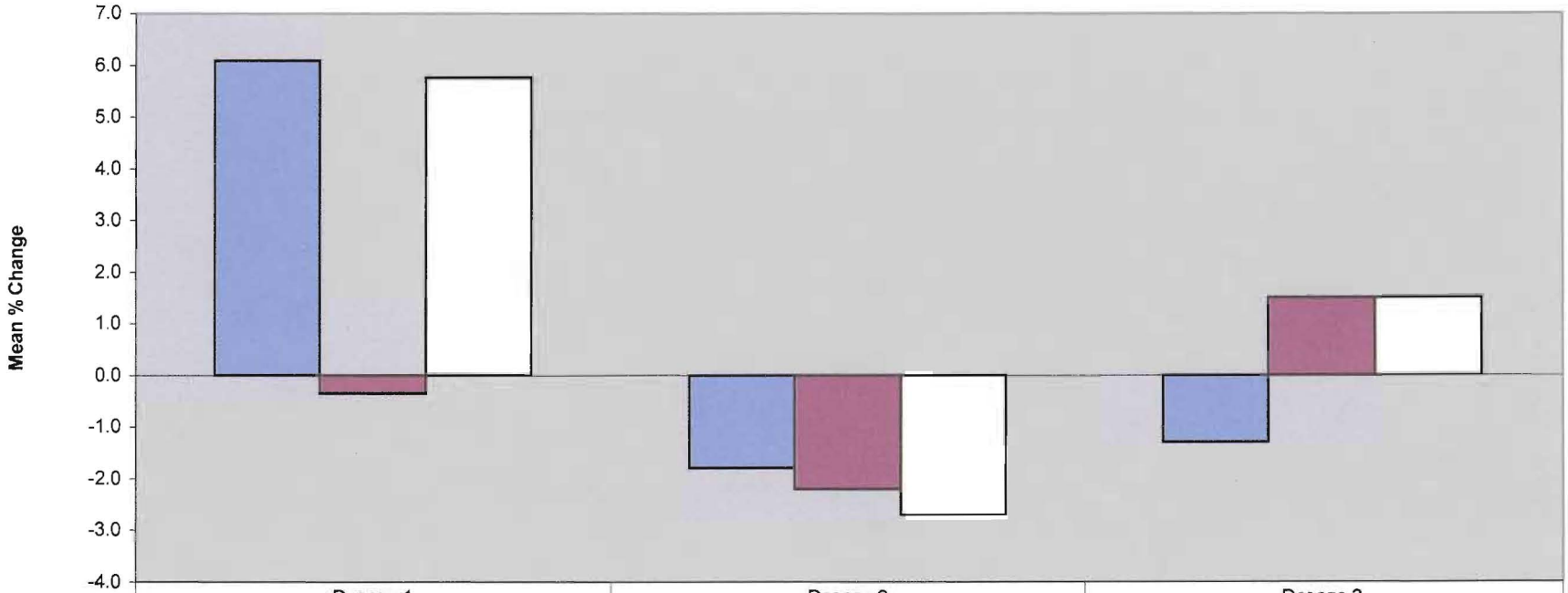
TABLE VI: GAS ANALYSES (A)

* = p< 0.05; % Δ = Relative Change; NS = Not Significant (p> 0.05)

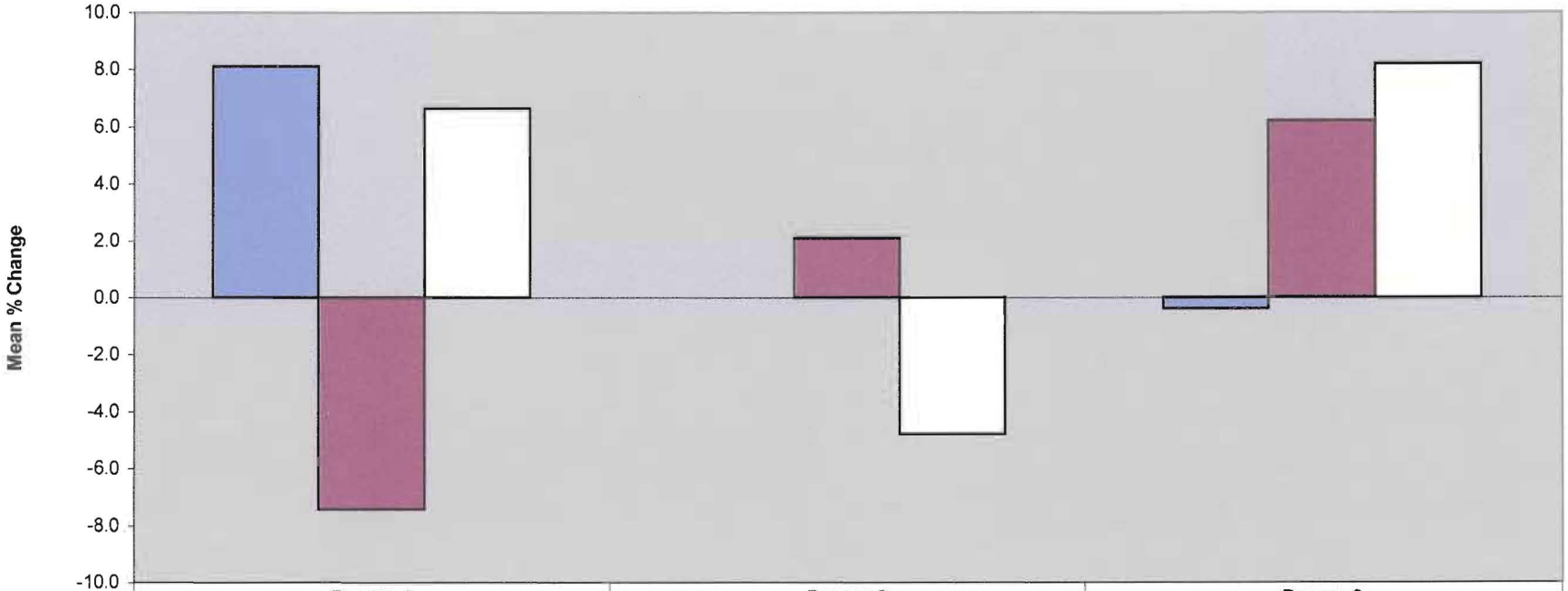
VARIABLES			PL: PLACEBO (N=10)					CF: CELLFOOD (N=10)					SW: SWITCH (N=10)					SIGNIFICANCE		
GAS VALUES	Cycle	UNITS	PRE-TEST. X ± SD		POST-TEST X ± SD		%Δ	PRE-TEST. X ± SD		POST-TEST X ± SD		%Δ	PRE-TEST. X ± SD		POST-TEST X ± SD		%Δ	PL vs CF	PL vs SW	CF vs SW
RER	1		1.2	0.1	1.2	0.1	**6.1**	1.1	0.0	1.1	0.1	**-0.4**	1.1	0.1	1.2	0.1	**5.8**			
RER	2		1.1	0.1	1.1	0.1	**-1.8**	1.2	0.1	1.1	0.1	**-2.2**	1.1	0.0	1.1	0.1	**-2.7**		NS	
RER	3		1.1	0.0	1.1	0.1	**-1.3**	1.1	0.1	1.2	0.1	**1.5**	1.2	0.1	1.2	0.1	**1.5**			
$VC0_2$	1	l/min	3559.2	633.5	3847.6	753.4	**8.1**	3687.2	825.5	3413.4	758.5	**-7.4**	3635.5	776.0	3876.4	947.0	**6.6**			
$VC0_2$	2	l/min	3635.5	776.0	3634.2	648.7	**0.0**	3559.2	633.5	3633.9	776.4	**2.1**	3687.2	825.5	3509.5	950.0	**-4.8**		NS	
$VC0_2$	3	l/min	3687.2	825.5	3671.3	982.2	**-0.4**	3635.5	776.0	3859.4	695.7	**6.2**	3559.2	633.5	3852.0	798.2	**8.2**			
$VE/VC0_2$	1	L	28.0	16.1	27.6	1.8	**-1.6**	28.8	2.8	27.7	2.8	**-3.7**	29.4	2.1	28.2	2.5	**-4.3**			
$VE/VC0_2$	2	L	29.4	2.1	29.1	2.1	**-1.2**	28.0	16.1	28.8	1.5	**2.7**	28.8	2.8	28.3	2.9	**-1.6**		NS	
$VE/VC0_2$	3	L	28.8	2.8	29.4	3.4	**2.1**	29.4	2.1	28.8	2.8	**-2.0**	28.0	16.1	27.6	2.0	**-1.3**			
$etCO_2$	1	mmHg	39.1	2.3	41.1	3.1	**5.1**	39.8	5.3	39.2	3.2	**-1.5**	37.7	2.1	39.3	4.2	**4.2**			
$etCO_2$	2	mmHg	37.7	2.1	38.6	2.8	**2.4**	39.1	2.3	38.3	2.3	**-2.0**	39.8	5.3	39.3	5.0	**-1.3**		NS	
$etCO_2$	3	mmHg	39.8	5.3	38.8	3.9	**-2.5**	37.7	2.1	38.9	3.6	**3.2**	39.1	2.3	39.8	3.4	**1.8**			
VE/VO_2	1	L	32.3	2.8	33.6	3.5	**4.2**	32.4	2.8	31.0	3.4	**-4.0 ***	33.6	1.6	34.0	2.4	**1.3**			
VE/VO_2	2	L	33.6	1.6	32.5	2.9	**-3.1**	32.3	2.8	32.3	3.2	**0.2**	32.4	2.8	31.0	2.4	**-4.5 ***		NS	
VE/VO_2	3	L	32.4	2.8	32.7	4.6	**0.7**	33.6	1.6	33.4	2.8	**-0.5**	32.3	2.8	32.2	2.8	**-0.1**			
etO_2	1	mmHg	96.5	1.9	96.5	2.5	**0.0**	96.4	2.3	94.7	2.8	**-1.8**	97.2	1.5	96.6	2.1	**-0.6**			
etO_2	2	mmHg	97.2	1.5	95.7	2.3	**-1.5**	96.5	1.9	95.7	2.6	**-0.8**	96.4	2.3	94.6	2.5	**-1.9**		NS	
etO_2	3	mmHg	96.4	2.3	95.8	4.0	**-0.6**	97.2	1.5	96.1	3.1	**-1.1**	96.5	1.9	96.3	2.3	**-0.2**			

