 115 115 115 115 115 115 115	A WSWPK LQA RRSG RRSG KAMGORN MV LQA VLQA VLQA VLQA SLN SLN SLN SLN SLN SLN SLN SLN	EYNAKV- KSK VGKVLR- KSK SYTVQK- KSK SYTVQK- KSK GKVLR- KSK VGKVLR- KSK VGKVLR- KSK HKALE- KGK TFLCVAGKAR IFCPT- KAR CSKCGSKAR ILLCER- KAR ILLCER- KAR ILLCER- KAR GAVARAES GAVARAES GAAVARAES GAAVARAES FRNRPGTKAQ RNNPGTKAQ RNNPGTKAQ RNNPGTKAQ	AK PNGKK - P GK PNGKK - P GK PNGKK - P GK PNGKK - P T K PNGKK - P T K PNGKK - P T K PNGKK - P T K PNGKK - P GK K PNGKK - P GK K PNGK - P GK K F V G S F GL V S C G S F S C S C S C S C S C S C S C S C S C S C	AT EE KKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE ASEEKKLYLE PAEEKKQYLE PPEEKKHYLD IVEEKKPYLD NSTDTKLYLE NGGDTKLYLE NGGDTKLYLE SSSDSKLYLE SSSDSKLYLE SSSDSKLYLE TE-DPKLYLD NNGE RPYLQ NNGE RPYLQ NNGE RPYLQ NNGE RPYLQ NNGE RPYLQ DYEEPKLYLD PTEEPKLYLD PTEEPKLYLD PTEEPKLYLD PTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DTEEPKLYLD DEMDSTLAVQ DEMDSTLAVQ DEMDSTLAVQ DEMDSTLAVQ DEMDSTLAVQ	PEYTKSRITD PEYTKSRITD PEYTRYKYTD PEYTRYKYTD PEYTRYKYTD PEYTRYKYTD PEYTRYKYTD PEYTRYRYD EYTKYRYD PELFKQLPGH ESVLERKLPE EGLLERKLPD EGVLERKLPE EGLLERKLPD GALERKLPE EGLERKLPD GALER	- VGFKELVVL - FEFKELVVL - FFKELVVL - VFFKQLVTL - VFFKQLVTL - VFFKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKELVVL - ADLKALVVL - ADLKALVVL - ADLKALVDL - CSNIKLLEP CSNIKLEP CSNIKLEP CSNIKLEP CSNIKLEP CSNIKLEP CSNIKLEP	P-REIDLNEW P-REIDLNEW P-QEIDLNEW P-QEIDLNEW P-QEIDLNEW P-REIDLNEW	LASNTITEH LASNTITEH LASNTITEH LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASNTITEN LASNTIALE LASHTLEN LASHTLEN LASH		78 83 879 795 880 887 87 888 887 878 888 888 888 888
140 81 128 129 50 111 43 44 44 172 12 117 20 115 151 163 82 89 99 15 84 6 191 33 67 94 6 178 199 80 101 113 114 134 105 115 116 134 105 115 116 134 105 115 116 134 105 115 116 134 105 115 116 134 105 116 134 105 117 117 105 116 117 105 116 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 115 117 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 117 105 105 105 105 105 105 105 105	- AV SWPK L A - CA - R SG - R SG - WY LQA - WY LQA - WY LQA - WSG - SIN - SIN	EYNAKV- KSK VGKVLR- KSK SYTVOK- KSK SYTVOK- KSK SYTVOK- KSK GKVLR- KSK VGKVLR- KSK VGKVLR- KSK VGKVLR- KSK VGKVLR- KSK IGKCSKA TELCAGKAR IFFCPT- KAR VFFCPT- KAR VFFCPT- KAR ILLCER- KAR ILLCER- KAR ILLCER- KAR GTAVANAAE GAAVASAAD HNNPGTKAQ RNNPGTKAQ RNNPGTKAQ RNNPGTKAQ RNNPGTKAQ RNNPGTKAQ RNNPGTKAQ	AK PNGKK - P GK PNGKK - P GK PNGKK - P GK PNGKK - P TK PNGKK - P TK PNGKK - P TK PNGKK - P GK CK C - P GK CK C - P GK CK C - P GK C - C GK	AT EEKKMYLE APEEKKLYLE ASEEKKLYLE ASEEKKLYLE ASEEKKLYLE PFEKKQYLE PFEKKQYLE PFEKKYLD IVEEKKPYLD NSTDTKLYLE NGDTKLYLE NGDTKLYLE NGDTKLYLE SSDSKLYLE - ERPYLQ NNGERPYLQ NNGERPYLQ NNGERPYLQ NNGERPYLQ PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD PTEPKLYLD DEMDSTLAVQ DEMDSTLAVQ DEMDSTLAVQ DEMDSTLAVQ DEMDSTLAVQ	PEYTKSRITD PEYTKSRITD PEYTKYRVTD PEYTRVRVTD PEYTRVRVTD PEYTRVRVTD EYTKYRVD PEYTKVRVD PETFKQLPGH ESVLERKLPE ESVLERKLPE EGLLERKLPD EGLLERKLPD COVERKLPD EGLERKLPD COVERKLPD EGLERKLPD COVERK	- VGFKELVVL - FEFKELVVL - VFFKQLVTL - VFFKQLVTL - VFFKQLVTL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKELVVL - FDLKLVVL - ADLKALVVL - ADLKLVVL - ADLKALVVL - ADLKLVL - AD	P - RE I D L N W P - RE I D L N W P - QE I D L N W P - QE I D L N W P - QE I D L N W P - RE I D L N W P - AG L Y N W P - GO L G W	LASNTTTEFH LASNTTTEFH LASNTTTEFH LASNTTEFN LASNTTEFN LASNTTEFN LASNTTEFN LASNTTEFN LASNTTEFN LASNTTEFN LASNTTEFN LASNTLALFE LASHTLAFF	HINLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS HINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS LINLOYSTIS HUNLYGTIS HVNLYGTIS HVNLYGTIS HVNLYGTIS HVNLYGTIS HVNLYGTIS HVNLYGTIS HVNLYGTIS HVNLYGTIS HVNLYGTIS HVNLYGTIS HVNLYGTIS HUNLSSALS HINLFSALS HINLFSALS LELNGLAVKL LELNGLAVKL LELNGLAVKL LELNGLAVKL LELNGLAVKL LELNGLAVKL	78 83 879 795 880 787 718 788 785 558 788 787 797 758 800 787 718 788 785 558 788 787 608 789 799 794 66 799 792 793 788 788 788 788 788 788 788 788 788 78

162	· · · · · · · · · I	RENEPGTKAK	DFYNWPDESF	EEMDSTLAVQ	QYIQONIRSD	CSNIDKILEP	P- EGODEGVW	KYEHL- ROFC	LELNGLAVKL	79
110	· · · · · · · · · I	RENEPGTKAK	DFYNWPDESF	EEMDSTLAVQ	QYIQQNIRSD	CSNIDKILEP	P- EGODEGVW	KYEHL- ROFC	LELNGLAVKL	79
49	· · · · · · · · · I	RENEPGTKAK	DFYNWQDESF	EEMDSTLAVQ	QYIQQNIRSD	CANIDKILEP	P- EGODEGVW	KYEHLSROFC	LELNGLAVKL	80
42	· · · · · · · · · I	BRNBPGTKAK	DFYNWSDESF	EEMDSTLAVQ	QYIQQNIRSD	CSNIEKIMEP	P- EGODEGVW	KYEHL- ROFC	LELNGLAVKL	79
34	MAEGTAVL	RENEPGTKA-			QYIQQNIRAD	CSNIDKILEP	P- EGODEGVW	KYEHL- ROFC	LELNGLAVKL	65
192	MVMAEGTAVL	RENEPGTKA-			QYIQQNIRAD	CSNIDKILEP	P- EGODEGVW	KYEHL- ROFC	LELNGLA	64
24								QEC	LELNGLAVKL	13
13	· · · · · · · · · L	RENEPGTKSK	DECRWPDEPL	EEMDSTLAVQ	QYIQQLIKED	PSNVELILTM	P- EAODEGVW	KYEHL- BOFC	MELNGLAVEL	79
118	· · · · · · · · · I	RENEPGTTSK	DECRWPDEPL	EEMDSTLAVQ	OFIQOLIKED	PSNVELILTM	P- EAQDEGVW	KYEHL- ROFC	MELNGLAVEL	79
135	· · · · · · · · · I	RENEPGTKAK	DECRWPDEPE	EEMDSTLAVQ	QYIQQLIENN	PANIDLILKM	P- ENQDEGVW	KYEHL- ROFC	MELNGLAVEL	79
152	· · · · · · · · · · · F	RENEPGTTAK	NECRWPDEPE	EEMDSTLAVQ	QYIQQMIRRD	PSNVDLILKM	P- EAQDEAVW	KYEHL- ROFC	MELNGLTVRL	79
