Indoleamine 2,3-dioxygenase-expressing leukemic dendritic cells impair a leukemia-specific immune response by inducing potent T regulatory cells

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Online Supplementary Table S1. Immunophenotype of immature and mature AML-DC derived from AML blasts of different FAB subtypes. AML blasts give rise to immature and mature AML-DC with comparable immunophenotype, regardless of the FAB classification.

	Immature DC		Mature DC		
	M0/M1	M4/M5	M0/M1	M4/M5	
% HLA-DR	84±6.2	86.5 ± 5	87.6±4.8	91.2 ± 4.2	
% CD86	52.1 ± 13.4	61.1 ± 12.8	74.9 ± 6.9	83.2 ± 4.1	
% CD40	62.7 ± 6.1	$73.4{\pm}2.8$	$86{\pm}5.4$	89.7±2.3	
% CD80	66.3 ± 7.5	71.1±5.8	80.8 ± 7.4	81.9 ± 5.8	
% CD83	21.7±.3	26.7 ± 5.1	41.2 ± 5.6	48.9 ± 3.8	

Online Supplementary Table S2. Expression of *IDO1* mRNA in AML cells of different FAB subtypes. The differentiation of blasts into immature and mature AML-DC results in a significant up-regulation of *IDO1* mRNA, regardless of the FAB classification.

	Mean ± SEM of (<i>ID01/ABL</i>)x1000		
	M0/M1	M4/M5	
Blasts	717.1 ± 863.7	2234.8 ± 3868.3	
Immature DC	22725.5 ± 2095.6	14260.9 ± 4115.1	
Mature DC	185716.5 ± 139964.2	151464.4 ± 109486.7	



Online Supplementary Figure S1. Leukemic origin of DC obtained from AML blasts. Leukemia-specific fusion gene-derived from t(8; 21) is amplified by polymerase chain reaction from cDNA of AML blasts (lane 2), immature AML-DC (lane 3) and mature AML-DC (lane 4). Positive (lane 6) and negative controls (lane 5 and 7) are also illustrated. The presence of the same molecular alteration in AML blasts and DC confirms the leukemic nature of AML blast-derived DC.



Online Supplementary Figure S2. Immunophenotype of immature and mature AML-DC. The high expression of HLA-DR, CD86, CD40 and CD80 and the intermediate expression of CD1a and CD83 confirm the differentiation of AML blasts into AML-DC. The addition of maturation stimuli causes down-regulation of CD1a and up-regulation of CD86, CD40, CD80 and CD83 levels, confirming the maturation of AML-DC.



Online Supplementary Figure S3. Anti-leukemic response is blocked by antibody against HLA-class II. (A) DC are pulsed with tetanus toxin, as control. Proliferation in response to tetanus toxin is inhibited by the presence of both anti-HLA-class I (**P<0.01) and anti-HLA-class II (**P<0.01) antibodies. (B) DC are pulsed with necrotic AML blasts. Proliferation in response to necrotic AML blasts is inhibited only by anti-HLA-class II (*P<0.05).