Patients	<i>JAK2</i> V617F	Age	WBC/mL	Hbg/dL	Hct %	Plt/mL	Duration (months)	Therapy	
PV220705	Hetero	79/F	8400	17.3	56.0	335	0	Phlebotomy+CHT	
PV569	Hetero	28/M	8700	20.8	61.9	358	0	Unknown	
PV583	Hetero	60/M	13100	17.2	52.5	832	8	СНТ	
PV653	Hetero	72/M	8000	18.4	59.2	676	0	Unknown	
PV766	Hetero	67/M	7100	15.2	52.2	493	0	CHT	
PV842	Hetero	51/M	10300	16.8	54.3	573	0	Phlebotomy	
PV870	Hetero	62/F	9300	17.6	53.6	448	0	Phlebotomy	
PV904	Hetero	70/M	10810	18.4	56.8	866	1	CHT	
PV909	Hetero	58/M	7170	16.8	53.6	578	90	Phlebotomy	
PV190705	Hetero	65/F	5800	15.4	44.4	259	134	СНТ	
PV1	Hetero	67/M	13700	12.5	39.6	859	48	HU,Bus,Anagrel,Phlebotomy	
PV3	Hetero	42/F	11000	15.8	46.3	560	60	HU or Phlebotomy	
PV4	Hetero	58/M	7800	15.2	49.2	364	48	Phlebotomy	
PV538	Homo	77/F	16600	13.9	46.0	306	65	CHT	
PV546	Homo	74/F	9300	16.5	56.8	425	2	Phlebotomy + CHT	
PV5	Homo	60/M	21900	14.4	47.3	862	180	HU and Phlebotomy	

Table S1. Clinical data on the PV patients included in cell/biochemical studies

Abbreviations: Anagrel=anagrelide, BUS=busulfan, CHT=not otherwise specified chemotherapy, Hb=hemoglobin, Hct= hematocrit, HU=hydroxyurea,Plt= Platelets, WBC=white blood cells; 0=new or recent diagnosis.

Table S2. Frequency of erythroid (CD235^{pos}) and non-erythroid (CD45^{pos}) cells generated in cultures of MNC from ND

or PV	patients in	the presence (+	-) or not (-)) of DXM, as indicated.
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	CD45 ^{pos}			CD235 ^{pos}		
		-DXM	+DXM	-DXM	+DXM	
ND	Day 11	38.6±17.0	8.2±1.3 *	57.4±16.0	87.2±4.6 *	
(n=5)	Day 13	37.8±21.0	7.0±2.6 *	55.7±19.0	86.0±7.1 *	
Heterozygous	Day 11	8.0±10.0 [§]	1.6±0.9 [§]	90.1±11.0 [§]	96.6±2.6 [§]	
(n=10)	Day 13	12.1±17.0 [§]	1.3±1.0 [§]	85.0±18.0 [§]	95.6±2.7 [§]	
Homozygous	Day 11	4.0±2.8 [§]	2.1±1.8 [§]	93.6±3.0 [§]	95.3±3.2 [§]	
(n=3)	Day 13	4.3±3.7 [§]	0.8±1.1 [§]	90.6±6.0 [§]	95.0±2.4	

Values statistically different (p<.05) from those observed in the corresponding group from ND (§) or from the parallel cultures without DXM (*) are indicated. Representative FACS analyses are presented in Figure S2.



Figure S1. Diagram of the human *GR* locus and schematic representation of examples of alternative splicing sites (A) and GR isoforms (B) expressed in the population. The presence of the A3669G polymorphism gives rise to an alternative splicing at exon 9 which results in a mRNA encoding the dominant negative β isoform. Red arrows indicate the positions of the oligos used for RT-PCR determination of GR α and β isoform expression (from Sanchez-Vega B et al, Mol Cancer Ther. 2006;5:3062-3070.)



Figure S2. Half-life of A3669G positive (circles) and A3669G negative (squares) GR β mRNA in COS-1 cells transfected with the expression plasmid pCMVGR β (squares), or vector pCM β mut, which contains a mutated "ATTTA motif" (to GTTTA") in the 3' UTR (circles). At 3 and 6 h after actinomycin-D treatment, the cells transfected with pCMVGR β show a significantly (p < 0.05) larger decrease in GR β mRNA levels than the cells transfected with the mutated vector. Data are expressed as mean (n = 3) ± SEM. Published by permission from Dr. Derijk and the editor of Journal of Rheumatology (see Derijk et al J Rheumatol 2001; 28:2383 for further details).



Figure S3. Representative FACS analysis for CD45 and CD235a expression of cells obtained after 13 days in cultures of MNC from ND and from patients with PV, as indicated. The cultures, the same as represented in Figure 2B, were stimulated with GFs with or without DXM. The mean (\pm SD) frequencies of CD45^{pos} and CD235a^{pos} cells observed in multiple experiments for each donor type are summarized in Table SII. Since the frequency of Propidium Iodide^{pos} cells was comparable (~10-15%) in all cultures analyzed, the propidium iodide (PI) staining and FSC and SSC analyses of only one representative experiment are presented, as reference. Cultured cells were divided by FSC/SSC analyses into small lymphocyte-sized cells (purple dots) and large erythroblast-size cells (blue dots). This color code is retained in the CD45/CD235a analyses to allow comparison of size distribution of cells obtained in the different cultures.



Figure S4. EPO alone induces proliferation and not maturation of EBs generated *ex-vivo* from PV patients. A) Representative FACS analyses for CD36 and CD235a expression of EBs obtained from ND (left panels) and PV patients (right panels) under HEMA conditions (GFs+DXM) and stimulated for 96 hrs with EPO (10 U/mL). B) Mean (\pm SD) fold increase (FI) in cell number and in the frequency of mature (CD36^{neg}CD235a^{high}) EBs observed in cultures of 3 separate ND and PV patients after 4 days with EPO alone.



Figure S5. JAK2V617F expression, as percent of wild type allele expression, in erythroid cells obtained in vitro with (+) or without (-) DXM from PV MNC heterozygous or homozygous for the JAK2V617F mutation. Results are compared with the percent of JAK2V617F expressed by granulocytes (PMN) present in the blood of the patients at the time of collection for this study.