21	· · · · · · · · · L	RENEPGTKAK	DESNWADEVE	EEMDSTLAVQ	QYIQQMIKKD	PSNVDQILTM	P- DGODEGVW	KYEHL- ROFC	MELNGLAVEL	79
116	· · · · · · · · · · I	RENEPGTKAK	DESNWPDEPE	EEMDSTLAVQ	QYIQQMIKKD	PSNVEQILTM	P- DGODEGVW	KYEHL- ROFC	MELNGLAVEL	79
171	· · · · · · · · · · V	RRNPPGTKAE	DFYKWSPQSF	DEMDSTLAVQ	QYIQOTIROD	FTDTETILTA	P- PGODEGVW	KYEQL- ROFC	LELNGLAIKL	79
62		RENEPGTKAC	DMYQWPDEPF	EEMDSTLAVQ	QYIQQQIRCN	CENVDAILES	P- EGODEGVW	KYEHL- ROFC	MELNGLAVKL	79
142	· · · · · · · · · · I	GAVRLTCGSP	DFYNWPDESF	EEMDSTLAVQ	QYIQQNIRAD	CSNIDKILEP	P- EGODEGVW	KYEHL- ROFC	LELNGLAVKL	79
97			DFFSWEEQAF	DEMDSTLAVQ	<b>QFIQQTIRKD</b>	ISNIDGILHP	P- EGODEGVW	KYEHL- ROFC	LELNGLAVKL	68
155	E	RENEPGTKAE	EWCNWPDEPF	EDMESTLAVQ	<b>QYIQOTIRRD</b>	ENNVDEILTA	P- EGODEVVW	KYEHL- ROFC	MELNGLAVEL	79
17	· · · · · · · · · · V	RENGPGTKEA	DWNCWPPLAF	EEMDSALNIQ	QYIQQTIKAN	PSDVEALLTP	P- LAYES	NFLCI- SPLL	PES YCVLK	74
85	· · · · · · · · · · V	RENGPGTKEA	DWNNWSPLAF	EEMDSALNIQ	QYIQQTIKAN	PADVATILTP	P- LDODEGVW	KYEHL- ROFC	IELNGLALLL	79
71	M	RRNRIGTKAE	FLWAWDVEDI	SOMNGPLAVO	EYIQELIRAD	SSNIKQIITP	P- PEVDIHVW	QYEHL- ROFI	LELNLLVTQL	79
Consensus		MLFQVGK	DKTFRPKKNF	EEGTHRYELH	KHAQATLGSG	-N-LRQAVML	P-EGEDLNEW	IAVHTVDFFN	QINLLYGTIS	
			Martin Contractor	-					-B Bed	
Conservation	Charles .			nn nann a					n Coll Co	
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	100		120		140		160			180	
			1	and the second second	1	And a state of the	1		-	1	
175	D- FCTEESCP	VMSA	GPKYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	QDQLDDETLF	PSKIG- VPFP	KNEMS-	AKT	150
2	E- ECTERTCP	VMSG	GPKYEY	RWQDDLK	YKKPTALPAP	QYMNLLMDWI	EVQINNEDIF	PTC-VGVPFP	KNELQ-	CKK	153
89	E- ECTERTCP	VMSG	GPKYEY	RWQDDLK	YKKPTALPAP	QYMNLLMDWI	EVQINNEDIF	PTC- VGVPFP	KNELQ-	СКК	153
28	E- ECTEQTCP	VMSG	GPKYEY	RWODDLK	YKKPTALPAP	OYMNELMDWI	EVQINNEDIF	PTC- VGVPFP	KNELQ-	СКК	153
165	E- ECTERTCP	VMSG	GPKYEY	RWQDDLK	YKKPTALPAP	OYMNELMDWI	EVQINNEDIF	PTC- VGVPFP	KNELQ-	СКК	153
174	E- ECTERTCP	VMSG	GPKYEY	RWQDDLK	YKKPTALPAP	QYMNELMDWI	EVQINNEEIF	PTC-VGVPFP	KNFLQ-	СКК	153
181	E- ECTERTCP	VMSG	GPKYEY	RWQDDLK	YKKPTALPAP	OYMNELMOWI	EVQINNEELE	PTC-VGVPFP	KNELQ-	СКК	153
195	E- ECTERTCP	VMSG	GPKYEY	RWODDLK	YKKPTALPAP	OYMNELMOWI	EVQINNEELE	PTC- VGVPFP	KNELQ-	СКК	153
22	E- ECTENTCP	VMSG	GPKYEY	HCQDDHK	YKKPTALPAL	QYMNELMDWI	EVQINNEDIE	PTC-VGVPFP	KNELQ-	CKK	153
187	E- ECTERTCP	VMSG	GPKYEY	RWQDDLK	YKKPTALPAP	OYMNELMOWI	EVGINNEDIF	PTS-VGVPFP	HNFEQ-	CKK	153
52	E- ECTENTOP	VMSG	GPKTET	HWODDEK	TKKPTALPAP	OYMNELMOWI	EVOINNEDIE	PIC-VGVPFP	KNELQ-	CKK	139
13/	D. COTENTOP	MSG.	CREVEN	RWODDNR	TKKPTALPAP	OWNNELMOWI	EVOINNEDIE	PTS. VOVPEP	KNELQ.	CKK	153
4	D. GCTEOSCP	VMSG	GPKYEY	RWODEOR	ERKPTALSAP	RYMPLIMDWI	EVOINNEDIE	PTN- VGTPEP	KTELO	AVRK	153
91	D. GCTEOSCP	VMSG	GPKYEY	RWODEOR	VRKPTAL SAP	RYMDLEMDWL	EVOINNEDLE	PTN- VGTPEP	KTELO.	VRK	153
8	D- GCTEOSCP	VMSG	GPKYEY	RWODEOR	ERKPTALSAP	RYMDLLMDWI	EVOINNEDIE	PTN-VER			141
185	D- GCTEOSCP	VMSG	GPKYEY	RWODEHK	FRKPTALSAP	RYMDLLMDWI	EAQINNEDLE	PTN-VGESV-			143
31	D- GCTEQSCP	IMSG	GPKYEY	RWODEHQ	FRKPTALSAP	RYMDLLMDWI	EVQINNEELF	PTH- VGTPEP	KNELQ-	VVKK	153
176	D- GCTEOSCP	VMSG	GPKYEY	RWODEHK	FRKPTALSAP	RYMDLLMDWI	EAQINNEDLF	PTN-VGTPFP	KNFLQ-1	<b>VRK</b>	153
167	D- GCTERSCP	IMSG	GPKYEY	RWODENK	FRRPTALSAP	RYMDLLMDWI	EVQINNEDVE	PTN-VGTPFP	KNFLQ-	VVKK	153
53	D- GCSEQSCP	VMSG	GPKYEY	RWODEHK	FRKPTALSAP	KYMDLLMDWI	EVQINNEDLF	PTS-VGTPFP	KNFLQ-	VVKK	153
78	D- SCTEQSCP	VMSG	GPKYEY	RWQDDNR	YRKPTAL SAP	KYMNLLMDWI	EVQINNEGIF	PTN-VGTPFP	KNFLQ-	VVKK	153
125	D- SCTEQSCP	VMSG	GPKYEY	RWQDDNR	YRKPTALSAP	KYMNELMDWI	EVQINNEGIF	PTN-VGTPFP	KNFLQ-	VKK	153
188	D-NCTEQSCP	MSG	GPKYEY	HWODEQK	YRKPTALSAP	KYMNLLMDWI	EVQINNEDIF	PTN-VGTPFP	KNFLQ-	VKK	153
102	D- SCTDQTCP	MSG	GPKYEY	HWODEHK	YKKPTALSAP	KYMSLLMDWI	EVQINNENTE	PTN-VGTPEP	KTEMQ-	AKK	153
108	B- SCIDOTCP	MSG	GPKYEY	RWODEHK	KAPTALSAP	KYMSLLMDWI	EVOLNNENTE	PTN-VGTPFP	KIFMQ-	AKK	153
158	D. SCIDOTCP	MASG	CORVEY	BWODEHK	KAPITLSAP	KYMSLLMDWI	EVOLNNENTE	PTN-VGTPPP	KNEMO-	ANN	153
159	D-SCIDUICP	VMSG	CONVEY	RWODERK	YKHPTILSAP	KYMSLLMDWI	EVOINNENTE	PTN-VGTPEP	KNEMQ-	ARK	153
170	D. ECTNESCP	MMSG	GPKYEY	RWODNDR	VKKDTNISAS	MYVAELMOWI	EHLINDEALE	PTK. VCTPEP	KSEKT	CKK	153
5	F. HOSESSOP	VMAG	GPRVEY	RWODERO	VRRPAKI SAP	RYMALLMOWL	EGLINDEDVE	PTR. VOVPEP	KNEOO	CTK	153
93	E-HCSETSCP	VMAG	···· GPRYEY	BWODEBO	VRRPAKI SAP	RYMALLMOWL	EGLINDEDVE	PTR-VGVPEP	KNEQQ-	CTK	153
177	E- BCSETSCP	VMAG	···· GPRYEY	BWODEBQ	YBBPAKLSAP	RYMALLMDWI	EGLINDEEVE	PTR-VGVPEP	KNEQQ-	CTK	153
198	E- BCSETSCP	VMAG	GPRYEY	RWODERQ	YRRPAKLSAP	RYMALLMOWI	EGLINDEEVE	PTR-VGVPEP	KNEQQ-	CTK	153
183	E- RCSETSCP	VMAG	GPRYEY	RWQDERQ	YRRPAKLSAP	RYMALLMDWI	EGLINDEEVF	PTR-VGVPFP	KNEQQ-	CTK	153
168	E- RCSETSCP	VMAG	GPRYEY	RWQDERQ	YRRPAKLSAP	RYMALLMDWI	ESLINDEDVE	PTR-VGVPFP	KNEQQ-	CTK	153
32	E- RCSETSCP	VMAG	GPRYEY	RWQDERQ	YRRPAKLSAP	RYMALLMDWI	EGLINDEDVF	PTR-VGVPFP	KNFQQ-	CTK	153
190	E- ECTEASCP	VMSG	GPRYEY	RWQDEQQ	YRRPAKLPAP	RYMSLLMDWI	EGLINNEDVF	PTQ- VGVPFP	KNFQQ-	CTK	153
79	E- FCTERSCP	IMCG	GLKYEY	RWQDDNK	YKRPTKVSAP	LYMNMLMEWI	ETLINNEDIF	PTR-MGVPFP	KNFQQ-	CNK	153
126	E- FCTERSCP	IMCG	GLKYEY	RWQDDNK	YKRPTKVSAP	LYMNMLMEWI	ETLINNEDIF	PTR-MGVPFP	KNFQQ-	CNK	153
138	E- YCTEKSCP	IMSG	GLKYEY	RWODDSK	YKKPTKLSAP	QYMCMLMDWI	EMLINNEDIF	PTR-IGVPFP	KQFQQ-	CTK	153
