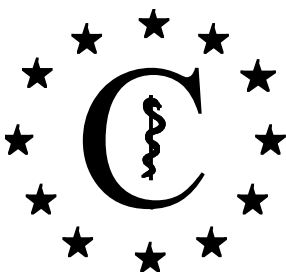




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## ABOUT THIS ISSUE & MORE

### What's inside?

<1> Two flash news, are presented by: (a) Dr. Moh Daha on the role of MASP-2 in myocardial and gastrointestinal injury and b) Dr. Kinga Hosszu on the downregulation of macrophages by platelet microparticles.

<2> Dr. Ghebrehiwet presents one complement team from Tel Aviv, Israel and two other teams represent groups working in the area of MBL/ficolin that were given the opportunity to present their views on the nomenclature of these proteins.

<3> Unfortunately, this issue also includes an "obituary" section to acknowledge the passing away of an outstanding colleague: Dr. Jürg Tschopp.

<4> Please also note that you can now get preliminary information about the XXIV ICS meeting in Crete by visiting the website at: [www.complement2012.org](http://www.complement2012.org)

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## FLASH NEWS

MASP2 & Ischemia reperfusion- Reporter: M. Daha

### Targeting of mannan-binding lectin associated serine protease-2 (MASP-2) confers protection from myocardial and gastrointestinal ischemia/reperfusion injury.

Schwaeble, W.J., N.J. Lynch, J.E. Clark, M.Marber, N.J. Samani, Y.M. Ali, T. Dudler, B.Parent, K.Lhotta, R.Wallis, C.A. Farrar, S.Sacks, H. Lee, M.Zhang, D. Iwaki, M.Takahashi, T.Fujita, C.E. Tedford, and C.M. Stover [PNAS \(Early edition\) April 18, 2011.](#)

In this recently published paper Wilhelm Schwaeble and colleagues in Leicester, London and USA provide strong evidence that IRI in the mouse is strongly dependent on activation of MASP-2 and that there is not a direct role for C4 in this type of injury. This activity of MASP-2 or may be the recognition molecules MBL and Ficolins point to a focused effect of the lectin pathway in the induction of IRI. The authors suggest a possible C4 independent bypass of complement activation that may be responsible for the observed effects. Indeed inhibition of MASP-2 with specific monoclonal antibody also prevented IRI.. As the authors conclude: a transient inhibition of MASP-2 functional activity may therefore provide an attractive therapeutic approach in treating a wide range of ischemia-induced inflammatory pathologies in lectin pathway-mediated disorders. As a matter of fact additional studies to highlight the importance of these mechanisms in primates and humans would be helpful.

Platelets microparticles & DCs- Reporter: K.Hosszu

### Microparticles (ectosomes) shed by stored human platelets downregulate macrophages and modify the development of dendritic cells.

Sadallah S, Eken C, Martin PJ, Schifferli JA. [J Immunol. 2011 Jun 1;186:6543-52. Epub 2011 Apr 27.](#)

The authors investigated novel aspects of microparticles (MP) released by platelets during storage. The notion that MP are formed by budding from the platelet surface is corroborated by data showing surface expression of platelet-derived surface proteins (CD61, CD36, CD47), including complement inhibitors (CD55, CD59), and proteins acquired from plasma (C1q, C3 fragments, factor H), but not CD63. Moreover, Annexin V binding confirmed the expression of phosphatidylserine on the MP surface. These characteristics suggest that the MPs correspond to platelet ectosomes (PLT-Ec). Further experiments revealed that PLT-Ecs can downregulate the inflammatory response of human macrophages, as shown by an increase in TGF- $\beta$ 1 release, and the nearly complete abrogation of TNF- $\alpha$  and IL-10 release induced by zymosan A and LPS. Monocyte derived immature dendritic cells (iDCs) cultured in the presence of PLT-Ec had significantly reduced expression of HLA-DP DQ DR and CD80, exhibited a decreased endocytic capacity, and their LPS-induced maturation was downmodulated. Taken together the data indicate that PLT-Ecs can regulate the normal immune response by modifying the functional properties of macrophages and iDCs. Since MPs are transfused in large amount to patients who are often critically immunosuppressed, further elucidation of their biological properties may be of great clinical interest.