FIGURE 4.10: RESPIRATORY EXCHANGE RATIO



	Dosage 1	Dosage 2	Dosage 3
Placebo	6.1	-1.8	-1.3
Cellfood	-0.4	-2.2	1.5
Switch	5.8	-2.7	1.5

FIGURE 4.11: $VC0_2$



	Dosage 1	Dosage 2	Dosage 3
Placebo	8.1	0.0	-0.4
Cellfood	-7.4	2.1	6.2
Switch	6.6	-4.8	8.2

Cycle 3 (High Dosage)

During this cycle both the Cellfood® and Switch™ showed an increases of 1.5% in RER. Cellfood® showed an increase of 6.2% in $VC0_2$ while Switch™ showed an increase of 8.2%. Placebo showed a decrease of 1% in RER while the $VC0_2$ decreased with 0.4%. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups.

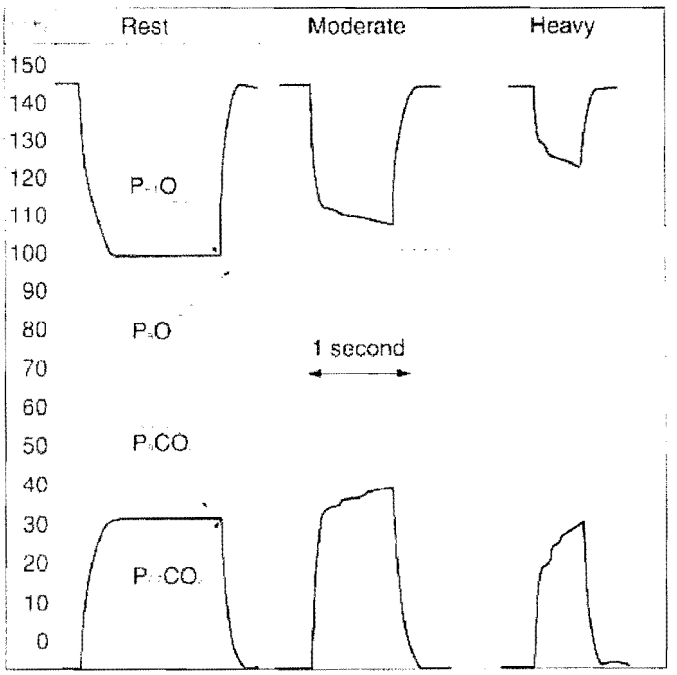
4.10.2 Breathing Equivalent for $C0_2$ (Figure 4.12) and End Tidal Partial Carbon Dioxide Pressure (Figure 4.13) (Table VI A)

Ventilatory equivalents are measures of breathing efficiency, which relate instantaneous minute ventilation to the metabolic rate of oxygen uptake or carbon dioxide output. Ventilatory equivalents are secondary variables derived as the ratios of instantaneous minute ventilation to oxygen uptake ($VE/V0_2$) or carbon dioxide output ($VE/VC0_2$). Being ratios of two flows, ventilatory equivalents have no units.

Resting ventilatory equivalents are variable, but generally 30-60. Ventilatory equivalents fall steadily during the early stages of incremental exercise.$VE/VC0_2$ does not begin to increase until VE becomes dissociated from $VC0_2$, i.e. when buffering mechanisms can no longer prevent a fall in blood pH and VE responds to carotid body stimulation. As long as the RER is less than 1.0, $VE/V0_2$ will be less than$VE/VC0_2$. Of particular importance are the plateau values that are on average 25 for $VE/V0_2$ and 28 for $VE/VC0_2$ for younger normal subjects. With advancing age, as the physiological dead space in the lung increases, the plateau values of the ventilatory equivalents are higher, e.g., 30 for $VE/V0_2$ and 33 for $VE/VC0_2$ (Cooper and Storer, 2001)

End tidal gas tensions are the partial pressures of oxygen and carbon dioxide observed at the end of each exhalation. The last gas exhaled from the lung is assumed to come from the alveolar compartment. Therefore, in the ideal lung, the end-tidal gas tensions would reflect the alveolar partial pressure of these gasses. The normal partial pressure profiles of exhaled oxygen and carbon dioxide are shown in accompanying sketch. The two profiles resemble “mirror images” of

each other and the relative magnitudes of the changes they reflect depend on the RER. The end-tidal partial pressures are often described as plateaus but in reality they are slopes. Towards the end of exhalation, the oxygen tension actually continues to decrease slowly whereas the carbon dioxide tension increases slowly. These changes, which are subtle at rest, represent the continuing gas exchange between the blood and the alveolar gas. During exercise, as the metabolic rate increases, these alveolar slopes become steeper (Cooper and Storer, 2001).

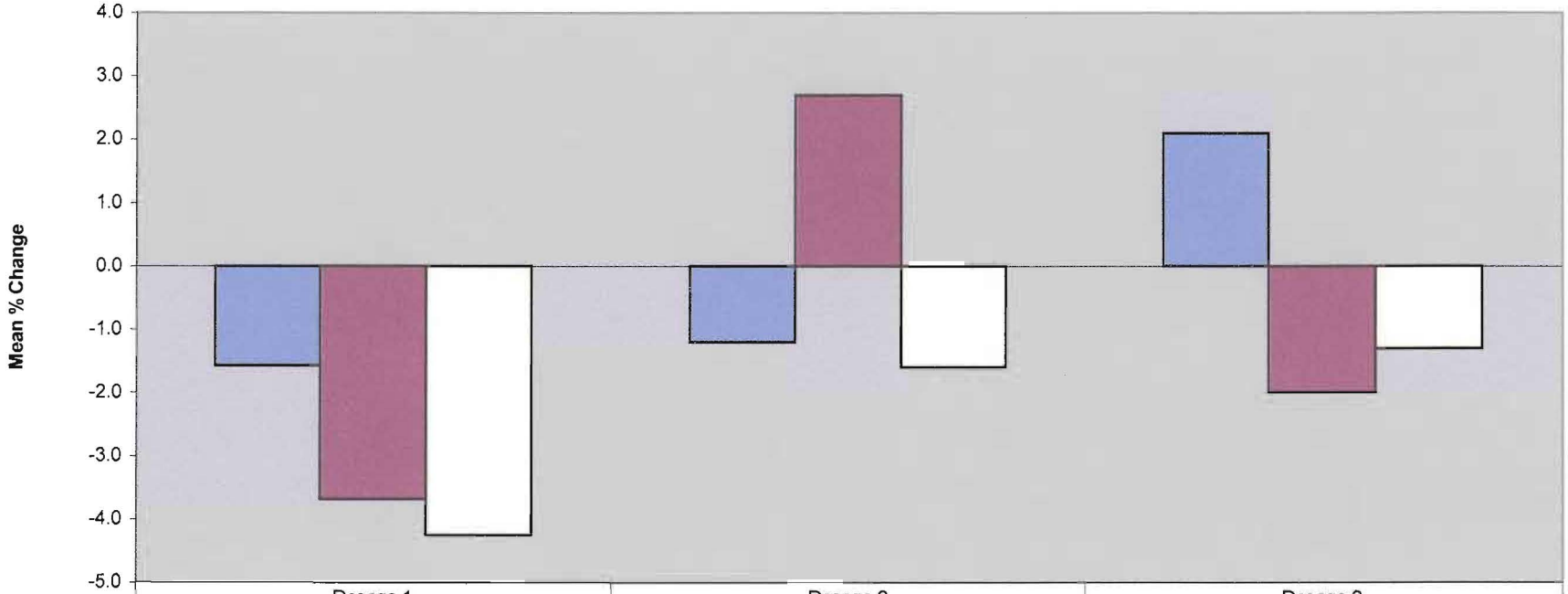


Profiles of exhaled oxygen and carbon dioxide at rest and during moderate or high-intensity exercise (Cooper and Storer, 2001)