47	E- YCSERTCP	IMSG	···· GLRYEY	RWRDGDD	YKRPTKLPAL	KYMNLLMDWI	ESLINNEDIF	PTRTIGVPFP	KNEQQ-	CKK	154
48	E- YCSERTCP	IMSG	GLRYEY	HWRDGDD	YKRPTKLPAL	KYMNLLMDWI	ESLINNEDIF	PTHTIGVPFP	KNEQQ-	CKK	154
103	E. ECTERTOP	MSG	CL DVEY	RWODGDE	YKKPIKLSAL	KIMNLLMDWI	ESLINNEDIE	PTR-VGVPFP	KNEQQ.		153
160	E. VCTERTOR	IMSG.	GLEVEN	RWRDCDD	VKKDTKLDAL	KYMNILLMDWI	ESLINDEDIE	PTP. VOVPEP	KNEOO		152
41	E. ECTEKSCP	IMSG	GPRVEY	RWODGEO	YKRPTKI PAL	IMMNELMNWI	ESLINNEDIE	PTR-VGVPEP	KNEQQ-	CKK	153
55	E- HCSETSCP	VMAG	GPRYEY	RWODERO	YRRPAKLSAP	RYMALLMOWL	ESLINDEEVE	PTR- VXXXXX	XXXXX-	XXXX	153
19	E- YCNETTCP	TMSG	GAKYEY	LWADGET	YKKPIQLPAP	RYIELLMOWV	ENGINNETLE	PVS-TOVPEP	KTEPS-	CKK	153
114	E- YCNETTCP	TMSDPT	ISTKYEY	LWADGEL	FKKPTQLPAP	RYVELLMDWV	ENGINNEALF	PVS-TDVPFP	KSEPT-	CKK	156
10	E- FCNETTCP	TMSG	GSRYEY	LWADGDL	YKKPTALSAQ	KYIEHLMDWI	ETQINNEAVE	PVS-TDVPFP	KNEIA-	SRK	153
133	D- YCTEQSCP	TMSG	GPRFEY	LWADGDK	YKKPTPLPAK	EYISHLMDWI	EMQINNQALE	PCT- SDLPFP	KHEDK-H	ICSK	153
150	E- YCDSASCP	TMSG	GARFEY	LWADGEK	YKKPTALPAP	QYVSLLMDWI	EAQINNETIF	PVS-TDVPFP	KTEVL-I	CRK	153
14	D- VCTCESCP	TMCG	GSRYEY	LWQDGLE	YKKPTRLPAP	QYMQLLMDWI	EVRINDESIE	PSS-TNVSEP	KDFRQ-	CKK	153
83	D- VCTRESCP	TMCG	GSRYEY	LWODGIE	YKKPTRLPAP	QYMQL LMDWI	EVRINDEHIE	PSS-TNVSFP	KDFRQ-	CKK	153
154	D- RCTEQTCP	TMSG	GKKFEY	HWBDNVH	YKKPTPLPAP	KYIDELMDWV	DAQINDPSLF	PTD-MGVPEP	KCYIP-	VKK	153
156	D- RCTEOTCP	TMSG	GKKFEY	HWRDNVH	YKKPTPLPAP	KYIDELMDWV	DAQINDPSLF	PTD-MGI			98
58	E-NCTHOTOP	MUA	GOKYEY		KATKL SAP	DXXTLLMDWI	ESILINGENUE	PTD ACODIO	KOLCH	VKT	151
59	E. ECTASSOR	MMC A	GAEVEN		TKPIPUSAP	OVMEELMCWL	ESPUNNEOLE	PPS. DBVDED	TNEKS.	EV KO	120
92	E- ECTEASCP	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWY	ODOLDDETLE	PSKIG-VPEP	KNEMS.	AKT	150
197	E- ECTEASCP	VMSA	···· GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWY	QDQLDDETLE	PSKIG- VPEP	KNEMS-	AKT	150
184	E- ECTEASCP	VMSA	GPRYEY	HWADGTN	<b>IKKPIKCSAP</b>	KYIDYLMTWY	ODOLDDETLE	PSKIG			137
201	E- ECTEASCP	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWY	ODOLDDETLE	PSKIG- E		-	140
182	E- FCTEASCP	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	ODOLDDETLE	PSKIG- VPFP	KNEMS-	AKT	150
88	E- FCTEASCP	VMSA	GTRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	ODOLDDETLE	PSKIG- VPFP	KNEMS-	AKT	150
173	E- FCTEASCP	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	ODOLDDETLE	PSKIG- VPFP	KNEMS-	AKT	150
194	E- FCTEASCP	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	ODOLDDETLE	PSKIG- VPFP	KNEMS-	AKT	150
164	E- FCTEASCP	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	ODOLDDETLF	PSKIG- VPFP	KNEMS-	AKT	150
87	E- ECTEASCP	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	QDQLDDETLF	PSKIG-VPFP	KNEMS-	AKT	150
27	E- ECTEASCP	MSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	ODOLDDETLE	PSKIG-VPFP	KNEMS-	AKT	150
100	E- ECTEASCP	WMSA	GORNEY	HWADGTN	KKPIKCSAP	KYIDYLMTWV	ODOLDDETLE	PSKIG-VPFP	KNEMS-	AKT	150
100	E. ECTEASOP	VMSA	COPRIEY	HWADGTN	IKKPYKCCAP	KYIDYIMTWV	ODOLDDETLE	PSKIC WPEP	KNEMS-	ANT	147
100	E. ECTETSCP	VMSA	GPRVEY	HWADGTN	IKKPIKCSAP	KYLDYLMTWY	ODOLDDETLE	PSKIG-VPFP	KNEMS-	ANT	147
106	E- ECTEPSCP	VMSA-	GPRVEV	HWADGTN	IKKPIKCSAP	KYLDYLMTWV	ODOLDDETLE	PSKIG- VPEP	KNEMS.	AKT	152
36	- LOT BEOVE				A A A A A A A A A A A A A A A A A A A			···· S- VPEP	KNEMS-	AKT	74
75	E- ECTESTOS	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWY	ODOLDDETLE	PSKIG- VPEP	KNEMS-	AKT	150
157	E- ECTETSCS	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWY	ODOLDDETLE	PSKIG- VPFP	KNEMS-	AKT	150
45	E- FCTEISCS	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWY	ODOLDDETLE	PSKIG- VPFP	KNEMS-	AKT	150
124	E- ECTESTCS	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	QDQLDDETLE	PSKIG- VPFP	KNFMS-	AKT	136
141			YEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	QDQLDDETLE	PSKIG- VPFP	KNEMS-	AKT	58
122	E- FCTESTCS	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	ODOLDDETLE	PSKIG- VPFP	KNEMS-	AKT	149
39	E- ECTEVKCS	VMSA	GPRYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	QDQLDDETLF	PSKIG- VPFP	KNFMS-	AKT	150
3	D- ECTEESCP	MMSA	GPKYEY	HWADGTN	KKPIKCSAP	<b>KY DY MTWV</b>	ODOLDDETLE	PSK G- VPEP	KNEMS-	AKT	150

90	D- ECTEESCP	VMSA	GPKYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	ODOLDDETLE	PSKIG-VPFP	KNEMS- VAKT	150
166	D- ECTEESCP	VMSA	GPKYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	ODOLDDETLE	PSKIG- VPFP	KNEMS- VAKT	150
136	D- FCTEESCP	VMSA	GPKYEY	HWADGTN	<b>IKKPIKCSAP</b>	KYIDYLMTWV	ODOLDDETLE	PSKIG-VPFP	KNEMS- VAKT	150
29	D- ECTEESCP	VMSA	···· GPKYEY	HWADGTN	IKKPIKCSAP	KYIDYIMTWY	ODOLDDETLE	PSKIG- VPEP	KNEMS- VAKT	152
105	D. FOTEESCO	VMCA	CREVEN	HWADCTN	IKKDIKCEAD	WWIDWINATING	ODOLDDETLE	DEKIC VDED	KNEME VAKT	152
101	D FOTEESCP	MACA	CORVEY	HWADOTH	IKKDIKODAD		OBOLDBETLE	PORIO UDER	DUENC VART	150
101	- CIEESCP	VM SA	GPNIEI	HWADGIN	INNPINCSAP		ODGEDDETER	PORIG- VPER	IN INS. VARI	150
107	D- CTEESCP	MMSA	···· GPRTET	HWADGIN	INNPINCSAP	KYIDYLMIWV	ODGEDDETEF	PSKIG-VPEK	HNEMS- VAKI	150
46	D- FCREESCS	MSA	GPKYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	QDQLDDETLE	PSKIEGVPFK	RNEMS- VAKT	151
37	D- ECTEESCP	LMSA	GPKYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	QDQLDDETLF	PSKIG- VPFP	KNEMS- VAKT	150