## SPOTLIGHT ON TEAMS

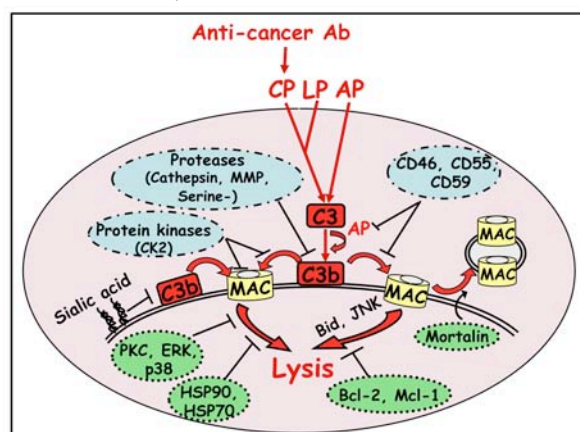
### *Complement in Tel Aviv, Israel*

In north Tel Aviv, in a neighborhood called Ramat Aviv (Spring Heights in Hebrew), just a stone's throw away from the Mediterranean seashore, stands the campus of Tel Aviv University, the largest university in Israel. Within the School of Medicine building, overlooking the sea, you can find Zvi Fishelson's complement laboratory. Zvi first trained as a CTL immunologist. However, after doing his post-doc in La Jolla, California (1980-3), under the guidance of the late Hans Muller-Eberhard, he then became a molecular complementologist. Following an 8-year position at the Weizmann Institute in Rehovot, Israel, he moved to Tel Aviv University in 1992, 20 years after completing his undergraduate degree there. Over the years, diverse complement-related aspects have been investigated in his lab, including complement involvement in tumor immunology, and parasite immunology and complement deficiencies.



Cancer immunotherapy takes advantage of the capacity of complement to kill antibody-coated cancer cells. A growing number of monoclonal antibodies designed for cancer therapy, such as Herceptin and Rituximab, are in routine clinical use. However, despite the potential protective capacity of these antibodies, their therapeutic efficacy is often disappointing. This is partly due to cancer resistance to the cytotoxic action of complement. The molecular basis of the complement resistance of cancer cells has been a major research topic of the group in Tel Aviv. Cancer cells employ several strategies to escape damage by the terminal complement proteins. Data obtained in collaboration with Michael Kirschfink in Heidelberg, Germany, demonstrated the contribution of over-expressed membrane complement regulatory proteins CD46, CD55 and CD59 to cancer

complement resistance. Extracellular proteases and protein kinases related to CK2 can also interrupt the complement cascade, and the deposition of the complement membrane attack complex (MAC). Heavy attack by antibodies and complement may overcome those extracellular protectors and deposit deadly amounts of MAC. However, the MAC will still need to overcome the intracellular survival factors. The death signals are negated by protective intracellular protein kinases, including PKC and ERK, and by heat shock proteins (hsp70 and hsp90). Recently, Zvi's group demonstrated that the mitochondrial hsp70 known as mortalin joins forces with other intracellular repair mechanisms in eliminating the MAC from the cell surface. For its efficient removal, the MAC is positioned in cholesterol-rich domains and endocytosed via caveolae.



Adapted from Gancz & Fishelson, Molec Immunol 46: 2794, 2009

Pre-clinical proof-of-concept experiments are currently being performed by the Tel Aviv team, in which the capacity of mortalin inhibitors to serve as adjuvants to antibody-based therapy is tested in tumor-bearing mice.