Cycle 1 (Low Dosage)

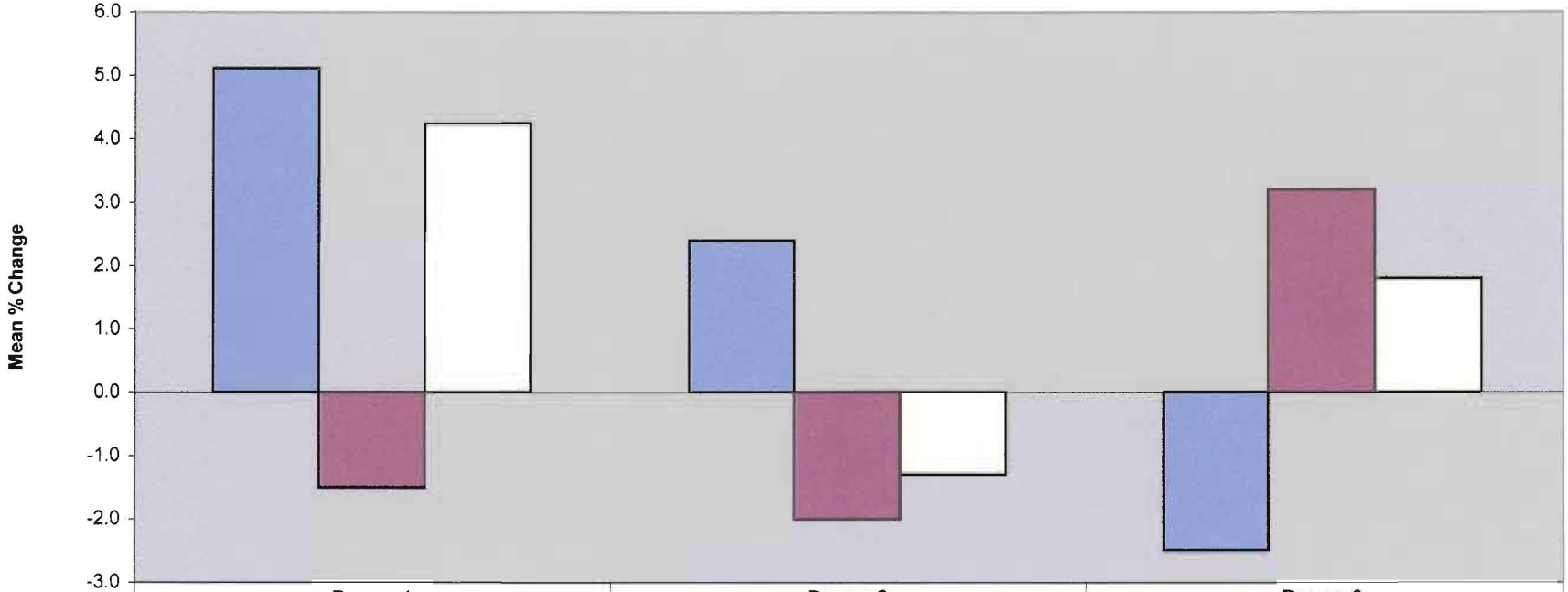
The ventilatory equivalent for carbon dioxide showed a decrease of 4.3% for Switch™ and a decrease of 3.7% for Cellfood®. The placebo showed a decrease of 1.6%. This indicates that Switch™ was most effective during this cycle, ensuring the best breathing efficiency. Switch™ showed an increase of 4.2% in the end tidal partial oxygen pressure while the Cellfood® showed a decrease of 1.5%. Placebo showed an increase of 5.1%. This indicates that Switch™ was the most effective during this cycle, ensuring the best breathing efficiency. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups.

FIGURE 4.12: BREATHING EQUIVALENT FOR $C0_2$



	Dosage 1	Dosage 2	Dosage 3
Placebo	-1.6	-1.2	2.1
Cellfood	-3.7	2.7	-2.0
Switch	-4.3	-1.6	-1.3

FIGURE 4.13: END TIDAL PARTIAL CARBON DIOXIDE PRESSURE



	Dosage 1	Dosage 2	Dosage 3
Placebo	5.1	2.4	-2.5
Cellfood	-1.5	-2.0	3.2
Switch	4.2	-1.3	1.8

Cycle 2 (Intermediate Dosage)

During this cycle Switch™ showed a decrease of 1.6% while Cellfood® showed an increase of 2.7% in the breathing equivalent for carbon dioxide. The placebo showed a decrease of 1.2%. This indicates that Switch™ was the most effective in improving the breathing efficiency during this cycle. Switch™ and Cellfood® showed decreases in end tidal partial carbon dioxide pressure (1.3% and 2.0% respectively). Placebo showed an increase of 2.4%. This indicates that neither Cellfood® nor Switch™ were very effective at this dosage. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups.

Cycle 3 (High Dosage)

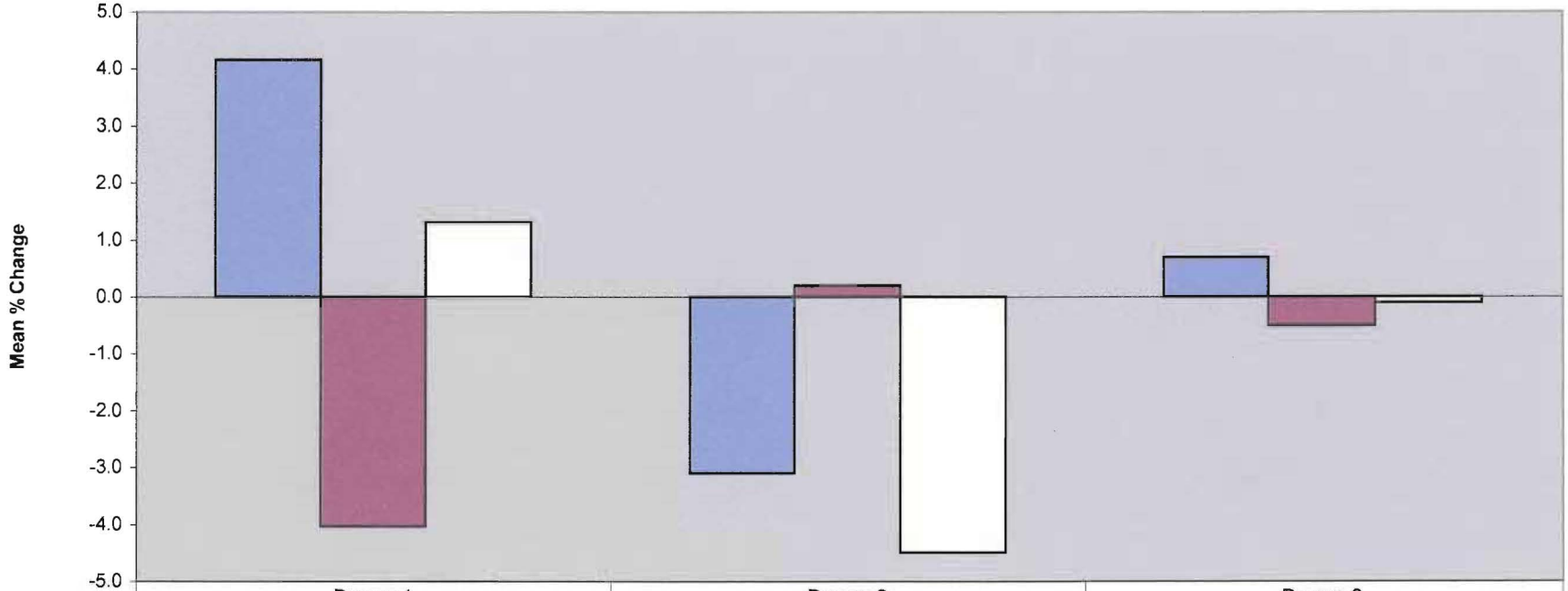
Cellfood® showed a decrease of 2% in the breathing equivalent for carbon dioxide, while Switch™ showed a decrease of 1.3%. Placebo showed an increase of 2.1%. This indicates that both Cellfood® and Switch™ were effective at improving breathing efficiency, but Cellfood® showed the best results during this cycle. Cellfood® showed an increase of 3.2% in the end tidal partial carbon dioxide pressure. Switch™ showed an increase of 1.8%. Placebo showed a decrease of 2.5%. This indicates that Cellfood® was the superior ergogenic aid when administered during the third cycle. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups.

