# "Panoptic Differential Training: Bio-Dynamics Approach for Sprint Time Trial Cycling Performance" – A Critical Perspective

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*Background: Intensity of the exercise mostly determines the dominant metabolic pathway for energy. In a sprint cycling 1km time trial, the dominant metabolic pathway must be ATP hydrolysis and glycolysis. Phosphocreatine (PCr) hydrolysis and Anaerobic Glycolysis lead to lactate and H+ accumulation in sarcolemmal cytosol, interfering the muscle contraction leading to fatigue and reduction of power output. To resist fatigue and to continue the high power output throughout the cycling sprint, the lactate flux is an essential phenomenon apart from the regeneration of the ATP. The appearance of H+ and lactate is simultaneous with high intensity exercise, hence the co transport of lactate-H+ is essential and the training should target both the systems to resist fatigue and sustain the cycling sprint power output at maximum throughout the time trial. Biodynamic Implications: Resting ATP and PCr stores of muscle seems less responsive to training and hence strengthening lactate transfer and oxidation appear better alternative along with more concentration on early oxidative phosphorylation. Monocarboxylate Transporter (MCT) isoforms like MCT1 and MCT4 expression should be increased to increase the lactate transport. Load of pH gradient along with lactate flux to be targeted during the training. MCTs also facilitate the H+ efflux and prevent the decrements in intracellular pH. Training Implications: High intensity training has significant influence on the status of both MCT1 and MCT4 ranging from 18% to 120%, though inter individual differences have been observed. Slow endurance training, like sub maximal sprint repetitions increases MCT1 and MCT4 expression leading to lactate uptake and oxidation. With the increase in sustaines sprints without much lactate accumulation during the initial seconds improves oxidative enzymal expression significantly. An acute high intensity sprint form of exercise could reduce the MCT1 expression considerably wherein high expression of H+ is seen in the myocyctes. . Recommendation: very high intensity sprint cycling in repetition need to be reduced to the minimum to avoid excess accumulation of H+. Instead differential training like repetitions of sub maximal runs with initial forty seconds of high intensity sprint cycling followed by sub maximal sprinting for*

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*another thirty or forty seconds. Supra maximal sprint cycling of sixty seconds to seventy seconds may be done once in a week with complete recovery in between for two to three times. The training protocol need to be structured in a more vivid form keeping in view of the bio dynamics of the sprint cycling instead of simple interval training.*

## Introduction

Expansion in research on bioenergetics of human muscle contraction has optimised the knowledge on training methodologies for improvement in physical performances. Slowly and progressively, the trainers and coaches are adopting highly evolved scientific training protocols for world class physical performances. Endowment of gene orientation towards a favourable physiology for being a world class athlete or sportsperson is not sufficient unless the same is complimented by highly scientifically evolved training methodologies. Hence, the nurturing of a sportsperson is essential to mould an athlete into a world beater. Sprint cycling being a supra maximal intensity effort for short duration, requires a complex metabolic pathway for energy and other bioenergetics to avoid early fatigue or to sustain the sprint cycling with exceptional timings. One kilometre sprint cycling lasting up to one minute to one minute and ten seconds requires training keeping in view of the bioenergetics of event. Unlike running, cycling involves few muscles more prominently like quadriceps, Gluts, fore leg muscles like gastrocnemius, soleus etc, the most dominant muscles being quads. High intensity sprint cycling requires high amounts of rapid energy generated throughout the cycling effort and coupled with efflux of fatigue promoting substances very efficiently.

Energy for this very high intensity or supra maximal effort is derived though the mixture of substrate phosphorylation or anaerobic phosphorylation an immediate and instant source of energy, Glycogenolytic or Glycolytic phosphorylation and also to some extent from oxidative phosphorylation. Adenosine Triphosphate (ATP) is formed by direct transfer of a phosphoryl ($PO_3$) group derived from the split of Creatine phosphate (PCr) to Adenosine diphosphate (ADP) leading to fast depletion of PCr reserves of working muscles during the substrate phosphorylation under an anaerobic metabolic pathway. Since, the PCr reserves in human skeletal muscle are limited (approximately 75 to 90 mmol/kgdm) the substrate phosphorylation ATP synthesis can provide for a few seconds of very high intensity sprint cycling.

