For the first time, these data provide evidence for the existence of spontaneous tumor-specific humoral immune responses against OFA/iLR in a significant proportion of MGUS/AMM patients representing a potential mechanism to control malignant cells by the host immune system. We are currently involved with experiments designing a therapeutic vaccine mediating the induction of both humoral and cellular immunity against OFA/iLR.

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The WHO 2008 classification of Ph-myeloproliferative disorders: statement of the Czech MPD Working Group

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The recently published WHO 2008 criteria for Ph-myeloproliferative disorders (MPD)[1] have, in the opinion of the Czech MPD Working Group, several pitfalls.

James *et al.*[2] and many groups thereafter have suggested that *JAK2*^{V617F} mutation and increased values of the red blood cell parameters (or possibly, increased total red cell mass) suffice for the diagnosis of polycythemia vera (PV). However, the WHO criteria[1] introduce additional 'minor' criteria: (a) histopathology; (b) erythropoietin (Epo) values and (c) endogenous erythroid colony formation (EEC). In *JAK2* mutated cases, one minor criterion is demanded, and in unmutated cases, two of them. The authors[1] advocate fulfilling the minor criteria as they fear of false *JAK2*^{V617F} positivity. However, if allelic discrimination assays are employed, this is extremely unlikely to happen: a discordant result was obtained in 0/261 samples screened by two alternative tests.[3] The minor criteria (b) and (c) may not be met at least in the early stages of PV—at this stage, Epo is quite frequently within the lower normal range and the EEC assay may be unavailable or falsely negative as it is technically rather difficult to perform (in the lab of one of us, EEC growth was demonstrated in only 79% of otherwise typical *JAK2*^{V617F}-positive MPDs; unpublished). In case, when two of the minor criteria (Epo and EEC) are missed, performing the (otherwise unnecessary) biopsy would be mandatory. However, the histological distinction of PV from essential thrombocythemia (ET) on the one hand and from primary myelofibrosis (PMF) on the other is sometimes not easy even for an experienced pathologist. Conversely, two minor criteria (Epo and EEC) may be occassionally met in cases of *JAK2*^{V617F}-negative idiopathic erythrocytosis.[1] One of the major criteria of the WHO 2008 for PV is the presence of *JAK2*^{V617F} or *JAK2* exon 12 mutation.[1] This may lead to substantial overlap and diagnostic confusion of the different entities, that is, true *JAK2*^{V617F}-positive PV and idiopathic erythrocytosis with *JAK2* exon 12 mutation. Moreover, the cases with exon 12 mutation (as the major criterion[1]) cannot be confirmed by histopathology (a minor criterion[1]) because in cases with exon 12 mutation, the picture is far different from the typical PV 'panmyelosis'.[4] We would prefer omitting the minor criteria whatsoever. The risk of 'false' negativity of the minor criteria (lack of both elevated Epo and EEC growth) by far overweights the possibility of an erroneous *JAK2*^{V617F} result. We agree with the authors[1]

that histopathological evaluation should be obligatory when *JAK2*^{V617F}-negative PV is suspected.

The new criteria for essential thrombocythemia use the threshold of 450×10^9/l platelets.[1] This cutoff value is not based on clinical data. It is known that thrombosis may occur at platelet counts above 400×10^9/l[5] and even at counts within the normal range. Therefore, quite reasonably, some of the published diagnostic guidelines use the cutoff equal to the upper normal limit of 400×10^9/l platelets.[6,7] The sole expert reading of histopathology (not even taking the platelet counts into account) is diagnostic for essential thrombocythemia.[5] Therefore, the fourth criterion of the new proposal, that is, exclusion of reactive thrombocytosis in cases without demonstration of *JAK2*^{V617F} or other clonal marker may be excessive. (The same holds true for an analogous formulation in the criteria for PMF).

The first major PMF criterion,[1] histopathology, verbally admits the diagnosis of 'prefibrotic cellular phase' of the disease. However, this stage is practically precluded by the demand to fulfill at least two minor criteria: it has to be understood that three of the four minor criteria given (leukoerythroblastosis, anemia and palpable splenomegaly)[1] are features of more advanced PMF and only extremely rarely occur at diagnosis of the prefibrotic stage of PMF.[8]

Renaming MPD as 'neoplasms' does not seem to be a well-chosen word either. Patients with a relatively benign myeloproliferation, such as essential thrombocythemia, will uselessly have this somewhat frightening word in their medical records.

The Czech MPD Working Group cannot advocate the currently published WHO 2008[1] criteria for routine usage for reasons given above. Morever, these criteria lack the nosologic clarity of the former criteria,[8] which were largely based on histopathology (originally worked out by the Hannover and later Cologne histopathological schools in Germany) and recognized the respective disease entity from its onset until late complications, without any need to change the name of the disease. Thus, we still recommend the WHO 2001 or ECP (European Clinical and Pathological) 2002 criteria[6,8] for usage, with only one correction: we have already agreed that presence of the *JAK2*^{V617F} mutation along with polyglobulia (or possibly, increased total red cell mass) may suffice for diagnosing typical PV, without the need of the biopsy.

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Genomic typing for patient-specific human leukocyte antigen-alleles is an efficient tool for relapse detection of high-risk hematopoietic malignancies after stem cell transplantation from alternative donors

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Partially human leukocyte antigen (HLA)-mismatched allogeneic hematopoietic stem cell transplantation (allo-HSCT) from haploidentical family, unrelated and cord blood donors, has during recent years become an important and feasible therapeutic option for the almost 50% of patients affected by high-risk hematologic malignancies lacking an HLA-identical sibling donor.[1] In these patients, the curative efficacy of allo-HSCT is critically dependent on timely detection of