4.10.3 Breathing Equivalent for 0_2 (Figure 4.14) and End Tidal Partial Oxygen Pressure (Figure 4.15) (Table VI A)

Cycle 1 (Low Dosage)

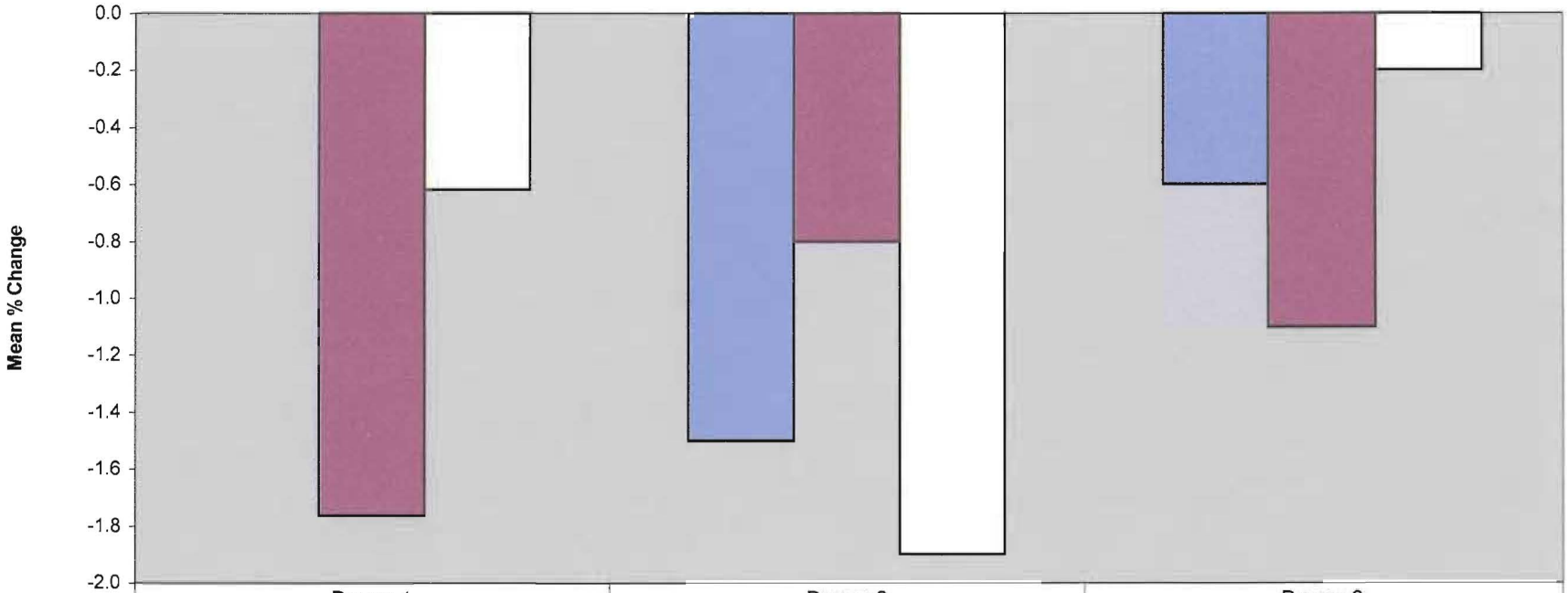
Cellfood® showed a decrease of 4% in the breathing equivalent for oxygen while Switch™ showed an increase of 1.3%. Placebo showed an increase of 4.2%. This indicates that Cellfood® was the most effective in improving breathing efficiency during this cycle. Cellfood® showed a decrease of 1.8% in the end tidal partial oxygen pressure. Switch™ showed a decrease of 0.6% while the placebo showed no changes when compared to the pre-test. These results confirms that Cellfood® was the most effective during this cycle. The within-group ergogenic improvement from base-line values of 4.0% in breathing equivalent observed in Cellfood® was statistically significant ($p<0.05$).

FIGURE 4.14: BREATHING EQUIVALENT FOR 0_2



	Dosage 1	Dosage 2	Dosage 3
Placebo	4.2	-3.1	0.7
Cellfood	-4.0	0.2	-0.5
Switch	1.3	-4.5	-0.1

FIGURE 4.15: END TIDAL PARTIAL OXYGEN PRESSURE



	Dosage 1	Dosage 2	Dosage 3
Placebo	0.0	-1.5	-0.6
Cellfood	-1.8	-0.8	-1.1
Switch	-0.6	-1.9	-0.2

Cycle 2 (Intermediate Dosage)

Switch™ showed a decrease of 4.5% in the breathing equivalent for oxygen. Cellfood® showed an increase of 0.2% while placebo showed a decrease of 3.1%. Switch™ was the most effective in improving the breathing efficiency during this cycle. Switch™ showed the greatest decrease in end tidal partial oxygen pressure at1.9%. Cellfood® showed a decrease of 0.8% and placebo showed a decrease of 1.5%. Overall, Switch™ had the most beneficial effect on the breathing efficiency during this cycle. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups. The within-group ergogenic improvement from base-line values of 4.5% in breathing equivalent observed in Switch™ was statistically significant ($p<0.05$).

Cycle 3 (High Dosage)

Cellfood® showed a decrease of 0.5% in the breathing equivalent for oxygen, while Switch™ showed an increase of 0.2%. The placebo showed an increase of 0.7%. Cellfood® showed a decrease of 1.1% in the end tidal partial pressure for oxygen while Switch™ and placebo showed decreases of 0.2% and 0.6%, respectively. Cellfood® was the most effective in improving the breathing efficiency during this cycle. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups.

4.10.4 Minute Ventilation (Figure 4.16), Tidal Volume (Figure 4.17) and Respiration Rate (Figure 4.18) (Table VI B)

Maximum minute ventilation is the highest value of ventilation which can be attained and measured during incremental exercise. With symptom-limited incremental exercise, minute ventilation typically increases from a resting value of 5-8 l/min up to 100-150 l/min. The response is non-linear. Every individual has a theoretical ventilatory capacity. This can be measured in the laboratory by performing a maximal voluntary ventilation (MVV) test. A normal individual uses

TABLE VI: GAS ANALYSES (B)

* = p< 0.05; % Δ = Relative Change; NS = Not Significant (p> 0.05)

VARIABLES			PL: PLACEBO (N=10)					CF: CELLFOOD (N=10)					SW: SWITCH (N=10)					SIGNIFICANCE		
GAS VALUES	Cycle	UNITS	PRE-TEST. X ± SD		POST-TEST X ± SD		%Δ	PRE-TEST. X ± SD		POST-TEST X ± SD		%Δ	PRE-TEST. X ± SD		POST-TEST X ± SD		%Δ	PL vs CF	PL vs SW	Cf vs SW
Minute Volume	1	l/min	100.1	19.9	106.9	26.2	6.9	105.1	20.3	94.3	17.6	-10.2	106.7	22.1	108.2	22.7	1.4	NS		
Minute Volume	2	l/min	106.7	22.1	105.3	17.9	-1.3	100.1	19.9	104.7	23.3	4.6	105.1	20.3	97.4	22.8	-7.3			
Minute Volume	3	l/min	105.1	20.3	107.7	31.6	2.5	106.7	22.1	111.4	23.3	4.4	100.1	19.9	106.7	23.6	6.6			
Tidal Volume	1	ml	1929.7	453.2	2068.8	489.1	7.2	2023.5	434.3	2059.4	313.5	1.8	2043.1	463.6	2165.6	497.8	6.0	NS		
Tidal Volume	2	ml	2043.1	463.6	2208.7	565.6	8.1	1929.7	453.2	2072.2	421.1	7.4	2.23.5	434.3	2108.8	355.1	4.2			
Tidal Volume	3	ml	2023.5	434.3	2127.8	511.3	5.2	2043.1	463.6	2297.0	626.7	12.4	1929.7	453.2	2083.5	450.9	8.0			
Respiration Rate	1	/min	52.6	5.7	52.1	5.5	-1.0	53.2	13.9	45.6	5.4	-14.3	52.7	6.1	50.2	6.2	-4.7	NS		
Respiration Rate	2	/min	52.7	6.1	49.2	8.0	-6.6	52.6	5.7	50.7	5.1	-3.6	53.2	13.9	45.9	7.1	-13.7			
Respiration Rate	3	/min	53.2	13.9	51.3	13.7	-3.6	52.7	6.1	50.0	8.0	-5.1	52.6	5.7	51.6	5.3	-1.9			
VO2max (absolute)	1	ml/min	3101.9	564.8	3187.1	702.5	2.7	3262.4	701.2	3042.8	647.48	-4.3	3178.5	689.1	3165.6	594.3	-0.4	NS		
VO2max (absolute)	2	ml/min	3178.5	689.1	3246.8	580.2	2.1	3101.9	564.8	3230.3	598.3	4.1	3262.4	701.2	3176.3	845.7	-2.6			
VO2max (absolute)	3	ml/min	3262.4	701.2	3314.1	906.6	1.6	3178.5	689.1	3337.0	664.1	5.0 *	3101.9	564.8	3294.4	598.4	6.2			
VO2 max (relative)	1	ml/kg/min	46.5	7.6	42.2	7.1	-9.3	45.9	9.9	47.0	7.8	2.4	45.1	7.4	48.3	5.6	7.1	NS		
VO2 max (relative)	2	ml/kg/min	45.1	7.4	46.6	8.0	3.2	46.5	7.6	45.5	7.8	-2.1	45.9	9.9	46.9	7.3	2.1			
VO2 max (relative)	3	ml/kg/min	45.9	9.9	49.4	7.5	7.7	45.1	7.4	45.3	4.0	0.5	46.5	7.6	46.5	9.5	-0.1			

50-75% of his or her ventilatory capacity at maximum exercise. Thus, a normal individual is not expected to exhibit ventilatory limitation. Athletes who have successfully extended their cardiovascular fitness use a higher proportion of their ventilatory capacity at maximum exercise (Cooper and Storer, 2001).