76	D- ECTEESCP	VMSA	GPKYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	QDQLDDETLF	PSKIG-VPFP	KNEMS- VAKT	150
123	D- ECTEESCP	VMSA	GPKYEY	HWADGTN	IKKPIKCSAP	KYIDYLMTWV	QDQLDDETLF	PSKIG- VPFP	KNEMS- VAKT	150
38	D- FCSEDSCP	VMSA	GPKYEY	HWADGTN	<b>IKKPIKCSAP</b>	KEIDYLMTWV	ODOLDDETLE	PSKIG- VPEP	KNEMS- VAKT	150
18	E- ECTEDICS	IMSA	···· GPKYEY	HWADGOT	VKKPIKCSAP	KYIDYLMTWY	ODOLDDETLE	PSKIG-VPEP	KNEIN- LAKT	151
113	E. ECTEDSCS	IMSA	GPKYEY	HWADGOT	VKKPIKCSAP	KYLDYLMTWY	ODOLDDETLE	PSKIG- VPEP	KNELO- LAKT	148
11	E. ECTEETCO	IMCA.	GREVEN	HWADGET	VERDINCEAD	KYIDYIMTWY	ODOLDDETLE	DEKIG. VDED	KNEHS CART	150
122	EECTEESCO	INCA	CREVEN	HWADOUT	VERDINCEAD	VY I BY LATWY	OPOLODETLE	DEVIC VDED	KNELC LAKT	150
132	E- CIEESCP	MOA	CONVEY	HWADGHT	VKKPIKCSAP	KT I DT LMT WY	ODOLDDETLE	PSKIG-VPPP	KNELS- LAKT	150
149	E- ECTEESCP	MSA	GPRIET	HWADGHT	VKKPIKCSAP	KYIDYLMIWV	QUQUUUEILE	PSKIG-VPFP	KNELS- TAKI	150
61	E- PCILESCP	VMSA	GPKYEY	HWADGII	IKKPIKCSAP	KYIDYLMIWV	ODGEDDETLE	PSKIG-VPFP	KNELA- LAKI	150
96	E- ECTTDKCP	MSA	GPKYEY	HWADGQT	VKKPIKCSAP	KYIDYLMTWV	QDQLDDETIF	PSKIG- VPFP	KNEMT - LAKT	149
169	E- ECTATKCE	VMSA	GPKYEY	HWADGVT	IKKPIKCSAP	RYIDYLMTWV	QCQLDNEEIF	PSAVG- VPFS	KNEMS- LAKT	150
153	E- ECTDDTCP	IMSA	GPKYEY	HWADGQT	VKKPLKCSAP	HYIDCLMIWI	QKQLENEAIF	PSKIG- APFP	RDFLN-VVKV	152
69	E- ECTPELCP	VMSA	GPKYEY	LWADGON	VRTPLKVSAS	EYIDYLMTWV	ENQLNNDSLF	PCQIG- IPFP	NTELS- VVKV	152
70	E- ECTPELCP	IMSA	GPKYEY	LWADGON	VKTPLKVSAS	EYIDYLMTWV	ENGLNNDSLF	PCQIG- IPFP	<b>KTFLS- VVKV</b>	146
64	E- ECTPENCS	TMSA	GPKYEY	RWADGVQ	IKKPIEVSAP	KYVEYLMDWI	EAQLDDESIF	PORLG- APEP	PNEKE- VVKT	151
119	E- ECTPESCP	TMTA	GPKYEY	RWADGVO	IKKPLEVSAP	KYVEYEMDWI	EGOLDDESLE	POKLG- TPEP	PNEKE- VVKT	150
143	F. ECTPONCE	TMTA	GPKYEY	RWADGVO	IKKPLEVSAP	KYVEYLMDWL	FTOIDDETLE	POBL G. APEP	ONEKD- VVKT	150
73	E. ECTRSNCP	TMTA	GPKYEY	RWADGUT	IKKPLEVSAP	KYVEYLMDWM	ESOLDDETLE	PORI G. APER	PNERD, VVKT	151
74	ECTRENCE	TMTA	CREVEN	PWADCHT	IKKDIEVSAD		ESOLDDETIE	POPLC. APLA	KTIK.	146
77	E FOTRONOR	THE	COMMEN	BWAD OVT	I KKDI EVOAD		FOOLDBETTE	PORLO APER	DUCOD UUUT	101
12	- CTPSNCP	CMTA	GPKTEY	WADGVI	KAPTEVSAP	KTVETLMDWI	ESOLUDETTE	PORLG- APPP	PNERU- VVKT	151
202	E- ECTPSNCP	SMTA	GPKYEY	HWADGVT	IKKPIEVSAP	KYVEYLMDWI	ESQLODETTE	PORLG- APPP	PNERD- VVKT	151
65	E- ECTAANCP	TMTA	GPKYEY	RWADGVT	IKKPIEVSAP	KYVEYLMDWI	EAQLDDELIF	POKLG- APFP	PNEQD- VVKT	151
144	E- FCTPENCS	TMTA	GPKYEY	RWADGVQ	IKKPIEVSAP	KYVEYLMDWI	ETQUDDETIF	PQKLG- AAFP	PNEKE- VVKT	152
120	E- ECTPTTCP	TMSA	GPKFEY	RWADGIQ	I KKPI EVSAP	KYVEYLMDWI	EVOLDDESIF	PQKLG- TPFP	<b>QNERE- VVKT</b>	152
121	E- FCTSSTCP	IMSA	GPKYEY	RWADGMK	VKKPVQVSAP	KYVEYLMDWV	ESQLDDEAIF	PQKIG- APFP	<b>QNFRE-VIRT</b>	151
66	E- ECTERTCE	VMSA	GGKYEY	LWADGVK	VKKPVRLSAP	EYINKLYDWI	EEQIDDDKTF	PQQFG- SPFP	PNEME-VIKT	150
130	E- ECTAASCP	QMNA	GPSYEY	YWQDDKI	YTKPTRMSAP	DYINNLLDWT	QEKLDDKKLF	PTEIG-VEFP	KNERK- VIQQ	148
145	E- FCTQTTCP	VMNAG	RYEY	RWADGTTIT-	KPKTVSAP	KYVEYLIDWV	ETEIDNEAIF	PKNPG- EPFP	PNFED- FVKR	91
146		- MKAG	RYEY	RWADGTTM	VSAP	EYVELLMNWI	ETQIDNEHIF	PKKTG- EPFP	PNFED- FVKR	58
147	E- ECSPOTCP	RMIA	TNEYEY	LWAFQKG	- QPPVSVSAP	KYVECLMRWC	QDQFDDESLF	PSKVT-GTFP	EGFIORVIOP	153
16	EAECTDIKCP	SMT A	HGRQY	TWT SDET	VLNTSAP	QYIDLSLTSC	QUNIDDENVE	PSEIG- KOFP	ANFEE- RCQT	117
86	ESECTDSKCP	SMT A	HGRQY	TWT SEGT	LLNT SAP	QYIDLSLTAC	QNNVDDENVF	PSEIG- KOFP	TDFEE- RCQA	128
131	T- FCTVKTCP	VMSA	AANFDY	TWLDNNR	KPVHLPAP	QYIEYVLAWI	ENRLHDQNVF	PTKAG- LPFP	SNELV- IVKA	150
148	E- YVTPDAYP	TMNA	GPHTDY	LWLDANN	ROVSLPAS	QYIDLALTWI	NNKVNDKNLF	PTKNG- LPFP	QQESR- DVQR	150
7	- EFCTGETCO	TMAVCN- TQ-	Y	YWYDERGK	KVKCTAP	QYVDFVMSSV	QKLVTDEDVF	PTKYG- REFP	SSFES- LVKK	146
95	- EECTGETCO	TMAVCN- TQ-	¥	YWYDERGK	KVKCTAP	QYVDFVMSSV	OKLVTDEDVF	PTKYG- REFP	SSFES- LVKK	149
179	- EFCTGETCO	TMAVCN- TQ-	Y	YWYDERGK	KVKCTAP	QYVDFVMSSV	OKLVTDEDVF	PTKYG- REFP	SSFES- LVRK	146
200	- EFCTGETCO	TMAVCN- TQ-	Y	YWYDERGK	KVKCTAP	QYVDFVMSSV	QKLVTDEDVF	PTKYG- REFP	SSFES- LVRK	149
35	- EFCTGEACO	TMAVCN- TQ-		YWYDERGK	KVKCTAP	QYVDFVMSSV	QKLVTDEDVF	PTKYG- REFP	SSFES- LVKK	154
140	- EFCTGESCO	TMAVCN- TQ-	Y	YWYDERGK	KIKCTAP	QYVDFVMSSV	OKLVTDEDVE	PTKYG- KEFP	NSFES- LVKK	149
81	- EFCTGETCO	TMAACN- TQ-	Y	YWYDERGK	KLKCTAP	QYIDFVMSSV	QKLVTDEDVF	PTKYG- REFP	SSFES- LIKK	150
128	- EFCTGETCO	TMAACN- TQ-	Y	YWYDERGK	KVKCTAP	QYIDFVMSSI	QKLVTDEDVF	PTKYG- REFP	SSFES- LVKK	150
129	- EFCTGETCO	TMAACN- TQ-	Y	YWYDERGK	KVKCTAP	QYIDEVMSSV	OKLVTDEDIF	PTKYG- REFP	SSFES- LVKK	146
50	- EFCTGDTCQ	AMNACN- TI-	Y	YWYDERGK	KTKCTAP	QYVDFVMSLC	OKLVTDEEIF	PTKYG- KEFP	NSFES- LVKK	154
111	- EFCTGETCO	AMTACN-TI-	Y	YWYDEKGK	KTKCTAP	QYVDFVMSLC	OKLYTDEELF	PTKYG- KEEP	NSFES-LVKK	151
43	- EECTGDTCO	AMTAYN-TI-	Y	YWYDERGK	KTKCTAP	QYVDLVMTEV	OKLVTDEELF	PTKYG- KDEP	NSFES-LVKK	151
44	- EECTGDTCP	AMSAYS- TT-		EWYDEKGK	KTKCTAP	QYVDEVMSSV	OKLYTDEDIE	PTKYG- KEEP	NTEDS-LVKK	149
172	EECTADTOP	IMOGPGOKO-		AWVDERGK-	KLKCTGP	OYVDYVMTEC	OKCASNODIE	PTKYA-OTEH	ESEYS- ELKK	139
12	- EECTOSGCA	DMTGPGNRT-		LWEDEKGK-	KTRVAAP	QYIDYVMTET	OKTYSDESIE	PTKYA- NEEP	GSEES- LARK	143
117	- EECTOSGCA	DMTGPGNRT.		LWEDEKGK-	KTRVAAP	QYIDYYMTET	OKTYSDESIE	PTKYA-NEEP	GSEES- LARK	150
20	- EECTTSGCP	DMTGPGT BM-		LWEDEKGK-	KTRYAAP	QYIDYYMTET	OKTVSDESLE	PTKYA-NEEP	SSEES- LARK	150
