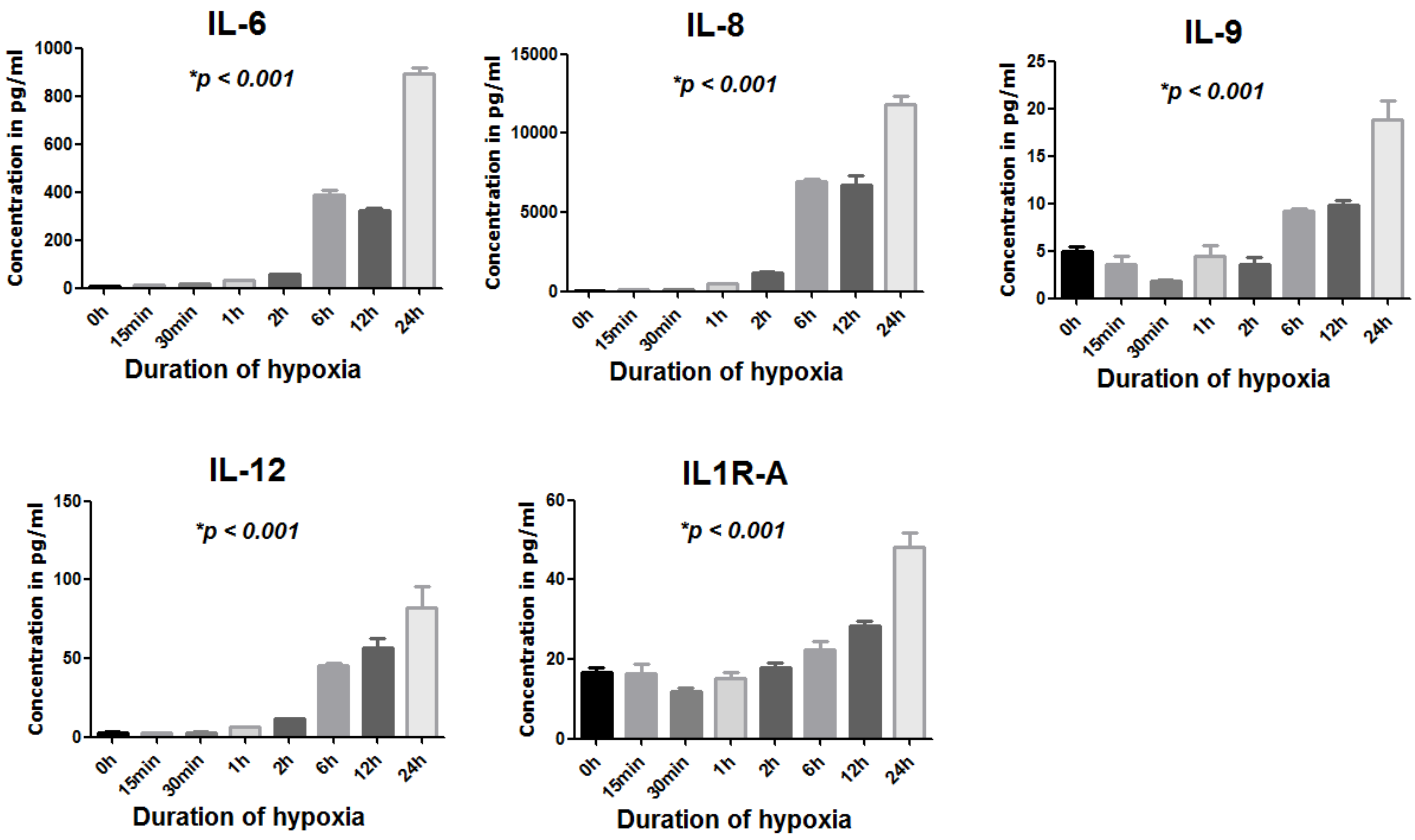
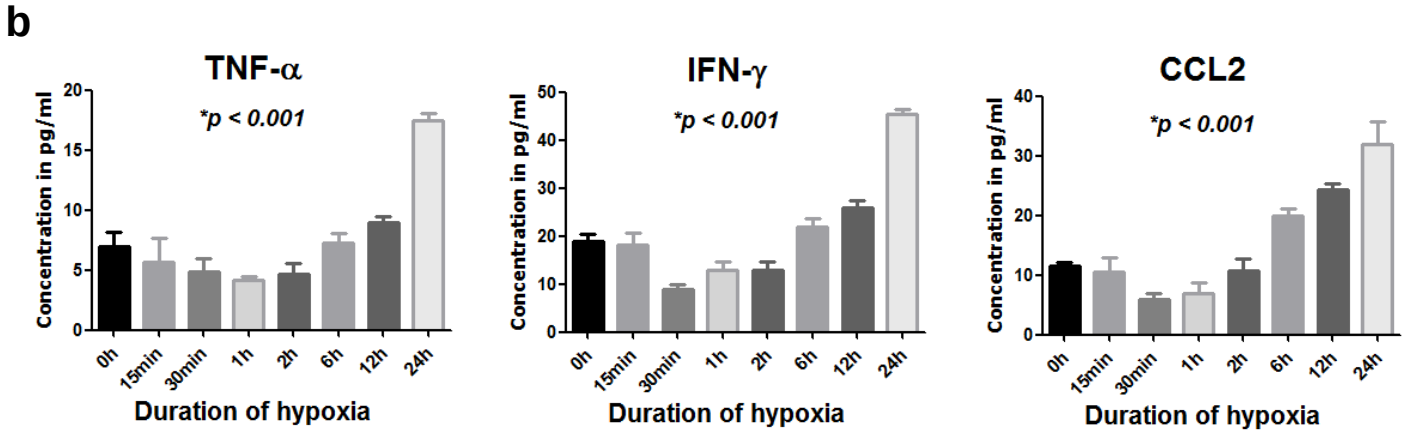
Supplementary figure 1. Secretion of pro-inflammatory cytokines and growth factors from hypoxic hSC. Multiplex ELISA was used to quantify the amounts of a large panel of interleukins (IL, a), inflammatory mediators (b) and growth factors (c) in the supernatants of hypoxic hSC (for the complete list of the analyzed cytokines, please refer to the supplementary methods). The concentrations were expressed in pg/ml. Experiments were repeated three times. The *p values correspond to the one-way ANOVA p values.