Another subject investigated by the group is that of how the terminal complement pathway kills cells. Sublytic doses of MAC are stimulatory to cells and, under certain conditions and in certain cells, even induce anti-cell death signals. Lytic doses of MAC cause a rapid cell death that has all the characteristics of necrosis and osmotic cell death. As shown recently by the group, the MAC activates more than one death signal. One of the death-signaling pathways involves MAC-dependent activation of JNK and Bid, thus having features of programmed necrosis. Members of the survival-signaling pathway are also being studied.

For many years the group has also been investigating the interaction of complement with the parasitic worm *Schistosoma mansoni*. The worm was shown to employ a series of evasion steps and mechanisms that eventually enable it to safely reside in patients in direct contact with blood. Early on, the invading larva transforms and loses its complement-activating glycocalyx. Next, large doses of proteases capable of cleaving complement proteins are released. Finally, the fully transformed worm expresses on its surface a C8/C9-inhibitor: paramyosin. This parasite would appear to have evolved a full armory against complement.

In collaboration with clinicians (Yitzhak Katz and Menachem Schlesinger) several complement-deficient patients have been characterized. One interesting case study involved a compound heterozygous C3 deficiency. This young male patient and his mother both had an abnormal C3, successfully synthesized but then retained in the endoplasmic reticulum and degraded by the proteasome. A mutation in the  $\beta$ -chain (Asp549 to Asn) of this C3 was identified by Lori Singer and Rick Wetsel. Also of interest is a description of properdin-deficiency in Tunisian Jews who suffered from a meningococcal disease.

All complement fans have an open invitation to visit the lab at the department of Cell and Developmental Biology, Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel.

e-mail: [lifish@post.tau.ac.il](mailto:lifish@post.tau.ac.il)

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## VIEWS

### THE LECTIN PATHWAY COMPONENTS NOMENCLATURE

#### *I- Nomenclature for the components of the lectin complement pathway*

Teizo Fujita, Yuichi Endo, Misao Matsushita and Minoru Takahashi, Japan

We really appreciate the opportunity to express our opinion on the nomenclature for the components of the lectin complement pathway.

#### 1. MBL

MBL was first described as mannan-binding protein (MBP) by Toshisuke Kawasaki, but it is now called mannan-binding lectin or mannose-binding lectin and abbreviations are both MBL. We would like to suggest that in general MBL stands for mannan/mannose-binding lectin. In human MBL is encoded by a single gene designated *MBL2*, but in mouse there are two isoforms of MBL, MBL-A (gene name ;*Mbl1*) and MBL-C (*Mbl2*).



## 2. Ficolin

We prefer to use L-ficolin, H-ficolin, and M-ficolin for human proteins although their gene names are *FCN2*, *FCN3* and *FCN1*, respectively. These protein names have been already circulated and we have never used the number such as ficolin1 and ficolin2. In mouse we used ficolin A and ficolin B, corresponding to human L-ficolin and M-ficolin, respectively, but they are slightly different from each other, and there is no murine H-ficolin, which is a pseudogene in mice. In phylogeny, L-ficolin and M-ficolin are closely related, and there is no evidence that L-ficolin was derived from M-ficolin.

In addition, we understand that ficolin is a group of lectins that are independent of collectins, because it lacks a CRD.

## 3. MASP

MBP-associated serine protease (MASP) was first reported by our group as a protease complexed with MBP. We also found that MASPs are associated with ficolins. It was recently reported that MASPs are also associated with CL-K1. Even so, for MASP we prefer to use MBL-associated serine protease, not MBL/ficolin/CL-K1-associated serine protease. The latter name is too complicated and besides there is the possibility that it may be found associated with other proteins in the future.

Obviously, it is established that there are three different MASP proteins (MASPs); MASP-1, MASP-2, and MASP-3, although MASP-1 and MASP-3 are translated from the identical gene as splicing variants.