115	EECTTSGCP	DMTGPGTPM		IWEDEKCK.	KTRYAAP	OVIDYVMTET	OKTYSDESIE	PTKYA-NEED	SSEES. LAPK	150
151	FECTMICOD	DMTGRGUPT		IWEDEKCK	KTRVAAR	OVIDVNATET	ORTHEDET	PTKYA MEED	SSEEC. WE	150
124	EECTOROCO	DMT CRCORT		WEDEKOK	KT BUAAP	OVIDVUNT	ONTITUT	DTKYA	CCEEC WER	150
105	FECTOSTCO	TACCRONT CO	WKANEGOLOG	LEIDBOODC.	KAEVAAP	I VEDVANCE	OBLITER	DTKAC CHEP	TOPUE LUCK	130
1105	FEATDETCH	TACCPONTAS	VNAVEGGEUG	EWT DOUCD	KIKOGAD	LYEDYAMOVY	ODULTDEDVE	PTRAG- SVEP	TOFLE LUOK	130
112	EFOTROTOP	TACCRONT		WWT DDUCA	KINGSAP	LYED YAMSTV	OFUT	PTKAC SAFP	TOFIE LUCK	150
21	- EFCIPSICP	TACGPONT		WIDDHGH	NENCSAP	LIFUTAMSTI	GELLIDEDVE	PINAG- SAFP	TOFIE- LVOK	150
103	- EFCTISICP	TACGPGNT		EWIDENGH	HENCSAP	LIFUTALSTI	GELLIDEDVE	PIRAG- AAPP	IGFEL- LVOK	150
82	- EFCITSSCP	IMKAWSIQ		QWIDEKGH	KKKCSAP	QYADYAASII	OKILIDEDLV	PIKHC- KEFP	KTEHP- STOK	149
98	GOFCINDICO	SMMAPG	···· NNIN	QWHDDKGK	KMKCSAP	QYTEFAMVNA	OKHIDDETTE	PTKYG- KVPP	SUFES- VIQH	151
99		THEORET	· · · · · · · · · · · · · · · · · · ·	GWHDDKGK	KMKCSAP	CHIEFAMVNA	CKHIDDETIE	PIKTG- KVFP	SUPES- VIQR	103
03	CTAASCP	INSGPGALT-		EWCODKGKK-	INASTAAP	CHIDION YV	ENTVHDDAVE	PSKEG-MIPP	STELT- TVKK	151
9	GECTERICP	UMOCTOC	GPKYEY	HWODDL	WEATHER		FTHE ROOM		BUCES CKK	90
15	- VCTLOSCP	HMSPPG	ISKA	TODERG	KHOVYPAM	arrie cvitac	EIMSHQEEIF	BTRIG- NKEN	ENTEP-AVKK	150
84	E- VCTQQSCP	HMSEPG	TSKA	TDERG	KHOVYPAV	CALDCALLOC	ESMSHQEEIF	PIKYG-NKEN	GNEEP- AVKK	150
6	USECHPOTCT	UMTAT	EQWIF	LCAAHKT	- PKE CPAI	DYTHHTLDGA	ACLENSNKYF	PSHVSIK	- ESSVAKLGS	149
191	- SECHPDICT	QMTAT	EQWIF	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSHVSIK	- ESSVAKLGS	145
33	QSECHPDICT	QMTAT	EQWIF	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	149
67	USECHPOTCT	UMTAT	EQWIF	CAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	149
94	USECHPOTCT	QMTAT	EQWIE	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSHVSIK	- ESSVAKLGS	149
68	OSECHPDICT	QMTAT	EQWIF	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	116
178	QSECHPDICT	QMTAT	EQWI F	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	126
199	QSECHPDTCT	QMTAT	EQWIF	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	149
80	QTECHPDTCT	QMTAT	EQWIF	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	149
127	QTECHPDTCT	QMTAT	EQWIF	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	149
		OMTAT	E CWIE	CAAHKT	- PKE CPAL	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	102
139	OSECHPDICI	WIT AT-			the second s			the loss set in the loss of th		

162	OSECHPOTOT	OMTAT		LCAAHKT	- PKE- OCPAL	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	150
49	OSECHPDICT	OMTAT	····· EOWLE	LCAAHKT	- PKE CPAL	DYTRHTLDGA	ACLENSNKYE	PSRVSIK	- ESSVAKLGS	150
42	ONECHPOTCT	QMTAT	EQWIE	LCAAHKT	- PKE CPAL	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	149
34	OSECHPDTCT	QMTAT	EQWIE	LCAAHKT	- PKE CPAL	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	135
192	- SECHPDTCT	QMTAT	EQWIF	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	133
24	OSECHPDTCT	QMTAT	EQWIE	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSXXXXX	- XXXVAKLGS	83
13	OKECSPSTCT	QMTAT	DOWLE	LCAAHKT	- PKE CPAL	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVTKLGS	149
118	QKECSPSTCT	QMTAT	DOWLE	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVTKLGS	149
135	<b>QGECHPETCT</b>	QMTAT	EQWIE	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	149
152	QAECHPETCT	QMTAT	EQWIE	LCAAHKT	- PKE CPAL	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	149
21	<b>QTQCEPATCT</b>	QMTAT	EQWIE	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- DSSVAKLGS	149
116	<b>QTQCEPATCN</b>	QMTAT	EQWIE	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVSKLGS	149
171	QAECTPMTCS	QMTAT	EQWIE	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSR FSIK	- DSSVAKLGS	149
62	ONECSPSTCT	QMTAT	EQWIE	LCAAHKT	- PKE CPAL	DYTRHTLDGA	ASLLNSNKYF	PSRVSIK	- ESSVAKLGS	149
142	QSECHPDTCT	QMTAT	EQWIE	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSR		136
97	OHECNPDTCS	QMTAT	EQWIE	LCAAHKT	- PKE CPAI	DYTRHTLDGA	ACLENSNKYF	PSRVSIK	- ESSVAKLGS	138
155	<b>QLECTPKTCP</b>	QMTAT	DOWIF	LCAAHKT	- PKE CPAV	DYTRHTLDGA	ACLENSSKYF	PSRVSIK	- VTSVNRLDS	149
17	LRECVPETCO	QMTAT	EQWIE	LCAAHKN	- PNE CPAI	DYTRHTLDGA	ATLLNSNKYF	PSR VNIK	- EISISKLGS	144
85	<b>QRECIPETCO</b>	QMTAT	EQWIE	LCAAHKN	- PNE CPAL	DYTRHTLDGA	ATLLNSNKYF	PSR VNIK	- EISISKLGS	149
71	KGLCTAQTCP	KMKAT	· · EDWLY	LCAAHKK	- AQE CSAI	DYMIHNLDOS	TSILTNIKTY	PSRVSIN	PONATNNEAF	150
Consensus	E-FCTEETCP	VMSA	GPKYEY	RWADGKT	-KKPTKCSAP	KYIDYLMDWI	QDQLNDETIF	PSK-G-VPFP	KNFMS-VAKK	
Conservation				-lalana	- Changella					

		200	10	220		24	0	260		
175	<b>ILKELERVYA</b>	HITHOHEDPV		TSEKHELEEV	QEENLIDER-		LAPL			204
2	ILCRLERVEV	HVYIHHEDRV	I VMGAEAHVN	TCYKHFYYFV	TEMNLIDRK-	<b>E</b>	LEPL			207
89	ILCRLFRVFV	HVYIHHFDRV	I VMGAEAHVN	TCYKHFYYFV	TEMNLIDRK-	E	LEPL			207
28	ILCRLFRVFV	HVYIHHEDRV	I VMGAEAHVN	TCYKHFYYFV	TEMNLIDRK-	· · · · E	LEPLKEMTSR	MCH		216
165	ILCRLERVEV	HVYIHHEDRV	IVMGAEAHVN	TCYKHEYYEV	TEMNLIDRK-		LEPL			207
1/4	I LCRLFRVFV	HVYIHHFDHV	I VMGAEAHVN	TCYKHFYYFV	TEMNLIDEK-		LEPL			207
101	ILCRIERVEV	HVYTHHEDRV	I VMGAEAHVN	TCYKHEYYEV	TEMNLIDEK-		LEPL			207
22	LCBLEBVEV	HVYTHHEDRY	IVMGAEAHVN	TCYKHEYYEY	TEMNLIDEK-		LEPL			207
