30 Upregulation of an IGF-1 splice variant in active muscle
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We have cloned the cDNA of a splice variant of IGF-1
(MGF) produced by active muscle. A 52bp insert at exon 5 dis-tinguishes it from the liver isoform L.IGF-1. MGF appears to act in an
autocrine manner which differentiates it from the systemic L.IGF-1.
Previously, both MGF and L.IGF-1 were shown to be upregulated
in skeletal muscle after 4 days of stretch and stimulation. MGF and
L.IGF-1 mRNA was quantified over a 7-day period using plaster
cast immobilization of the lower limb of young Sprague-Dawley
rats, combined with 1h of electrical stimulation of the sciatic nerve
at 30Hz. To our knowledge, we are the first to show expression of
MGF mRNA localized in the peripheral nuclei of stretched muscle
by in situ hybridization. Preliminary results showed an increase in
MGF mRNA at day 5 compared to day 1 and confirmed by real-
time PCR which showed a 62% increase of MGF in the experimental
leg compared to the contralateral at day 5 and even greater increase
if compared to normal muscle. L.IGF-1 showed only 17%
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of both L.IGF-1 and MGF transcripts.

We hypothesise that carbon monoxide (CO) induced cardiac dys-
function is associated with ROS production in an
ischaemia/reperfusion (I/R)-like injury. Isolated rat hearts were used
to investigate the effects of CO on cardiac function. Hearts were
exposed to CO (30 min) under normoxic conditions followed by a
90 min CO-free period. In hearts treated with 0.01 and 0.05% CO
heart rate (HR) decreased to ca. 85 ± 10% (mean±SE) compared to
to controls (0% CO). The 0.01% CO group showed signs of contrac-
tile recovery during the post-CO period, whereas the 0.05% CO
group showed no recovery. Coronary flow (CF) decreased signifi-
cantly (P<0.05) by the end of the post-CO period, to 93 ± 0.7% and
87 ± 3% of controls with 0.01 and 0.05% CO, respectively. Activities
of LDH, CK and troponin I were elevated in perfusate from treated
hearts. To investigate the likely role of ROS hearts were perfused in
the presence of two antioxidants (Trolox C and ascorbate). These
eliminated the decrease in CF following exposure to 0.05% CO
(30 min) relative to controls. Whereas the decline in HR was
augmented with antioxidant treatment to 57 ± 14%. These findings
suggest some oxidative role in regulating contractile function that
cannot be attributed solely to cytochrome c oxidase inhibition since
normoxic conditions were used. ROS may be produced during the
(hypoxic) CO period and are responsible for a decline in coronary
function following CO exposure.

31 Effects of multiple hyperbaric oxygen treatments on blood
function
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Hyperbaric oxygen therapy (HBOT) is used to treat chronic tissue
injuries, but may increase the risk of oxidative damage. The blood
function of 31 patients (mean age 61, 20 males, 11 females) referred
for HBOT was followed. 24 patients completed 20 treatments.
Analysis of temporal trends in data from all patients suggests no sta-
tistically significant changes (P>0.05 by ANOVA or
Kruskall-Willis) in total white/red/platelet counts, total haemoglo-in, HCT, MCV, and plasma protein, although lymphocyte counts
increased from 1.3 ± 0.1 to 1.8 ± 0.2 (mean±SE, × 10^9 cells L^−1,
P>0.05) by the end of the 20th treatment. Platelet protein content
increased by 23% and arachidonic acid-dependent platelet aggrega-
tion increased by 24%. Collagen-dependent platelet aggregation was
unaffected. Lactate ratios in platelets fell marginally (by 15%).
Blood glucose improved in females (21% increase) but not males
(17% decrease). Blood lactate decreased significantly (P<0.02, paired t-test) from 3.2±0.2 to 2.5±0.1 mmol L^−1 (mean±SE) by the
end of the study, with values reduced in all patients after the first
administration. No changes in plasma FRAP (ANOVA, P>0.05) were
observed.

32 Carbon monoxide toxicity in the heart: evidence for
ischaemia/reperfusion like injury
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In resting white muscle glycolytic activity is low, but during work the flux through glycolysis must, within
seconds, be increased several hundredfold. PFK is regarded
as the key regulatory enzyme of glycolysis, but how its activity
is controlled in muscle is not fully understood.

We wanted to study a physiological form of muscle work
in intact and otherwise not stressed animals. To this end
we use frogs (Rana temporaria) because they sit still for
hours, yet are stimulated to swim vigorously when transferred
into water (swim reflex). The frogs swim between 1s and 5min
(Ex.1) when their gastrocnemius muscles were tested for
glycolytic intermediates and effectors of PFK. Some frogs,
fatigued by 5min swimming, were allowed to rest for 2h,
and then induced to swim again (Ex.2). All metabolites
responded similarly to Ex.1 and Ex.2, except fructose
2,6-bisphosphate (F2,6P2), a most potent activator of muscle
PFK. The content of F2,6P2 was increased more than 10-fold
after just 1s of Ex.1, it remained at a plateau for about 30s
of Ex.1, then decreased to pre-exercise levels, and
virtually disappeared during recovery. Surprisingly, F2,6P2
did not increase during Ex.2. Possible mechanisms for this
strikingly different response to work of rested and pre-
exercised muscle will be discussed.