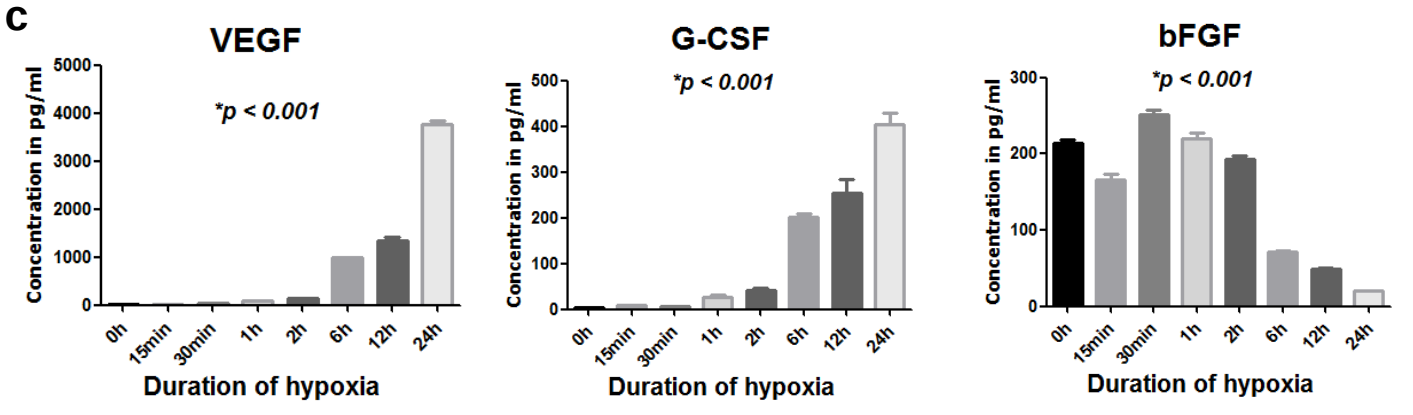
Supplementary figure 2. Impact of hypoxia upon the secretion of platelet-derived growth factor (PDGF), IL-1beta, IL-4, IL-10, IL-13 and IL-17 from hSC. The amounts of these cytokines in hSC supernatants demonstrated a significant variation between normoxic and hypoxic conditions, but their levels constantly remained below 10pg/ml, thereby potentially limiting their biological relevance. Experiments were repeated three times. The *p values correspond to the one-way ANOVA p values.

Supplementary figure 3. The impact of interleukin-6 and interleukin-1β on hSC activation by hypoxia and PCC. (a) Hypoxic hSC were supplied with specific blocking antibodies against interleukin-6 (anti-IL-6) or interleukin-1β (anti-IL-1β) or with nonimmunized mouse IgG1 antibodies in their growth media and compared for the intracellular amouts of GFAP (G), Nestin (N) and Vimentin (V) at 24 hours of hypoxia exposure via immunoblotting. Blockade of IL-1β in hypoxic hSC supernatants did not influence intracellular G, N or V, but there was a tendency towards decreased G and N after IL-6-blockade, though not significant. Unpaired t-test. Experiments were repeated three times. (b) hSC were treated with the supernatants/SN of the PCC lines SU86.86 or T3M4 for 24 hours and compared to control hSC (Ctrl., grown in their serum-free growth medium supplied with serum-free PCC medium) for the phosphorylation status of the IL-6 downstream mediator STAT3 via immunoblotting (left), densitometry of the blot membranes (middle) and

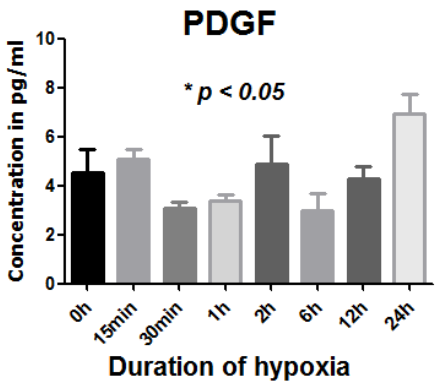
via double-immunolabeling against phospho-STAT3 (pSTAT3) and with DAPI. Experiments were repeated three times. (c) However, addition of anti-IL-6 – but not of anti-IL-1β – significantly attenuated N expression (when added to the supernatants of the SU86.86 PCC) or *both* G and N expression in hSC (when added to the supernatants of the T3M4 PCC) after 48 hours of SN treatment. Ga: GAPDH. Experiments were performed in triplicates. Results were expressed as % of loading control (GAPDH). Unpaired t-test.