Tidal volume (VT) is the volume of a single breath. Normal VT varies according to body size and also varies from breath to breath. During exercise VT increases in a nonlinear fashion reaching a plateau value equal to approximately 50-60% of vital capacity at about 70% of $V0_2max$.

Respiration rate (RR) is the number of breaths taken per minute. A normal resting respiration rate is 8-12 breaths per minute. Characteristically RR increases steadily to a maximum value of between 30 and 40 per minute. RR rarely exceeds 50 per minute. However, some elite athletes may exhibit RR values as high as 80 per minute at maximum exercise (Cooper and Storer, 2001).

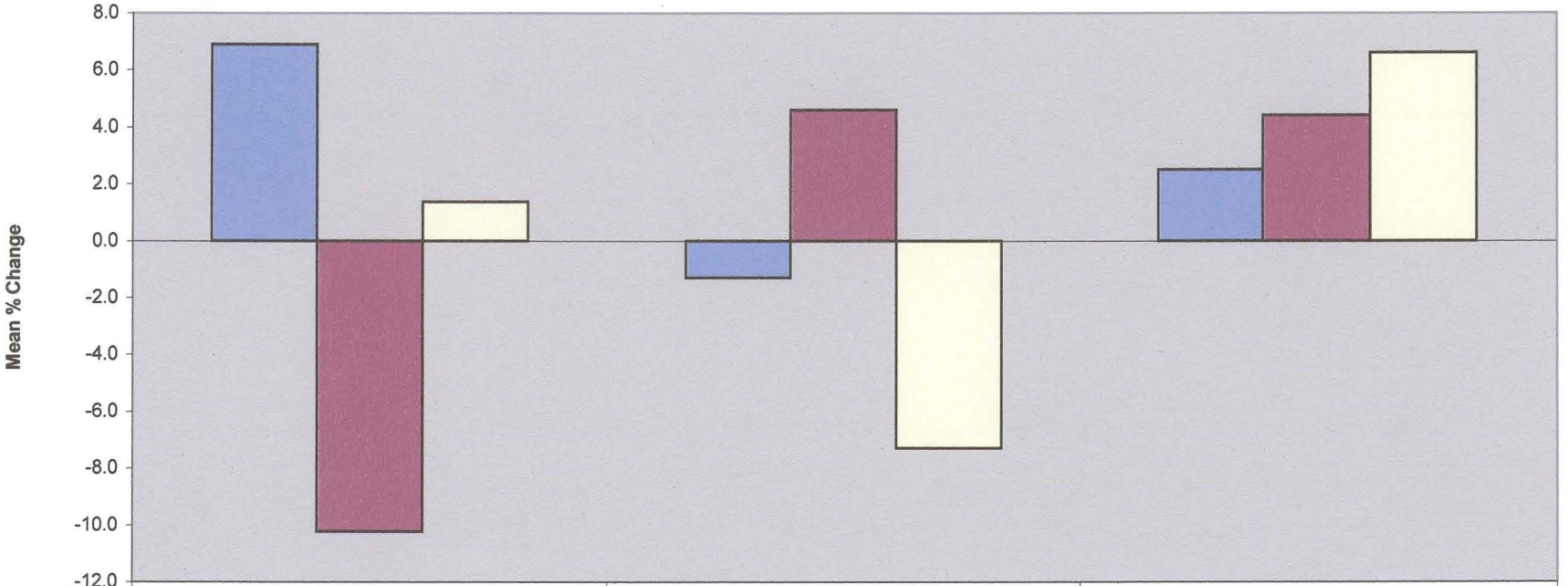
Cycle 1 (Low Dosage)

During this cycle Switch™ showed an increase of 1.4% in minute volume. This increase was possible due to an increase of 6% in tidal volume and a decrease of 4.7% in respiration rate. Cellfood® showed a decrease of 10.2% in minute volume. This was due to an increase of 1.8% in tidal volume but a decrease of 14.3% in respiration rate. The placebo showed an increase of 6.9% in minute volume. This increase was possible due to an increase of 7.2% in tidal volume and a decrease of 1% in the respiration rate. Switch™ was the more beneficial supplement during this cycle, but did not prove more beneficial than the placebo. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups.

Cycle 2 (Intermediate Dosage)

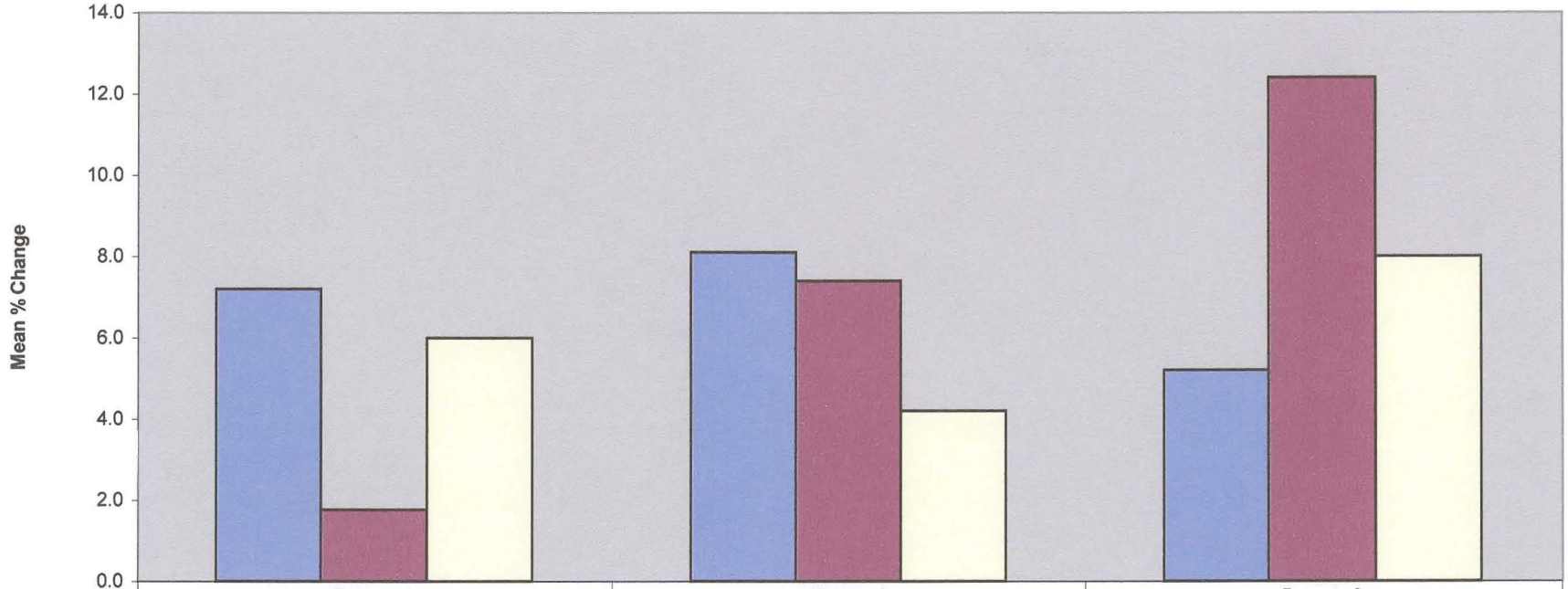
Cellfood® was the only group that showed an increase (4.6%) in minute volume during this cycle. This increase was possible due to an increase of 7.2% in tidal volume and a decrease of 3.6% in the respiration rate. Switch™ showed a decrease of 7.3% in minute volume. This was influenced by an increase of 4.2% in tidal volume but a decrease of 13.7% in the respiration rate. The placebo showed a