187	I LCRLERVEV	HVYIHHEDRV	I VMGAEAHVN	TCYKHFYYFV	TEMNLIDEK-		LEPLK			208
52	ILCRLERVEV	HVYTHHEDRV	I VMGAEAHVN	TCYKHFYYFV	TEMNLIR	E	LEPL			191
137	ILCRLERVEV	HVYIHHEDRI	I LIGAEAHVN	TCYKHFYYFV	TELNEIDRK-	E	LEPL			207
77	ILCRLERVEV	HVYIHHEDRI	IMMGAEAHVN	TCYKHFYYFV	TELNLVDRK-		LEPL			207
4	IL SHLERVEV	HVYIHHEDRI	AQMGSEAHVN	TCYKHEYYEV	TEESTIDPK-		LEPLK			208
8	IL SALEAMEN	···· DDITDY	SLETPPVR		I SEIDER.		MAL NO.			155
185	VGR	GSGIHSLPEK	SGRPSTVAHT	RNASALG						173
31	ILSRLFRVFV	HVYIHHFDRI	AQMGSEAHVN	TCYKHFYYFV	KEFGLIDTK-	E	LEPLK			208
176	ILSRLERVEV	HVYIHHEDRI	AQMGSEAHVN	TCYKHFYYFV	KEFGLIDTK-	E	LEPLK			208
167	ILSRLERVEV	HVYIHHEDRI	AQLGSEAHVN	TCYKHFYYFV	TEFGLIDTK-	· · · · · · · · · · · E	LEPLK			208
53	I LSRLFRVFV	HVYIHHEDRI	AQMGAEAHVN	TCYKHFYYFV	REFGLIDTK-		LEPLK			208
125	I SBI FRVEV	HVYIHHEERI	THMGAEAHVN	TCYKHEYYEY	TEINLIDTK		I E PI			207
188	LSBLEBVEV	HVYIHHEDRI	TOMOSEAHVN	TCYKHEYYEV	KEENLIDTK-		LEPLK			208
102	ILSRLFRVFV	HVYIHHFDRL	SOMGAEAHVN	TCYKHFYYFV	TEFNLTDHK-	E	LEPL			207
108	ILSRLFRVFV	HVYIHHFDRV	SQMGAEAHVN	TCYKHFYYFV	TEFNLTDHK-	E	LEPL			207
158	ILSRLFRVFV	HVYIHHEDRV	SQMGAEAHVN	TCYKHFYYFV	IEFNLMDHK-	· · · · · · · · · · E	LEPLK	•••••	•••••	208
159	I LSRLERVEV	HVYTHHEDRV	SOMGAEAHVN	TCYKHFYYFV	IEENLMDHK-		LEPLI		5-15 - 14 - 14 - 14 - 14 - 14 - 14 - 14	208
170	IL SHLERVEV	HVYTHHEDRV	HSMGAEAHVS	ACYKHEEVEV	KCEGLVDKK-					207
5	LITBLERVEY	HVYIHHEDSI	LSMGAEAHVN	TCYKHEYYEI	OFFSLVDOR-		LEPL			207
93	ILTBLEBVEV	HVYIHHEDSI	LSMGAEAHVN	TCYKHFYYFI	QEFSLVDQR-	· · · · · · · · · · · ·	LEPL			207
177	ILTRLERVEV	HVYIHHFDSI	LSMGAEAHVN	TCYKHFYYFI	REFSLVDQR-	E	LEPL			207
198	ILTRLERVEV	HVYIHHFDSI	LSMGAEAHVN	TCYKHFYYFI	REFSLVDQR-	E	LEPLREMTER	ICH		216
183	ILTRLEBVEV	HVYIHHEDSI	LSMGAEAHVN	TCYKHFYYFI	REFSLVDOR-	· · · · · · · · · · E	LEPL		•••••	207
168	ILTRLERVEV	HVYIHHEDSI	LSMGAEAHVN	TCYKHEYYEI	REFSLVDOR-		LEPL			207
190	ILTRI FRVEV	HVYIHHEDGI	LAMGAEAHVN	TCYKHEYYEI	OFFSLVDHR.		EPIR.			208
79	ILTRLERVEY	HVYIHHEDAL	ISVGAEAHVN	TCYKHEYYEI	TEFSLVDHR-		LEPL			207
126	ILTRLERVEV	HVYIHHEDAL	I SVGAEAHVN	TCYKHFYYFI	TEFSLVNHR-	<b>E</b>	LEPL			207
138	ILTRLFRVFV	HVYIHHFDSI	INMGAEAHVN	TCYKHFYYFI	REFSLVDHR-	E	LEPL			207
47	ILSRLFRVFV	HVYIHHFDSI	CSMGAEAHIN	TCYKHYYYFI	SEFHLIDHS-	E	LEPLKEMTEK	ICN	• • • • • • • • • • •	217
48	ILSRLERVEV	HVYIHHEDSI	CSMGAEAHIN	TCYKHYYYFI	SEFHLIDHS-	· · · · · · · · · · · E	LEPL			208
103	ILSRLFRVFV	HVYIHHEDSI	CSMGAEAHIN	TCYKHYYFI	SEENLIDUS-		LEPL			207
160	ILSBLERVEV	HVYIHHEDSI	CSMGAEAHIN	TCYKHYYEEI	SEENLIDNS.		I EPI R.			208
41	ILSELFEVEV	HVYIHHEDMI	CSIGAEAHIN	TCYKHYYYFI	SEFSLIDHS-	E	LVPL			207
55	XXXXXXXXXXX	XXXXXXXXXXX	XXXXXXXXXXX	XXXXXXXXXXX	XXXXXVDQ					201
19	ILTRLERVEV	HVYIHHEDRI	FSIGAEAHVN	TCYKHFYYFV	TEFDLMSAK-	E	LEPLA			208
114	ILABLERVEV	HVYIHHEDRI	VSIGAEAHVN	TCYKHFYYFI	QEFDLMSAK-		LEPLAVMTAQ	MCKDV	•••••	221
10	ILTRLERVEV	HVYTHHEDRI	VSIGAEAHVN	ACYKHEYYEV	QEFDMISAK-		LEPLQ			208
150	I TRI FRVEV	HVYIHHEDRI	VALGAEAHVN	TCYKHEYYEY	TEFELINTK.		LEPLAE.			200
14	ILTRLERVEV	HVYIHHEDRI	RELGAEPHAN	TLYKHEYEEV	TEYGMV SAK-		LEALKOMTER	LEPSNRRAP	IPSANAERO-	232
83	I LTRLERVEV	HVYIHHFDRI	RELGAEPHAN	TLYKHFYFFV	TEYGMVSTK-	E	LEALKOMTER	LLEPSNRRAP	IPSANAFRS-	232
154	IFGRLERVEV	HVYIHHEDRL	HEIGAEAHVN	TCYKHFYYFV	TYFDLIDKK-	E	LEPLN			208
156	IFY	FTWI BRCS	LSEMLYS	NCKENIWP				• • • • • • • • • • • •		124
58	I FKRMERVYA	HMYYNHEKDA	KULGEEAHLN	TAEKHEMCEV	NEFDLIDKK-		EPLKGV			208
57	IEKBLERVYA	HITHSHEEV	KKIGEEAHLN	TAEKHECLEY	FEEDLVSKK		VEPM.			103
92	LKRLERVYA	HITHOHEDSV	MOLQEEAHLN	TSEKHELEEV	QVSEPC					196
197	ILKRLERVYA	HIYHQHEDSV	MQLQEEAHLN	TSFKHFIFF.						189
184						• • • • • • • • • • •	• • • • • • • • • • •		•••••	137
201	LWK-	UTHIOUTHOR		YSFF-			LADE			147
182	I LKRLFRVYG	HIYHOHEDSV	MOLOEEAHLN	TSEKHELEEV	QEENLIDHC-					204
173	ILKELEBVYA	HITHOHEDSV	MOLOEGAHLN	TSEKHELEEV	OFENI IDER.					204
194	ILKELERVYA	HIYHQHEDSV	MQLQEEAHLN	TSEKHELEEV	QEENLIDER-		LAPL			204
164	ILKRLFRVYA	HIYHQHEDSV	MQLQEEAHLN	TSFKHFIFFV	QEENLIDER-	E	LAPL			204
87	I LKRL FRVYA	HIYHQHEDSV	MQLQEEAHLN	TSFKHFIFFV	QEENLIDER-	· · · · · · · · · · E	LAPL			204
27	ILKRLERVYA	HIYHQHEDSV	MQLQEEAHLN	TSFKHFIFFV	QEENLIDER-	· · · · · · · · · E	LAPL			204
100	I LKRLFRVYA	HIVHQHEDSV	MQLQEEAHLN	TSEKHEIFEV	GEENLIDER-	· · · · · · · · · · E	LAPL			204
186	ILKELERVYA	HIYYOHEDAY	MOLOFEAHLN	TSEKHELEEV	OFENI TORP		LAPLGELIEK	EGONUN		218
100	ILKELEBYYA	HIYHOHEDSV	MOLQEEAHLN	TSEKHELEEV	QEENLIDER-		LAPL			199
106	ILKELFEVYA	HIYHQHEDSV	MQLQEEAHLN	TSFKHFIFFV	QEENLIDER-	E	LAPLOELIEK	LGSKDR		218
36	ILKRLERVYA	HIYHQHFDSV	MQLQEEAHLN	TSFKHFIFFV	QEENLIDER-	· · · · · · · · E	LAPL			128
75	ILKELERVYA	HIYHQHEDAV	MQLQEEAHLN	TSEKHEIFEV	QEENLIDER-	· · · · · · · · · · ·	LAPL	•••••		204
157	I LKRLFRVYA	HINHOHEDSV	MOLOEEAHLN	TSEKHEIFEV	GEENLIDER-		LAPL			204
124	ILKELERVYA	HITHOHEDAY	MOLOFEAHLN	TSEKHELEEV	QEENLIDER.		LAPL			190
141	ILKELERVYA	HIYHOHEDSV	MRLQEEAHLN	TSEKHELEEV						98
122	I LKRLFRVYA	HIYHQHEDAV	MQLQEEAHLN	TSEKHEIFEV	QEENLIDER-	E	LAPL			203
39	ILKELFEVYA	HIYHQHEDAV	IQLQEEAHLN	TSFKHFIFFV	QEENLIDER-	E	LAPL			204
3	<b>LKRLERVYA</b>	HITHOHEDPV	OLOEEAHLN	TSEKHELEEV	OFFNLIDER-	E				204

90	LIKRIERVYA	HIVHOHEDPV	<b>IOLOFEAHIN</b>	TSEKHELEEV	OFENILIDER.		TAPI.			204