Concerning the alternative splicing variants of the *MASP* genes, there are two additional products; sMAP (small MBL-associated protein) or MAP19 from the *MASP2* gene and MAP44 or MAP-1 from the *MASP1/3* gene. I love the name of sMAP, because it is the name of a popular Japanese musical group. However, since the additional splicing variant from *MASP1/3* gene was found, we do not insist on the name of "sMAP". We would like to suggest MAP19 (also called sMAP) and MAP44. Concerning MAP-1, it is already used as the acronym of methionine aminopeptidase 1 and mannosidase processing 1. To distinguish the alternative splicing variants that lack a serine protease domain from MASPs and also the MAP-1 (methionine aminopeptidase 1 and mannosidase processing 1) described above, we think that MAP19 and MAP44 are better terms that are coincident with the original reports (small letter p and m.w. are important).

Gene name	Protein name
<i>MASP1/3</i>	MASP-1, MASP-3 MAP44 (MAP-1)
<i>MASP2</i>	MASP-2 MAP19 (sMAP)

## 4. Miscellaneous

Collectin kidney 1 (CL-K1), Collectin placenta 1 (CL-P1) and Collectin liver 1 (CL-L1) were reported by Nobutaka Wakamiya, Asahikawa, Japan. He would like to use CL-K1, CL-P1 and CL-L1 as protein names, and *COLEC11*, *COLEC12* and *COLEC10*, as gene names respectively.

## II. Remarks and suggestions regarding a consensus nomenclature for the proteins of the lectin pathway of complement activation

*Steffen Thiel and Jens Christian Jensenius, Aarhus University, Denmark.*

We believe that terminology encompassing some biological meaning is helpful for remembering, understanding and for communicating outside the immediate expert group and for teaching students. Also, it has been a very long tradition, whenever possible, to honour the discoverers of plants, animals or indeed proteins. Additionally, we believe it is of the utmost importance to strive for original and unique names to avoid confusion.

The following presents arguments for the nomenclature of proteins of the pathway of complement activation now most often referred to as the lectin pathway of complement activation, in the following named the lectin pathway. This term is somewhat of a misnomer as ficolins may also initiate the pathway, as these are acetyl-pattern binding, rather than carbohydrate-pattern specific, but for lack of a better term, we accept "lectin pathway".

The first pattern recognition molecule (PRM) that was discovered of this pathway should be named mannan-binding lectin and abbreviated MBL. It was first termed mannan-binding *protein* (MBP) by its discoverers, Kawasaki and Yamashina in 1978 (1), but an 1995 ad-hoc committee in Brighton headed by MW Turner suggested that the abbreviation, MBP was confusing with several other proteins being thus denoted (e.g., myelin basic protein, major basic protein, maltose-binding protein), and that it was quite pertinent to acknowledge the lectin nature of the protein.

The protein should be named mannan-binding lectin, as its discovery and purification were dependent on its calcium-dependent binding to the polysaccharide mannan, which is extracted from baker's yeast. The binding to carbohydrates classified the protein as a lectin, and its biological functions require its binding to patterns of carbohydrates on microorganisms. Other researchers, who entered the field later, renamed the protein "mannose-binding protein" or "mannose-binding lectin". They used the monosaccharide mannose instead of mannan for the purification of the lectin. Importantly the biological function of the protein is mediated through binding to patterns on oligo and polysaccharides rather than to monosaccharides. Examination of binding to a variety of monosaccharides even shows that mannose is not the monosaccharide bound with highest affinity by MBL. Thus the later coined term mannose-binding lectin has often led unsuspecting scientists to believe that this protein has some special selectivity for mannose, and indeed to proceed with experiments suffering from this misunderstanding.

Four other PRMs have later been included in the lectin pathway.