FIGURE 4.16: MINUTE VENTILATION



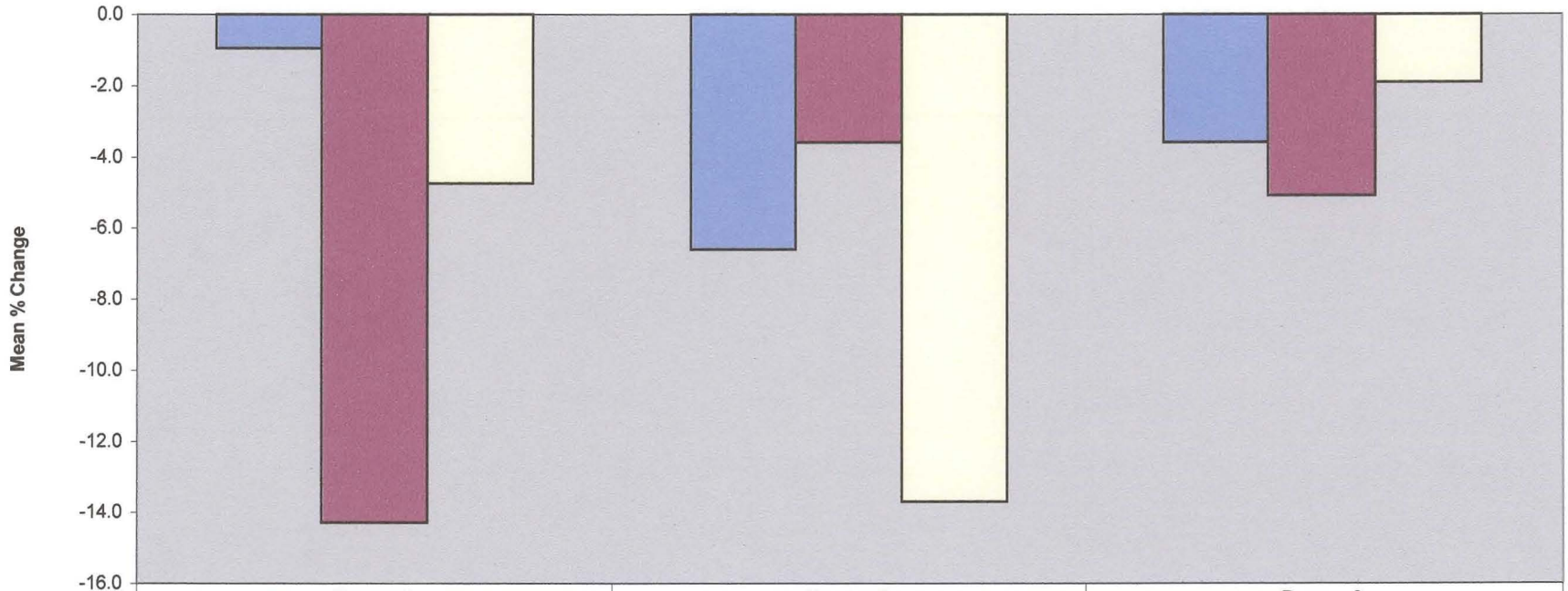
	Dosage 1	Dosage 2	Dosage 3
Placebo	6.9	-1.3	2.5
Cellfood	-10.2	4.6	4.4
Switch	1.4	-7.3	6.6

FIGURE 4.17: TIDAL VOLUME



	Dosage 1	Dosage 2	Dosage 3
Placebo	7.2	8.1	5.2
Cellfood	1.8	7.4	12.4
Switch	6.0	4.2	8.0

FIGURE 4.18: RESPIRATION RATE



	Dosage 1	Dosage 2	Dosage 3
Placebo	-1.0	-6.6	-3.6
Cellfood	-14.3	-3.6	-5.1
Switch	-4.7	-13.7	-1.9

decrease of 1.3% in minute volume. This was influenced by an increase of 5.2% in tidal volume but a decrease of 3.6% in the respiration rate. Cellfood® was the most beneficial supplement during this cycle. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups.

Cycle 3 (High Dosage)

Switch™ showed an increase of 6.6% in minute volume. This increase was established through an increase of 8.0% in tidal volume and a decrease of 1.9% in the respiration rate. Cellfood® showed an increase of 4.4% in minute volume. There was an increase of 12.4% in tidal volume and a decrease of 5.1% in the respiration rate. The placebo showed an increase of 2.5% in the minute volume through an increase of 5.2% in the tidal volume and a decrease of 3.6% in the respiration rate. Switch™ had the most beneficial effect on the minute volume during this cycle. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups.

4.10.5 Maximal Oxygen Uptake ($V0_2$ max): Absolute (Figure 4.19) and relative (Figure 4.20) (Table VI B)

The components of the system and their interrelationship are as follows:

$V0_2$ (oxygen transported) = SV x HR x a-$v0_2$ diff (arteriovenous oxygen difference)

From the above equation we can derive that to maintain a certain $V0_2$ during exercise with lower comparative heart rates the subjects must either experience an increase in stroke volume or a increase in arteriovenous oxygen difference. Athletes who excel in endurance sports generally have a large capacity for aerobic energy transfer. The maximal oxygen consumption recorded for competitors in distance running are at most double those of sedentary men and woman. This is not to say that the VO_2 max is the only determinant of endurance performance. Other factors, principally those at local tissue level such as capillary density, enzymes, mitochondrial size and number, and muscle fibre type, exert a strong

influence on a muscle's capacity to sustain a high level of aerobic exercise (McArdle et al., 1996). The VO_2 max does, however, provide important information on the capacity of the long term energy system. In addition, this measure has significant physiological meaning in that attaining a high VO_2 max requires the integration of a high level of ventilatory, cardiovascular, and neuromuscular functions (McArdle et al., 1996). A healthy but sedentary adult male might have a $V0_2$ max of 35ml/kg/min. In a normal individual performing incremental exercise, $V0_2$ can increase by as much as sixteen-fold. $V0_2$ max is clearly related to the type of exercise performed. The prediction of normal $V0_2$ should therefore take into account exercise mode, gender, age, and body size (Cooper and Storer, 2001).

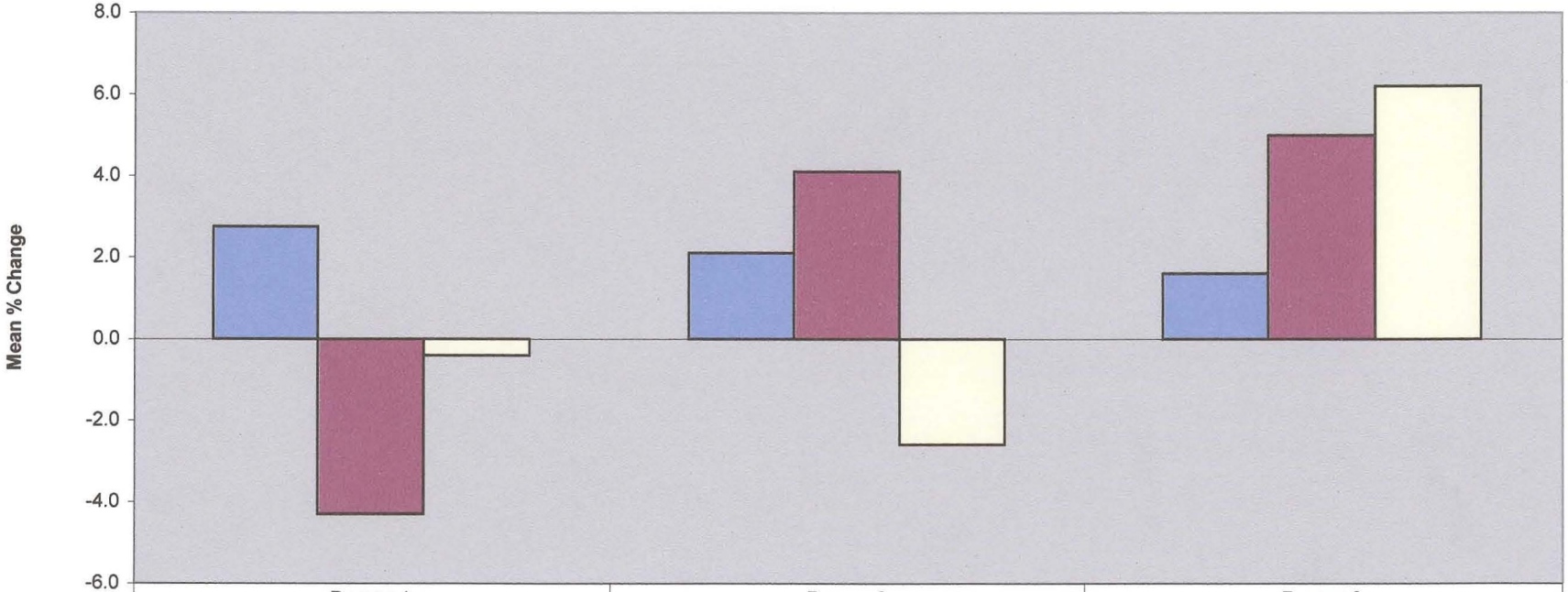
Cycle 1 (Low Dosage)

Switch™ showed a decrease of 0.4% in absolute $V0_2$max with an increase of 7.1% in relative $V0_2$ max. Cellfood® showed a decrease of 4.3% in absolute $V0_2$ max with an increase of 2.4% in relative $V0_2$ max. The placebo showed an increase of 2.7% in absolute $V0_2$ max and a decrease of 9.3% in relative $V0_2$ max. This indicates that neither Cellfood® nor Switch™ were effective in increasing the absolute $V0_2$ max although both supplements increased the relative oxygen consumption of the subjects. This is a anomalous trend that could only be explained when linking it to a decrease in body weight. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups.