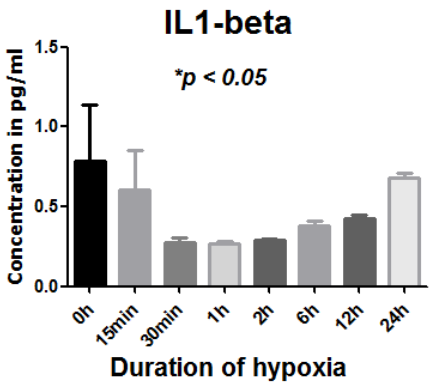

a
IL-6
*p < 0.001
Concentration in pg/ml
1000
800
600
400
200
0
0h
15min
30min
1h
2h
6h
12h
24h
Duration of hypoxia
IL-8
*p < 0.001
Concentration in pg/ml
15000
10000
5000
0
Duration of hypoxia
IL-9
*p < 0.001
Concentration in pg/ml
25
20
15
10
5
0
Duration of hypoxia
IL-12
*p < 0.001
Concentration in pg/ml
150
100
50
0
Duration of hypoxia
IL1R-A
*p < 0.001
Concentration in pg/ml
60
40
20
0
Duration of hypoxia

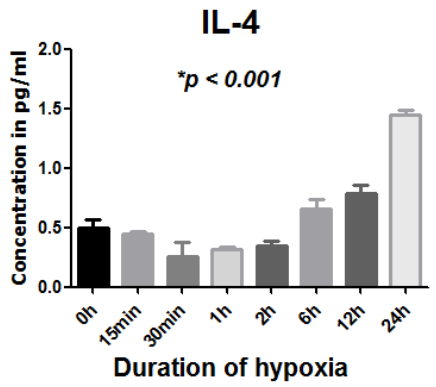

b
TNF-α
*p < 0.001
Concentration in pg/ml
20
15
10
5
0
0h
15min
30min
1h
2h
6h
12h
24h
Duration of hypoxia
IFN-γ
*p < 0.001
Concentration in pg/ml
50
40
30
20
10
0
Duration of hypoxia
CCL2
*p < 0.001
Concentration in pg/ml
40
30
20
10
0
Duration of hypoxia

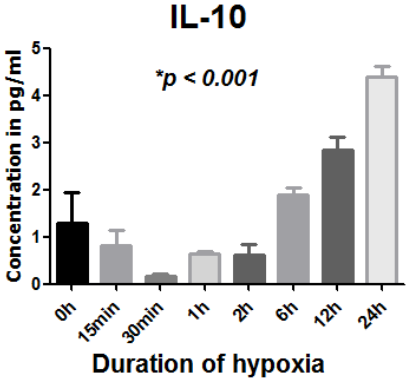

c
VEGF
*p < 0.001
Concentration in pg/ml
5000
4000
3000
2000
1000
0
0h
15min
30min
1h
2h
6h
12h
24h
Duration of hypoxia
G-CSF
*p < 0.001
Concentration in pg/ml
500
400
300
200
100
0
Duration of hypoxia
bFGF
*p < 0.001
Concentration in pg/ml
300
200
100
0
Duration of hypoxia

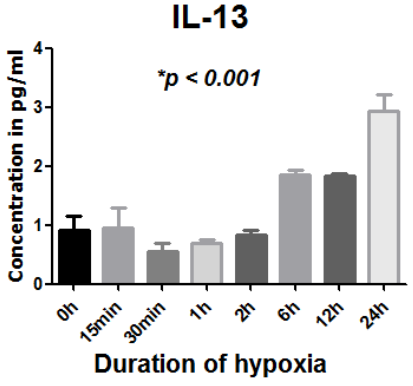
Supplementary Figure 2, Demir et al.

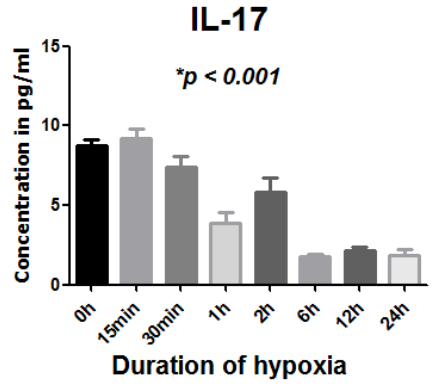












Supplementary Figure 3, Demir et al.