The group of Wakamiya and colleagues reported in 2006 on the protein collectin kidney 1, CL-K1 (in a series of experiments also identifying the collectins CL-P1 and CL-L1)(2). CL-K1 may have a role in the lectin pathway as it was recently reported to bind proteins also bound by the other members of the pathway (see below). The original finding has been followed by only three subsequent papers. CL-K1 has been termed "collectin-11" in the fourth paper devoted to this protein. Somehow the gene was named COLEC11 (with only one "l"). If collectin-11 is to be used instead of CL-K1, other

proteins should likewise be renamed, i.e., MBL should be named collectin-1. There are 12 proteins denoted collectin (collectin-1 to collectin-12), three of which are found only in bovidae. Clearly, this would be a confusing and simplistic terminology, and we strongly encourage the use of the more established and meaningful terms, as indicated herein.

A separate question concerns the three ficolins. In fact, one might argue that they are not proper lectins (as mentioned above) since they will bind acetyl groups whether on sugars or presented otherwise, e.g., on a protein backbone. The name ficolin was used first by Ichijo and colleagues.

We believe in the terminologies arrived at after some confusion with many synonyms. We would argue for keeping the well-known and useful H, L and M terminology, i.e., H-ficolin (a.k.a., Hakata antigen or ficolin-3), L-ficolin (liver expressed, a.k.a., transforming growth factor (TGF)- $\beta$ 1-binding protein, elastin-binding protein, hucolin, ficolin-2 or P35) and M-ficolin (monocyte expressed, a.k.a., P35-related protein or ficolin-1). These terms embody historical and biological meaning.

A number of proteins are found to be associated with the PRMs mentioned above. There seems to be no arguments about the abbreviations MASP-1, MASP-2 and MASP-3. Matsushita and Fujita and colleagues, discoverers of the first MBL-associated serine protease, originally coined "MASP", being an abbreviation of "MBL associated serine proteases". We termed this enzyme MASP-1, when we discovered first MASP-2 and next MASP-3.

For the alternative splice product of the MASP2 gene two names were suggested simultaneously by Fujita and colleagues and us: sMAP (for small MBL-associated protein) and MAP19 (for MBL-associated protein of 19 kDa). While we were also originally happy with sMAP, we dropped this when discovering that this acronym was used for other proteins (e.g., small acidic protein, Smg GDS-associated protein and SmAP protein of *Pyrobaculum aerophilum*). Now that we know that there is indeed also another small MBL and ficolin associated protein (see below), it seems useful to keep the unambiguous term MAP19. If one were to select sMAP one would perhaps have to use the term sMAP-1 and the new protein sMAP-2, which we believe could be confusing (as they arise from the MASP2 and MASP1 genes, respectively).

Continuing along this line of thoughts, we selected the term MAP44 for the alternative, non-enzymatic splice product of the MASP1 gene (three proteins are encoded by this gene, MASP-1, MASP-3 and MAP44), described by us in 2009 (MAP44 again being an unambiguous term). The group of Peter Garred and colleagues, who only a few months later described the same protein, named it MAP-1 (quote, "This protein is named MBL/ficolin-associated protein 1 (MAP-1 corresponding to MASP1 isoform 3)"). We believe the arguments put forward for this acronym to be somewhat weak, the term easily confused with MASP, and on PubMed this term shows up as acronym for quite a few proteins, e.g., "microtubule associated protein 1" or "Modulator of Apoptosis", while MAP44 only shows relevant papers.

## Obituary

### Jürg Tschopp 1951-2011

#### *Reflections on Jürg's Complement period*



Jürg Tschopp, a dear colleague and friend, died on March 22, 2011 at only 59 years old. His heart betrayed him during an Alps' vacation with his family. What a great loss to his wife Erna, his daughter Muriel and his son Dominique, but also to his many friends worldwide and to medical science at large.