Cycle 2 (Intermediate Dosage)

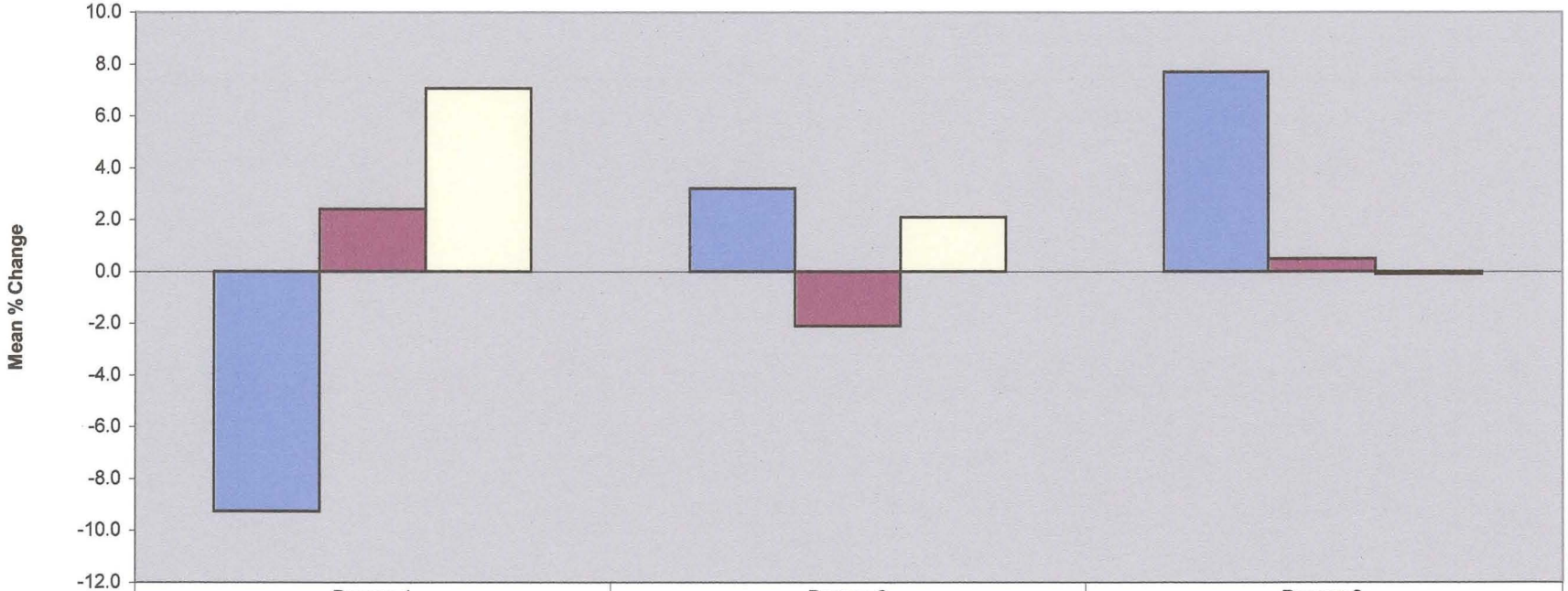
Cellfood® showed an increase of 4.1% in absolute $V0_2$ max but a decrease of 2.1% in the relative oxygen consumption. Switch™ showed a decrease of 2.6% in absolute $V0_2$ max but still an increase of 2.1% in the relative oxygen consumption. Placebo showed an increase of 2.1% in absolute $V0_2$ max with an increase of 7.7% in relative oxygen consumption. During this cycle Cellfood® was the most effective in increasing the absolute oxygen consumption. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups.

FIGURE 4.19: ABSOLUTE $V0_2$ MAX



	Dosage 1	Dosage 2	Dosage 3
Placebo	2.7	2.1	1.6
Cellfood	-4.3	4.1	5.0
Switch	-0.4	-2.6	6.2

FIGURE 4.20: RELATIVE $V0_2$ MAX



	Dosage 1	Dosage 2	Dosage 3
Placebo	-9.3	3.2	7.7
Cellfood	2.4	-2.1	0.5
Switch	7.1	2.1	-0.1

Cycle 3 (High Dosage)

Switch™ showed an increase of 6.2% in absolute $V0_2$ max while the relative oxygen consumption decreased with 0.1%. Cellfood® showed an increase of 5.0% in absolute $V0_2$ max together with an increase of 0.5% in relative oxygen consumption. Placebo showed an increase of 1.6% in absolute $V0_2$ max, and an increase of 7.7% in relative oxygen consumption. Cellfood® was the most effective in increasing the absolute maximal oxygen consumption during this cycle. There were, however, no statistically significant differences ($p>0.05$) in the changes between groups. The within-group ergogenic improvement from base-line values of 5.0% in absolute $V0_2$max observed in Cellfood® was statistically significant ($p<0.05$).

4.11 Comparative Findings

In general, findings of this study showed that Cellfood® and Switch™ had ergogenic benefits in the following variables:

- Ferretin
- Haemoglobin
- Red blood cell count
- Hematocrit
- Glucose
- Pulse Oximetry
- RPE
- Heart Rate
- Lactate
- RER
- $VC0_2$
- $VE/VC0_2$
- $etC0_2$
- $VE/V0_2$
- $et0_2$
- VT
- RR

- Relative $V0_2max$

Considering that this was the first exercise-related study conducted on both Cellfood® and Switch™, directly comparative findings are not available. Therefore the only prudent approach is to integrate these findings with the literature by relating the performance of other ergogenic aids and methods that also claim similar benefits for those variables measured during this study.

Haematology Related Ergogenic Aids

- Iron Supplementation

  As the daily intake of iron, especially in females, is only marginally above that which is just sufficient to balance their normal daily iron losses, the additional iron losses caused by running may be sufficient to cause iron deficiency. Runners most likely to become iron deficient are those who run high weekly mileages; women runners who may loose large amounts of blood due to monthly menstruation; and those who eat iron-deficient diets. Foods that have a high iron content include liver, red meat, egg yolk, legumes, dark green leafy vegetables, molasses and whole grain. Vegetarians who eat no meat or eggs are considered to be particularly prone to iron deficiency. All these groups could benefit by eating more red meat, the dark meat of poultry, or liver, and by taking iron tablets orally if their blood haemoglobin levels are found to be sufficiently low to indicate that they might have iron-deficiency anaemia. Only if an athlete is found to have an iron-deficiency anaemia should iron therapy be considered. A drawback to iron therapy is that iron tablets tend to cause indigestion and constipation. Of the commercially available iron tablets, those least likely to cause problems are the "slow-release" forms and the iron chelates. Iron is best absorbed when taken with orange juice, this is due to the vitamin C present in the orange juice that aids in iron absorption (Noakes, 1992). Although neither Cellfood® nor Switch™ changed the ferretin values significantly, both of these supplements were effective in increasing the ferretin values of the athletes without any known detrimental side effects.

- Blood Doping

  Blood doping is one (illegal and dangerous) way of trying to increase the body's natural red blood cell counts. With the procedure, usually between 1 to 4 units of a person's own blood (autologos) are withdrawn, the plasma is removed and immediately reinfused, and the packed red cells are placed in frozen storage. To prevent a dramatic reduction in blood cell concentration, each unit of blood is withdrawn over a 3 to 8 week period because it generally takes this long for the person to re-establish normal red cell levels. The stored cells are reinfused 1 to 7 days before an endurance event. As a result, the red cell count and haemoglobin level of the blood is often elevated some 8 to 20%. Scientific studies of blood doping and endurance performance have produced conflicting results. Several studies have shown that blood doping increases endurance performance between 13% and 39% (measured by a treadmill run to exhaustion) and maximal oxygen consumption between 5% and 31% in both non-athletic and highly-trained endurance athletes (Belko, 1987; Ekblom et al., 1973; Ekblom et al., 1976). An equal number of studies (mostly from earlier literature), however, have found no effects of blood doping on endurance performance, maximal oxygen consumption, heart rate responses during exercise, or perceived exertion (Cunningham, 1978; Pate et al., 1979; Videman and Rytomaa, 1977). An examination of research design differences clarifies much of the conflicting evidence. Two critical factors became apparent: (1) when between 800 and 1200ml of blood, or its equivalent, is reinfused (as opposed to 450 to 500ml), aerobic capacity and endurance increases and (2) when 5 to 6 weeks elapse before reinfusion, "positive" results also are seen (Fox and Bowers, 1993).