166	IL VOLEDVYA	HIVHOHEDRY	LOLOFEAHLN	TCERNETEEV	OF ENLLOPP					204
126		HIVHOUEDDV	TOTOFFAHLEN	TOERUELEEV	OF ENLLOPPO	101010-0010-0010-001		10-57-00-20-00-00-00-00-00-00-00-00-00-00-00-		204
130	I L NALEAVIA	HITHUHFUPV	ICLOEEAHLN	I SEKHELEEV	OFFICE OFFICE			TTOWN.		204
29	ILKHLPHVYA	HITHQHEDPV	TOLOEEAHLN	ISEKHETEEV	GEENLIDER-		LAPEQUELIEK	LISKUH		218
196	ILKELFEVYA	HIYHQHEDPV	IQLQEEAHLN	TSEKHEIEEV	QEENLIDER-		LAPLQEL			209
101	ILKRLFRVYA	HIYHQHEDSV	MQLQEEAHLN	<b>TSFKHFIFFV</b>	QEENLIDRK-		LVPL			204
107	ILKRLFRVYA	H I YHQH FD SV	IQLQEEAHLN	<b>TSEKHEIEEV</b>	QEENLIDRK-		LVPL			204
46	<b>ILKRLFRVYA</b>	HIYHHHFDSV	IQLQEEAHLN	TSFKHFIFFV	QEENLIDRK-		LVPL			205
37	LEKRLERVYA	HIYHOHEESV	LOLOFEAHLN	TSEKHELEEV	OFENLIDEK-		LAPL			204
76	LIKELEBVYA	HIVHOHEDSV	LOLOFEAHLN	TSEKHELEEV	OFENI IDER.		OAPI			204
122	IL KRIERVYA	HINHOHEDCV	LOLOFEAHLN	TOEVUELEEV	OF ENLLOPPO		OAD			204
125	TENALEAVIA	ni indri u sv	TOLOEEAHLN	I SP KHEI FEV	GEFNEIDAR.		GAPL			204
38	ILKHLPHVYA	HIYHQHEDAV	MOLQEEAHLN	TSEKHETEEV	GEENLIDHK-		LAPL			204
18	ILKRLFRVYA	HIYHQHESEV	VRLSEEAHLN	TSEKHEIYEV	QEENLIDER-		LAPL			205
113	ILKRLFRVYA	HIYHQHESEV	VRLSEEAHLN	TSFKHFIYFV	QEENLIDER-		LAPL			202
11	ILKRLFRVYA	HIYHQHETEV	VTLGEEAHLN	TSEKHEIEEV	QEENLIERR-		LAPL			204
132	<b>ILKRLFRVYA</b>	HIYHOHE SOV	VOLGEEAHLN	TSEKHEIEEV	QEFSLIERR-		QAPL			204
149	<b>ILKBLEBVYA</b>	HIYHOHESEV	VOLGEEAHLN	TSEKHELEEV	OFENLIERR-		LAPL			204
61	LIKBLEBVYA	HIVHOHEKHY	VSLGEEAHLN	TSEKHELEEV	OFEST LOKR.					204
06	LIVPLEPVYA	HIVHOHEKEI	VILAFEAHIN	TSERVEIVEV	OF ENLLOPK	consistence of a rate of				202
160	IL VOLEDVYA	HIVHOUECOV	MOLOFEAHLN	TCEVUEIVEV	OF ENLLOPP					203
109	TENNERNYTA	HITHQHE SUV	MOLGEEAHLN	I SEKHELLEV	GEENLIUGHA.			TTT TTTTTT		204
153	ILKRLFRVYA	HIYYOHETEV	HDLQEEAHLN	TSFKHFIYFV	LEFNLVQKR-		LVPLOHLIDL	LTTDESVTYN	QHNNDNNNN	232
69	IFKRLFRVYA	HIYHSHFQHI	MALELEYHLN	TCEKHEIYEI	DEFKLVESK-		LAPLAELIQQ	FKARKENPTM	NQGM	226
70	IFKRLFRVYA	HIYHSHFQHI	MALELEYHLN	TCFKHFIYFI	DEFKLVEDK-		LAPL			200
64	<b>IFKRLFRVYA</b>	HIYHSHFQKI	VSLKEEAHLN	TCEKHEILET	HEFGLIDKK-		LAPL			205
119	<b>IFKRLFRVYA</b>	HIYHSHFOKI	VSLKEEAHLN	TCEKHEILET	TEFGLIDKK-		LAPL			204
143	<b>I EKBLEBVYA</b>	HIYHSHEQKI	VSLKEEAHLN	TCEKHEILET	HEEGLIDKK-	E	LAPL			204
73	I EKBLEBVYA	HITHSHEOKI	VSLKEEAHEN	TCEKHEVLET	WEERLIEKA-		LAPL			205
74										146
72	I EKRI ERVYA	HVVHSHEOFT	VSIREEAHEN	TCERHEVIET	WEERLICKA					205
202	I EKOLEOVIA	HITHSHEGKI	VOLKEEAHLN	TOPRHEVLET	WEEDLIERA-					205
202	TERREPRVYA	HITHSHFORI	SLKEEAHLN	TOPKHEVLET	WEFRLIDKA-		LAPL			205
65	IFKRLFRVYA	HIYHSHFQKI	VSLKEEAHLN	TCEKHEVLET	WEFRLIDKG-		LAPL			205
144	IFKRLFRVYA	HIYHSHFQKI	VSLKEEAHLN	TCFKHFILFT	HEFVLIDKK-		LAPLQELIES	I LAPY		217
120	IFKRLFRVYA	HIYHTHFOKI	VSLKEEAHLN	TCEKHETLET	WEFKLIDKA-	· · · · · · · · · · · E	LAPLIDLIES	IVS		215
121	I FKRLFRVYS	HMYHSHEQMI	LKLKEEAHLS	TCFKHEVLF-						190
66	VEKBLEBVYA	HIYHSHEKAL	CSLGEEAHLN	TCEKHELEEV	THYNLVDEK-		LAPL			204
130	I FRELERIYA	HIYCSHEHVM	VAMELESYLN	TSEKHEVEEC	REEGLMDNK-		YAPM			202
145	II PKIEPVYA	HIVYSHEHEI	VALNEGAHLN	TCERHELLEY	SEEOLVOKEK		MAPL			146
145	IL DELEDVYA	HIVICHERVI	VTINEGALLEN	TCENPYLLEY	SEEGLUDKE		MUDI			112
140	TERREPRYTA	<b>HINGSHEPKI</b>	VIENEGAHEN	TOPRHELEV	SEPULYDRE.		My PILLE	No.		112
14/	<b>ILBRLEHVYA</b>	HIYCHHENEI	LELNLQTVLN	TSFRHECLEA	GEFELLHPA-		EGPLLELVME	LHDH		217
16	IMARLERIYA	HVYFAHVSHF	KEIKALPHLN	TSFKQEVLEA	NOFHLLNKE-		TEPL			171
86	IMRRLFRIYA	HVYFAHVNHF	KAIKALAHLN	TSFKQFVLFA	NOFOLLNKE-		TEPL			182
131	IYKQMERIEA	HMYYAHYAEI	LHLSLEAHWN	SFFAHFIAFG	KEFQLLDKR-	D	TAPLKDLIVV	LENQGNI		217
148	IMVOMERIEA	HIYHHHEDKI	VHLSLEAHWN	SFESHEISFA	KEEKIIDBK-		MAPLLPLIES	FEKQGKIIYN		220
7	ICKYLEHVLG	HIYWAHEKET	LALELHGHEN	TLYVHEILEA	BEENLLDPKE	T	A- MM			200
95	ICKYLEHVIG	HIVWAHEKET	LALEIHGHIN	TINVHELLEA	REENLIDEKE		A. WMDD			205
170	I CONTERNITA	HI WWALLENET	LALELHOHLN	TINULFILEA	DEENLLBOKE					200
1/9	I CHALFAVLA	HITWAHEKEI	LALELHGHLN	ILIVHEILEA	REFNELDPRE		A- 1			200
200	ICHHLFHVLA	HIYWAHEKET	LALELHGHLN	TLYVHEILEA	REENLLDPKE		A- MDD			205
35	ICKYLFHVLA	HIYWSHEKET	LALELHGHLN	TLYVHFILFA	REENLLDPKE	T	A- WMDDLTEV	LCSAGGRGGG	GGDGASGGGT	234
140	ICKYLFHVLA	HIYSSHFKET	LALELHGHLN	TLYTHEILEI	REFNLVDLKE	• • • • • • • • • T	T- IMDDLTEV	LCSSSGSSSN	GSGNGSSNSA	229
81	<b>ICRYLFHVLA</b>	HIYSAHFKEI	TALELHGHLN	TLFIHELLEV	REFSLLDPKE	T	S- ILDDL			207
128	<b>ICRYLEHVVA</b>	HIYWAHEKEI	TVLELHGHLN	TLEIHELLEV	REFSLLDPKE	T	S- VLDDL			207
129	<b>ICRYLFHVLA</b>	HIYSAHEKEI	AALELHGHIN	TLEIHELLEV	REFSLLDPKE	T	S- IL			200
50	<b>ICRYLEHVLA</b>	HLYWAHEKET	VALELOGHEN	TLYAHEIVEV	BEENLVDPKE	T	C- IMDDLSEV	LSSSONHVT-		223
111	ICRYLEHVIA	HIYWAHEKET	VALDLOGHLN	TIVAHEIVEI	REENLVDPKE		C. M			205
43	VCRYLEHVIA	HIYWAHEKEI	VALDLOGHLN	TIVAHELVEL	REENLIDPKE		C. IMD.			206
44	ICRYLEHVIA	UIVWOUVVET	VANDINCHIN	TINTUEIVEI	REENLMDOKE		C. IMME			200
170		HUNNYCHEADI	KONDUNCHLT	TUERDETHIE	DEFULVEROF		C TM			102
112	LI BLO FIVIA	HI YAAD CAL	ALLOUNDEL		PRENI PROF					193
12	TEREOPHVIA	HLYAAHEHEI	ALLGEHIHLN	LIFAHLIALH	RRENLIDERE					197
117	TEREGENVIA	HEYAAHEHEI	ALLGEHTHEN	LIFAHLTALH	HAENLIDEKE		D- VERDLEVA	LALIDDISGO	USSSSVHEHS	230
20	TVHLLFHVIA	HLYAAHFREV	IMLGLHAHLN	LTFAHLTAFH	HRENLIEPKE	· · · · · · · · · · · I	E- VLRDLEIA	LALTODPTAP	AVTTGADGKA	230