Hence, the second pathway of anaerobic ATP production i.e. Glycogenolytic or Glycolytic phosphorylation provides ATP during high intensity or supra maximal exercise along with the substrate phosphorylation. The sustained sprint cycling beyond thirty seconds requires a higher proportion of ATP synthesis from the Glycolytic pathway than the PCr system. The PCr reserves will get exhausted almost to nil within the first fifteen to twenty seconds and hence a major portion of the ATP provision should come from

Glycolytic pathway (Forbes et. al. 2009). Though the aerobic metabolic pathway of ATP synthesis also complements during the one km sprint cycling, the proportion is significantly less when compared to anaerobic pathways. Hence the maximum dependency for ATP synthesis during the one kilometre sprint cycling rests on anaerobic metabolic pathways. High rate of ATP demand during the sprint cycling, causes for the initiation of more vigorous anaerobic phosphorylation leading to significant accumulation of ADP, Adenosine Monophosphate (AMP), Phosphate ions (Pi) in the sarcoplasm. Under conditions of excessive accumulation of ADP, AMP, Pi in the muscle environment the PCr use and the Glycogennolytic/Glycolytic flux will also be maximized. With increments in Glycolytic flux, the key glycolytic enzymes Glycogen Phosphorylase (PHOS) and Phosphofructokinase (PFK) cause for the synthesis of ATP at a very rapid rate leading to the formation of pyruvate, which in turn will be converted to Lactate by the near equilibrium and very abundant enzyme Lactate Dehydrogenase (LDH). During one km sprint cycling the rapid increase in the demand of energy for the leg muscles is associated with higher glycolytic flux and subsequent production and accumulation of lactate and protons (Burgomaster et. al. 2007). But, the capacity of anaerobic glycolysis in synthesising the ATP may not be related to the resting muscle glycogen values, but strongly related to the ionic changes during the high rates of Glycolytic flux. LDH catalyses the lactate production from pyruvate to maintain the high rates of Glycolytic flux during the supra maximal physical efforts like one km sprint cycling. This reaction paves way for the supply of proton-electron supplier nicotinamide adenine dinucleotide ($NAD^+$) to glycolysis for rapid production of ATPs; hence lactate production in Glycolysis cannot be seen as end product or as waste product of Glycolysis, but as a promoter of anaerobic glycolysis under high rates of energy demand (Lamb et. al. 2006).

Though not significant when compared to the anaerobic phosphorylation, oxidative phosphorylation also plays some portion in providing ATP supply to the working muscle during the later stages of the one km sprint cycling effort along with the anaerobic pathway. Beta oxidation and electron transport chains are sequential like substrate transport into the cell, cytoplasmic metabolism, transport into the mitochondrion, mitochondrial metabolism, electron delivery to the respiratory chain, the activities of complex I or II, complex III, complex IV, ATP synthesis, proton leak, ATP export to the cytoplasm and cell ATP utilization. These complex phases of oxidative phosphorylation depend on two electrophysiological phenomena called the plasma membrane potential and proton motive force. These two phenomena work in concurrence and contribute for the influx of substrate into mitochondria or efflux of protons from mitochondria and thereby control the oxidative metabolic pathway (Juel et. al. 2004). Substrate oxidation which include uptake of substrate, enzyme processing, pooling of ubiquinone and cyctochrome and electron transport chain complexes, leading to higher ATP turnover by the action of adenine nucleotide translocase, phosphate transporter and ATP synthase.