Over the past 25 years, Jürg Tschopp, with his team in the department of Biochemistry, the University of Lausanne, Switzerland, has been a leader in the fields of Apoptosis and Inflammation. The deep feeling of loss is reflected in the four Obituaries published recently in *Nature* (*Nature* 472: 296, 2011), *Nature Immunology* (*Nature Immunology* 12: 367, 2011), *Immunity* (*Immunity* 34: 451, 2011) and *The European Journal of Immunology* (*Eur. J. Immunol.* 41: 1189-90, 2011). He was the "discoverer" of the inflammasome, of novel pathways of cell death and regulation of peripheral B cell activity (via BAFF) and, with the collaborative contributions of others, exemplified the rapid translation of basic science into beneficial therapies for patients who presented with mystifying ailments. Jürg epitomized the "outside the box" thinking - actually never perceiving a box - which allowed creative extensions from observations. Importantly, however, he was a biochemist with "innate" awareness of physical and kinetic reality, and as the ultimate experimentalist, tested hypotheses with precision.

Here we would like to reflect on the less well noted yet definitive contributions he made even as a graduate student and postdoctoral fellow during his Complement period. Trained in the laboratory of Jürgen Engel at the University of Basel, one of a handful of experts at the time in the investigation of the classical complement initiator recognition complex, C1, Jürg published several papers in high impact journals, combining electron microscopy, ultracentrifugation sedimentation and enzymatic assays to explore the structure and activation of the C1 complex and its constituents, C1r2s2.



In 1979, he received his PhD in biophysics and moved to the Research Institute of the Scripps Clinic in La Jolla, California, to join as a postdoc the complement group of Hans Müller-Eberhard.

Jürg's interest was drawn to the mysteries of the membrane attack complex of complement and its relationship to membrane pores and the electron microscopic 'complement lesions' on cell membranes and bacteria. At that time, three collaborating teams studied it at the Scripps, the teams of Hans Muller-Eberhard, Eckhard Podack and Alfred Esser. Jürg joined forces with Eckhard Podack in the discovery that complement component C9, in isolation, could polymerize and form structures resembling complement pores. Jürg became



instrumental in helping to fully characterize the process of C9 polymerization and describe how poly C9 is integrated into the membrane attack complex. These breakthrough discoveries were published in a series of high quality papers in high impact journals and have since been enshrined in textbooks of immunology. As Podack says: "For me, Jürg was one of my best postdoctoral fellows and friend, a highly respected and worthy competitor and a brilliant scientist who created a lasting scientific legacy - an ideal we all would like to attain."

At that time, Jürg also collaborated with Bob Ziccardi, also at Scripps, in a fundamental demonstration of the dynamic nature of the native C1 complex and providing definitive data (dissociation constants) demonstrating that the C1 reconstituted from purified C1q, C1r and C1s had biochemical properties of native C1, and was thus a valid complex through which to study activation of the classical complement pathway (an issue for some at the time).

In 1982 Jürg returned to Switzerland and joined the faculty of the University of Lausanne in Epalinges. He engaged in intense research on the cytolytic granules of cytotoxic lymphocytes. While Eckhard Podack and Gunther Dennert discovered Perforin, the pore-forming protein of

NK cells that mediate cytotoxicity by polymerization in a similar manner as C9, Jürg characterized the activity and coined the names of Granzymes, the active pro-apoptotic proteases stored within the cytolytic granules.

Jürg continued with complement-related research till 1994, in parallel to his research on cytolytic lymphocytes and published extensively. In



collaboration with numerous colleagues he kept pursuing insights into the structure and function of the terminal complement proteins and into complement activation. Thus, with Dieter Jenne and Lars French, they identified in serum the complement inhibitory activity of Clusterin (SP40,40) and demonstrated its direct binding to C7, C8 and C9. With Henry Isliker, they showed that cardiolipin, the mitochondrial membrane phospholipid, activates complement through C1 in an antibody-independent manner.

Jürg while rigorous in scientific thought is remembered with a smile on his face and numerous quips to make the moment fun. He was a symbol of friendship and generosity, always ready to share knowhow and reagents, expecting no revenues. Always the thoughtful gentleman and ever pushing the scientific envelope with joy, he is and will be missed.

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