115	ILRLLFHVIA	HLYAAHFREV	ALLGLHAHLN	LTFAHLTALH	REFULTEPKE	· · · · · · · · · · · I	E-VLRDLEIA	LALTDDPVPS	SATSSTGSST	230
151	I LRLLYHVVA	HIYHCHEREV	ALLGLHAHLN	CVFAHLTLLN	ORFNLIDPKE	· · · · · · · · · · · T	E-ILGDLEAA			210
134	IVALLEHVVA	HLYSAHFKEV	VMLGLHAHLN	LTFAHMTALQ	HRESLIESKE	T	E- VLKDLEIA	LRLTEDHESA	NNNERGETST	230
105	VELLEERTLA	HIYWSHYKET	LVLGLHPHLN	TLETHLTLEC	ROHALLEPED	T	E- PL			192
112	VELLEERTLA	HIYWSHYKEM	LVLGLHPHLN	TLETHLTLEC	ROHALLELED	T	E- PL			181
51	VELLEEBTLA	HIYWCHYBET	LALGLHPHLN	TLEAHLTLEC	ROHALLEAED	T	E- PLODLIAA	LRQQA		215
163	VELLLEBILA	HIYWSHYNEA	LALGLHPHIN	TMEAHLTLEC	HQHA					194
82	TERLIEHIIG	HINTSHAKTY	VNLELHPHIN	TEXCHILLEC	AFFOLLOSVE		S- ESEDETTA	I		210
02	II BIMEOVIE	HIVEAUVEON	TRIDIUNUIN	TIETHMAN	OFEKILLOY		SCALEDINEA	HISICICCO	SNPAS	227
90	LERIMEOVE	HIVEAUVEON	TRIOLUUUT	TIETUANULTA	OFERIL		SCOLEDINEA	LHIS CLOSS	SNDAS	170
99	LENEMFOVEE	HECAHTEOM	REPERHEN	CUEL NMYLYA	USER LEOKE		ADI	LAISESESS	SURAS	1/9
63	RELEFIVEA	HEFCAHYADE	KLELHAHLN	CVEINEYLEN	MEENILDPKE		AP			205
9	TECHLEWVEV	HVYIHHEDRL	TVMGAEAHVN	<b>TCYKHFYYFI</b>	IEMNLIDHK-		LEPL			144
15	MLAHLFHCMG	HMYQNHWDVL	GALQLEPQCA	MVFAHIAEIG	RIFNELDTKE	ODIVEDLVIE	LEPILPVLTQ	TLSLDHGDLP	HDADBAIRVP	240
84	MLSHLFHCMG	HMYLKHWDVL	GALQLEPQCA	IVFAHIAELG	RTESLLDAKD	QEQVDECVTE	VRPILPVLSQ	TLSLDDPDHP	HDGDRSSRVP	240
6	VCRRIYRIFS	HAYFHHRQIF	DEVENETELC	HRETKEV	MKYNLMSKON	· · · · · · · · · · ·	IVPILEEEVQ	NS		209
191	VCRRIYRIFS	HAYFHHRQIF	DEVENETELC	HRETKEV	MKYNLMSKON	· · · · · · · · · · I	IVPILEEEVQ	NS		205
33	VCBRIYRIES	HAYEHHBOIE	DEVENETELC	HBETKEV	MKYNLMSKON		IVPILEEEVO	NS		209
67	VCBBIYBIES	HAYENHBOLE	DEVENETELC	HB ETKEY	MKYNLMSKON		IVPIL EEEVO	NS		209
94	VCBBINBIES	HAYENHBOLE	DEVENETELC	HR ETKEY	MKYNIMSKON		IVPIL FEEVO	NS		209
68	VCBBIVBIES	HAYEHHBOLE	DEVENETELC	HR ETKEY	MKYNEMSKEN		IVP.			167
170	VCRRIVELES	HAVEHUDOLE	DEVENETERO	HP	MEYNEMOK		IVP.			177
1/0	TORNITHIES	HAVEHUNGTE	DEVENETEC		MANNEMSKON		TWO TT PROFESS	NONCOROFIC		210
100		TATENERUIE		HA EIKEV	MAINLMSKON		TVPILEREVQ	NON DUESEA-		210
199	VCRRIVRIFS	HAVENNES	DEVENCET	UD ET DE	ALL MALL HAR OWNER	the second se		NC		1114
199 80	VCRRIVRIES	HAYEHHRQIE	DEVENETFLC	HR FTKEV	MKYNLMSKDN		IVPILEEEVQ	NS		203
199 80 127	VCRRIVRIES VCRRIVRIES VCRRIVRIES	HAYFHHRQIF HAYFHHRQIF	DEYENET FLC DEYENET FLC	HRFTKFV HRFTKFV	MKYNLMSKON MKYNLMSKON		IVPILEEEVO	NS		209
199 80 127 139	VCRRIYRIFS VCRRIYRIFS VCRRIYRIFS	HAYFHHROIF HAYFHHROIF HAYFHHROIF	DEYENET FLC DEYENET FLC DEYENET FLC	HR <b>FTKFV</b> HR <b>FTKFV</b> HR <b>FTKFV</b>	MKYN LMSKDN MKYN LMSKDN MKYN LMSKDN			NS		209 153

162	VCRRIYRIES	HAYFHHRQIF	DKYE						 174
110	VCRRIVRIFS	HAYFHHRQIF	DKYENETFLC	HR FTREV	MKYNLMSKDN	· · · · · · · · · · I	IVPILEEEVQ	NT	 211
49	VCRRIYRIES	HAYFHHRQIF	DKYENETFLC	HRFTRFV	MKYNLMSKDN	L	IVPILEEEVQ	NT	 210
42	VCRRIYRIFS	HAYEHHRQIE	DKYENETELC	HR FTREV	MKYNLMSKDN	· · · · · · · · · L	IVPILEEEVQ	SA	 209
34	VCRRIYRIFS	HAYFHHRQIF	DEVENETELC	HR FTKEV	MKYNLMSKDN	· · · · · · · · · · I	IVP		 186
192	VCRRIYRIES	HAYFHHRQIF	DEVENETELC	HR FTKEV	MKYNLMSKDN	<b>.</b>	IVP		 184
24	VCRRIYRIES	HAYFHHRQIF	DEVENETELC	HR FTKEV	MKYNLMSKDN	· · · · · · · · · · L	IVP		 134
13	VCRRVYRIFS	HAYFHHRRIF	DEFEAETYLC	HR FTHEV	TKYNLMSKEN	L	<b>IVPINVGENA</b>	AP	 209
118	VCRRVYRIES	HAFFHHRRIF	DEFEAETYLC	HR FTHEV	TKYNLMSKEN	L	IVPI SDGENA	AP	 209
135	VCRRVYRIES	HAYFHHRSIF	DEFENETELC	KR FTQFV	TKYNLMSKDN	· · · · · · · · · L	IVPILEDENT	PG	 209
152	VSRRVYRIFS	HAYYHHRTIF	DEFENETFLC	BR FTAEV	TKYNLMSKES	· · · · · · · · · L	<b>IVPIMEEEGT</b>		 207
21	VCRRVYRIFS	HAYFHHRRIF	NEFEEETSLC	LR FTNEV	TKYTLMSKEN		<b>IVPIPECELT</b>	PG	 209
116	VCRRVYRIES	HAYYHHBBIF	NEFEEETYLC	LB FTHEV	TKYSLMSKEN	· · · · · · · · · L	IVPIPEGELT	PG	 209
171	VCRRVYRIES	HAYYHHBHIF	DESENETYLC	RR FTTEV	IRYNLMSRDN	· · · · · · · · · · L	IVPI		 201
62	VCRRVYRIFS	HAFFHHRQIY	DOFENETHLC	QR FTSYV	LKYDLMAKDN	<b>L</b>	IVPM		 201
142									 136
97	VCRRIYRIFS	HAYFHHRTLF	DDYENETFLC	RR FTTFI	FALLMAL				 182
155	VCRRVYRIFS	HAYYHHBEIF	DAFEESTALC	KR FTTFV	LKYNLMSKDN	<b>.</b>	IVPIPGAVDG		 207
17	VARRVYRIES	HAFFHHRKLF	DEFENETHLC	KR FTTYV	SKYNLMQQEH	L	IVPILPNQQQ	QQTAQ	 207
85	VARRVYRIFS	HAFFHHRKLF	DEFENETHLC	KR FTTYV	SKYNLMQQEH	L	IVPILPNQQQ	QQQTT	 212
71	IVRRLYRLFS	HTYFNHKEIF	EDFENEMFLC	TR FTEFA	LKFDLMSPKL	📗	T   P		 201
Consensus	ILKRLFRVFA	HIYHHHFDRV	IQLGEEAHLN	TCFKHFIYFV	QEFNLIDRK-	E	LEPL		
	m Bedden		D-D-D			-	270 <b>B</b> as		 1
Conservation			-nin III					-	

Figure 1S. Multiple alignment of the 192 Mob-domain containing proteins. The label number refers the fourth column in Table 1S of the supplementary materials and corresponds to the gene code.

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148 7 95 179 200 35 140 81 128 129 50 111 43 44 172 12 117 20 51 51	GAQNHVKER- GA	ATATAAASLI	DGDSAAPPIC	TOPEAGAGCK	PAGSSGTIGG		GDT	217 220 200 205 243 231 207 207 207 207 207 203 205 205 206 205 193 197 293 239 251 210
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148 7 95 179 200 35 140 81 128 129 50 111 43 44 172 127 117 20 115 151 134	GAQNHVKER- GA	ATATAAASLI SNSADSGSVS TACGDN	DGDSAAPPIC	TOPEAGAGCK	PAGSSGEEGG		GDT	217 220 200 205 243 231 207 207 207 200 223 200 200 200 205 206 205 193 197 293 239 2251 210 246
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