

fluorescent intensity (MFI) of intracellular  $\kappa$ -B kinase  $\beta$  (IKK $\beta$ ), as a downstream activation product of the NF $\kappa$ B pathway. MACE events were recorded at follow-up.

**Results** We recruited 96 patients (average age 61.5 years $\pm$ 13.3; 64.6% male). Patients were followed-up for a median of 187 days (112–222 days). MACE events occurred in 14 patients (14.6%). Using logistic regression analysis, increased total monocyte count ( $p<0.032$ ), Mon2 counts ( $p<0.047$ ) and Mon3 IKK $\beta$  ( $p<0.013$ ) were significantly predictive of MACE at follow-up (Abstract 125 table 1). Mon2 counts were an independent predictor of MACE after adjusting for age and sex.

**Abstract 125 Table 1 Monocyte subpopulations and IKK $\beta$  predict MACE**

	Monocytes	OR (95% CIs)	p Value
Phenotypic characterisation and enumeration	Total Mon	1.002 (1 to 1.004)	0.032
	Mon1	1.001 (0.998 to 1.003)	0.111
	Mon2	1.008 (1.003 to 1.013)	0.047
	Mon3	1.01 (0.999 to 1.022)	0.388
Functional assessment	IKK $\beta$ Mon1	0.982 (0.944 to 1.022)	0.388
	IKK $\beta$ Mon2	0.983 (0.948 to 1.019)	0.373
	IKK $\beta$ Mon3	1.038 (0.993 to 1.086)	0.013

**Conclusion** Increased total monocyte and Mon2 counts in the first 24 h post infarction is predictive of MACE in STEMI patients. Mon3, despite an assumed role in reparation and fibroblast deposition, are also predictive of MACE. Monocytes remained functionally active throughout the acute and healing phases, and thus may have prognostic implications.

## 126 DYNAMICS OF THE THREE HUMAN MONOCYTE SUBSETS OVER 30 DAYS IN ST-ELEVATION MYOCARDIAL INFARCTION

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**Introduction** Monocytes are intimately involved in the pathophysiology of myocardial infarction (MI), with different subsets thought to have distinct roles in cardiac repair. We have described that human monocytes can be divided phenotypically into 3 subsets by their surface expression of CD14, CD16 and CCR2: CD14+CD16–CCR2+ (Mon1, “classical” monocytes), CD14+CD16+CCR2+ (Mon2) and CD14lowCD16+CCR2– (Mon3). Having observed a threefold increase in Mon2 at admission with STEMI, we aimed to establish the dynamics in total monocyte count, subset count and relative proportions of the subsets in the 30 days following ST elevation myocardial infarction (STEMI) treated by primary percutaneous coronary intervention (PPCI).

**Methods** Monocyte subsets were measured by flow cytometry in 50 patients (57.5 $\pm$ 11.7 years, 86% male) with STEMI at 4 time points: within 24 h after PPCI, day 3, day 7 and day 30 after MI onset. All patients underwent PPCI. Exclusion criteria comprised factors known to affect monocyte count.

**Results** The peak number of total monocytes, Mon1 and Mon2 occurred on day 1, with comparable values on day 3. The total monocyte count and Mon1 reduced significantly by day 30, to levels seen in stable coronary artery disease. Mon2 count reduced significantly earlier, by day 7. No changes were seen in Mon3 count. Mon2 predominates over Mon3 on day 1, with the reverse pattern seen at day 30, where the Mon2 proportion had reduced significantly and the Mon3 proportion had increased significantly.

**Conclusions** We observed prominent differences in the dynamics of monocyte subsets, particularly the minor subsets (Mon2 and Mon3) which have been suggested to play distinct roles in myocardial reparative processes. The dramatic increase in number of Mon2 after STEMI followed by significant reduction by day suggests a specific role in the acute phase of MI. These novel findings may contribute to further understanding the pathophysiology of the recovery processes following acute MI.

**Abstract 126 Table 1**

	Day 1	Day 3	Day 7	Day 30	ANOVA p value
Total monocytes (per $\mu$ l)	994 $\pm$ 66.2	967 $\pm$ 71.0	861 $\pm$ 70.3	670 $\pm$ 32.4* $\dagger$	<0.0001
Monocyte subsets					
Mon1	810 $\pm$ 57.7	785 $\pm$ 58.5	712 $\pm$ 62.9	557 $\pm$ 31.3* $\dagger$	<0.0001
Mon2	108 $\pm$ 16.0	105 $\pm$ 15.9	73.6 $\pm$ 13.1 $\dagger$	45.3 $\pm$ 5.61* $\dagger$	<0.0001
Mon3	72.2 $\pm$ 7.07	77.1 $\pm$ 10.1	75.1 $\pm$ 5.71	67.3 $\pm$ 4.31	0.55
Relative proportions					
Mon1 %	81.4 $\pm$ 1.61	81.3 $\pm$ 1.60	82.1 $\pm$ 1.55	82.4 $\pm$ 1.33	0.81
Mon2 %	10.9 $\pm$ 1.36	10.5 $\pm$ 1.24	8.58 $\pm$ 1.37	7.36 $\pm$ 1.05* $\dagger$	0.002
Mon3 %	7.61 $\pm$ 0.82	8.23 $\pm$ 0.81	9.37 $\pm$ 0.68	10.3 $\pm$ 0.64* $\dagger$	0.001

\* $p<0.05$  vs day 1.

$\dagger p<0.05$  vs day 3.

## 127 DEMONSTRATION OF INTRACORONARY MICROPARTICLE EXPRESSION AND THEIR ASSOCIATION WITH ACTIVATED PLATELET MONOCYTE AGGREGATE IN HUMAN ST ELEVATION MYOCARDIAL INFARCTION

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**Background** Platelets play a central role in the pathophysiology of acute coronary syndrome (ACS). During ACS activated platelets express p-selectin which binds to the p-selectin glycoprotein ligand on the monocyte, forming platelet monocyte aggregates (PMA). PMA expression is a sensitive marker of platelet activation. A microparticle (MP) is a submicron membrane vesicle derived from virtually any cell during various biological processes (cell activation, differentiation or apoptosis). Recent experimental and clinical data point towards a causal effect of MP and mainly platelet derived MP [PMP] in the pathogenesis and disease progression of atherosclerosis and coronary artery disease.

**Aim** To investigate the relationship between levels of intracoronary MP and PMA in ST elevation ACS.

**Methods** Patients with ST elevation ACS who underwent primary percutaneous coronary angioplasty were recruited. Blood samples for PMA, MP levels and soluble marker of platelet activation (p-selectin) were collected from the infarct related coronary artery. PMA and MP levels were estimated using fluorescent monoclonal antibodies and flow cytometry. CD61+CD14+CD62P+ events are PMA expressing p-Selectin [activated PMA] and CD61+CD14+CD142+ events are PMA expressing tissue factor [TF+ PMA]. Total MPs were identified as AnV+ MP and PMP as AnV+CD42+/or AnV+CD42+CD62+ MP. Endothelial derived MPs (EMP) were identified as AnV+CD105+CD42– events and/or AnV+CD62E+CD42– events. ELISA was used for p-selectin measurement.

**Results** The mean total AnV+ MP was 3 661 000/ml of plasma. PMP was statistically higher than EMP  $p=0.01$  (mean PMP (SD) 879 986/ml (1 166 000), mean EMP (SD) 89 099/ml (38 867). There was a strongly positive correlation between total Ann V+ MP with