- Erythropoietin

  Another technique for boosting red blood cells became apparent in the late 1980's. Erythropoietin (EPO), a hormone produced by the kidneys, stimulates the production of RBC's under conditions of hypoxia (chronic low oxygen tension in the blood) and anemia. It travels via the circulation to the bone marrow, where it stimulates the production of red cells. The rate of formation of new red cells, is in part determined by EPO. Injections

of EPO are very effective, athletes can expect enhancements in endurance performance of 5% or more (Sawka and Joyner et al., 1998; Birkeland et al., 2000). The benefits of this technique are similar to those achieved by blood doping. The non-therapeutic uses of EPO poses a significant health risk. The inappropriate use of EPO increases the thickness or viscosity of the blood so that the blood has difficulty passing through small blood vessels, in essence simulating the disease of erythrocythemia and polycythemia. When this increased viscosity effect is combined with the dehydration that is encountered in competitive athletics, the viscosity of the blood increases further, producing sludging of the blood in the vessels. At hematocrit above 55%, the blood viscosity increases exponentially, thereby substantially increasing the risk of a coronary artery occlusion or a cerebral artery occlusion. Similarly, occlusions can occur in other blood vessels producing other complications. With this in mind, it has been speculated that EPO may have contributed to the unusually high number of deaths that have occurred in competitive cyclists from the Netherlands and Belgium (MIMS, 1996; Fox and Bowers, 1993; McArdle et al., 1991; Noakes, 1992). Both Cellfood® and Switch™ were effective in increasing both red blood cell count and hematocrit values throughout the study. Theses increases never exceeded the normal physiological range and therefore is much safer than the previously mentioned techniques of blood doping and EPO.

Metabolism Related Ergogenic Aids

- Carbohydrate

Carbohydrate (CHO) loading is one of the more popular methods of nutritional modification used by endurance athletes to improve performance. It is also one of the most studied ergogenic aids for athletic performance (Walberg-Rankin, 1995). Although the judicious adherence to this dietary technique can significantly improve specific performances, there are also some negative aspects that could prove detrimental (McArdle et al., 1993). Reduction of body stores of carbohydrate and blood glucose is related to the perception of fatigue and the inability to maintain high-quality performance. This has been clearly shown with

aerobic, endurance events of moderate intensity of over 90 minutes duration. Carbohydrate intake may also have relevance for athletes involved in short, high-intensity events, especially if body weight control is an issue (Walberg-Rankin, 1995). The process of glycogen loading (carbohydrate loading) may be incorporated to elevate muscle glycogen stores above their normal resting levels prior to endurance competition (Fox and Bowers, 1993). Generally glycogen super-compensation is applicable where the athlete is continuously in motion for more than an hour at a time. Super-compensation may have value for events with an anaerobic component to the extent that lowered levels of glycogen can have adverse effects on lactate production (anaerobic power) (Fox and Bowers, 1993). Some recent studies further confirm that consumption of a high-carbohydrate diet for 2 or more days prior to an endurance event enhances performance relative to a low carbohydrate diet. For example, O'Keefe et al. (1989) found that 1 week on a 72% carbohydrate diet allowed cyclists to exercise at 80% of $V0_2$ max for 113 minutes, whereas they could only cycle 60 minutes when they consumed a 13% carbohydrate diet for the same period (Walberg-Rankin, 1995). Under circumstances where an athlete must perform multiple events in one day, super-compensation is appropriate. For these purposes one can simply increase the dietary intake of carbohydrates for 48 to 72 hours prior to competition. The practice of ingesting glucose 30 to 45 minutes before competition is not recommended. It can lead to a rapid fall in blood glucose levels with the onset of exercise and increase the rate of glycogen utilization. One other point of consideration is that with the storage of one gram of glucose about 2.7 grams of water will be taken into storage. Thus, with a storage of 700 gm of glucose an additional storage of about 1.9 kg of water will occur. An athlete should thus not be surprised to have a pre-competition weight gain, which may be an advantage or disadvantage depending on the event.

- Bicarbonate Loading ($NaHC0_3$)

  Blood doping is one known method to try and lower lactate levels in endurance athletes. It is now clear that blood doping reduces blood lactate

levels during exercise and alters the lactate turn point to higher running speeds. These effects are likely to be the more important explanations for the increased running performance after blood doping (Noakes, 1992). The ergogenic properties of $NaHC0_3$ ingestion have been the focus of various investigations (Verbitsky et al., 1997.; Matson and Tran, 1993). It has been shown that during short-term, high-intensity physical activity progressive metabolic acidosis due to lactic acid accumulation and a drop of pH takes place, in both the blood and working muscles (Verbitsky et al., 1997). The resultant accumulation of H^+ within the muscle directly inhibits the contractile process by inhibiting the release of Ca^{2+} from the sarcoplasmic reticulum , as well as by reducing the activity of glycolytic enzymes, thus impairing the propagation of neural impulses. As exercise progresses, various buffering mechanisms function to neutralize this effect. Eventually, when the intracellular buffering capacity is exceeded, H^+ diffuse into the blood, causing a drop in extracellular pH. This, in turn, stimulates extracellular buffering mechanisms of which $HC0^-_3$ os one of the most effective constituents. It is thus expected that acute ingestion of $NaHC0_3$ should at this stage enhance the buffering capacity in the blood and hence delay exhaustion and improve performance (Verbitsky et al., 1997).

- L- Carnitine Loading

  L- Carnitine is a well-known supplement often used to try and lower lactate concentrations, it plays a role in regulating the balance between key chemicals in metabolic processes and is known to act as a buffer for pyruvate, thus reducing muscle lactate accumulation associated with fatigue (Armsey and Green, 1997). The possible mechanisms by which carnitine could have a positive effect include enhancing oxidation of fatty acids, a critical energy compound during exercise; preserving muscle glycogen during exercise, a factor potentially related to fatigue resistance; shifting fuel use towards glucose, thereby decreasing the oxygen requirement of exercise; improving resistance to muscle fatigue; and increasing the oxidative capacity of skeletal muscle (Phillips, 1997). It has been reported that after a period of carnitine supplementation, well-trained

subjects increased their maximal oxygen consumption. The key claim of this supplement is that it can enhance fat metabolism by increasing the transport of fat to its site of oxidation. Long-chain fatty acid oxidation in all tissues is carnitine dependent; therefore, hereditary and acquired carnitine deficiencies cause triglyceride to accumulate in the skeletal muscles, impair fatty acid utilization, and reduce exercise capacity. Carnitine supplementation can usually reverse these changes (Hawley, 1998). Neither Cellfood® nor Switch™ lowered lactate accumulation significantly during any cycles of the study, but it seems that when administered at the correct dosages, low dosage for Switch™ and the high dosage for Cellfood®, these supplements were effective in lowering lactate accumulation throughout the treadmill test.

Oxygenation Related Ergogenic Aids

- Oxygen Enriched Air

  It is common to observe athletes breathing oxygen-enriched or hyperoxic gas mixtures during times out, at half time, or following strenuous exercise. The belief is that this procedure significantly enhances the blood's oxygen carrying capacity and thus facilitates oxygen transport to the exercising muscles. The fact is however, that when healthy people breathe ambient air at sea level, the haemoglobin in arterial blood leaving the lungs is about 95 to 98% saturated with oxygen. Thus, breathing high concentrations of oxygen could increase oxygen transport by haemoglobin to only a small extent, i.e., about 1 ml of extra oxygen for every 100ml of whole blood. The oxygen dissolved in plasma when breathing a hyperoxic mixture would also increase slightly from its normal quantity of 0.3ml to about 0.7ml per 100ml of blood. Thus, the blood's oxygen-carrying capacity under hyperoxic conditions would be increased potentially by about 1.4ml of oxygen for every 100ml of blood, with 1.0ml extra attached to hemoglobin and 0.4ml extra dissolved in plasma (McArdle et al., 1996). Regardless, there is a rather large body of information indicating that breathing oxygen-enriched air (33 to 100% oxygen) has a beneficial effect

on exercise performance (Allen and Pandolf, 1977; Hughes et al., 1968; Miller, 1952). Oxygen breathing during both light and heavy exercise has resulted in reduced blood lactic acid levels, heart rates, ventilation volumes, and a significant increase in maximal oxygen consumption.

- $HB0_2$

  Hyperbaric oxygen is another method of oxygen therapy well known to sport. Professional athletes have reportedly received hyperbaric oxygen before sports participation, believing that performance would improve. Contradictory findings have been reported regarding the effect of a single hyperbaric oxygen treatment on aerobic performance. A Yugoslavian study (Staples and Clement, 1996), demonstrated that hyperbaric oxygen prior to treadmill running to volitional exhaustion increased peak running velocity and maximal oxygen consumption when measured 30 minutes and 3 hours post treatment. $HB0_2$ was administered for 60 minutes at 2.8 ATA. Enhanced performance and $V0_2$ max were attributed to additional oxygen storage in skeletal muscle. However to their knowledge, this link has yet to be definitely established (Delaney, 2001). In contrast, two recent studies (James et al., 1993; Potera, 1995), reported no change in submaximal and maximal exercise performance following hyperbaric oxygen therapy. Both Cellfood® and Switch™ were effective in increasing the haemoglobin concentration of the athletes throughout the study. These increases never exceeded the normal physiological ranges for the variable. The increased haemoglobin concentration together with an increase in haemoglobin saturation (although not significant at all running speeds) could benefit the athlete in an increased oxygen delivery throughout the body and working muscles.