**Bioenergetics of Fatigue**

Bioenergetics of fatigue during sprint cycling: Stored PCr in muscles can provide ATP supply for only a few seconds during the initial phase of the sprint cycling. Even the stored muscle ATP provide energy for few seconds and together the stored ATP and PCr reserves could provide energy for fifteen to twenty seconds of the sprint cycling before both the reserves go depleted almost to their minimum. It is difficult to augment the reserves of muscle PCr stores through physical training, though inter individual variations exist in the amounts of muscle PCrs ranging from 100 to 150 mmol/kgdm). Oral administration of creatine could enhance the plasma concentrations of creatine leading to enhancements in PCr in skeletal muscles (Evans et. al. 2012). Pre exercise state of muscle glycogen reserves play an important part in enhancing the muscle glygogenolysis leading to enhanced glycolysis in skeletal working muscles. Incidentally, the resting muscle glycogen reserves can be increased through sub maximal exercise than supra maximal exercise. The insulin stimulated Glucose Transporter (GLUT-4) translocation will account for increase in uptake of glucose into sarcolemma leading to enhanced glycolysis. It is observed that the increased GLUT-4 translocation occurs when there is a decrease in muscle glycogen reserves during the sprint cycling. High intensity and sub maximal intensity exercise causes increased availability of GLUT-4 and also better translocation to sarcolemmal surface. Increase in intracellular acidity (decrease in $p^{H)}$ causes accelerated accumulation of lactate/$H^+$ in sarcoplasm. High levels of appearance of proton presence along with the lactate leading to cellular acidification augments for earlier fatigue and reduction in contraction-coupling strength and consequent reduction in power output during the sprint cycling. This significantly affects the timing of the cycling time trial. To delay the development of fatigue, the effective efflux of $H^+$ is essential and also the lactate transport mechanism should augment to reduce the cellular acidification (Brooks 2009). It is also observed that large amounts of lactate negatively influence the contraction-coupling mechanism (Kristensen et. al. 2004) of the skeletal muscle fibre and will cause the reduction of the power output of contraction. Well defined lactate transporter isoforms in human skeletal muscle are a Monocarboxilate Transporter 1 (MCT 1) and MCT 4, though they vary as per the muscle fibre type (Merezhinskaya and Fishbein 2009). These transporters facilitate lactate flux as per the concentration of lactate, and also maintain proton gradiance allowing protons to efflux or influx (Kitaoka et. al. 2012). Keeping in view of the importance of lactate transport (efflux and influx) and extrusion of protons, MCT 1 and MCT 4 are considered as very important regulatory candidates of cellular $p^H$ and in resistance to fatigue during the very high intensity exercise conditions like one km sprint cycling trial (Thomas et. al. 2005). Though MCT 4 is not correlated with any fibre type, MCT 1 is found predominantly in oxidative muscle fibres. MCT 4 is endowed with high lactate transport capacity and hence plays a vital role in extrusion of lactate from glycolytic fibres. MCT 1 predominantly takes up lactate from circulation leading to clearance of lactate from circulation.

MCT1 and MCT4 play important roles in the regulation of intracellular pH during high-intensity exercise, since they mediate most, but not all, of the $H^+$ efflux.

## Training Implications on Bioenergetics of Fatigue

Though the PCr reserves may not be influenced through the physical training, it would be possible to enhance the muscle glycogen stores significantly to enhance the glycolytic energy resources for sprint cycling performance. Since, the PCr reserves of the skeletal muscle varies from person to person, the training implication is still significant in enhancing the PCr levels within the maximum limits and maintain these reserves throughout the competitive season (Hickner et. al. 2010). High intensity to very high intensity sprint cycling repetitions of optimal duration are essential to enhance and maintain the PCr and muscle Glycogen reserves (Glaister 2005). Glycogen supercompensation could be achieved through exhaustive anaerobic glycolytic workouts which drain the muscle glycogen reserves to the very minimum (Siegler et. al. 2006). When the muscle glycogen reserves are very minimal, post high intensity sprints the glycogen sysnthase activity is more and hence the resting muscle glycogen values may be enhanced. The PCr reserve in the muscle may not be appreciably increased beyond the normal resting levels, except by oral ingestion of creatine appropriately post sprint exercises (Herda et. al. 2009), this system needs less training concentration, as the training for muscle glycogen is sufficient. Sustained sprint cycling at very high intensity, like in 500 to 1000 meters sprinting, needs much emphasis on anaerobic glycolytic flux and lactate flux with some emphasis on the PDH activity. Endurance trained individuals of elite in nature showed higher MCT 1 and also MCT 4 when compared to less endurance trained individuals (Bentley et. al. 2009). Also the lactate transport capacity was found as significantly higher in individuals with accumulated high level endurance training (Vo2 max at least 70 $ml.min^{-}1.kg^{-1}$). Endurance training seems to elevate the MCT 1 isoform transporter more significantly causing higher lactate transport capacity. MCT 1 content could be enhanced by 18% to 61% by endurance training of high volume with sufficiently moderate intensity, whereas MCT 4 though can be increased through high volume of endurance type exercise, there are large inter individual differences. But, MCT 1 and MCT 4 content has been reported to increase by 15% to 120% and by up to 60% respectively, in response to high intensity training. Hence, sufficiently high intensity endurance training for large durations needed for enhancements in MCT 1 content whereas high to very high intensity physical exertion is essential to bring sufficient enhancements in MCT 4 content (Green et. al. 2008). Fatigue indices measured during continuous, 1-min, all-out and intermittent (repeated 10-s cycling sprints interspersed with 30 s of recovery) supramaximal exercise, were inversely related to MCT1 content, but not to MCT4 content, in 15 humans with a different training status. Significantly highest lactate transport capacities

were observed in two track cyclists (>100 $pmol \cdot cm^{-2} \cdot s^{-1}$), who competed in the 4-km pursuit (one obtained a bronze medal at the 1992 Olympic Games) and who combined training on the road with high-intensity track training ($\dot{V}O_{2\,max} \sim 78\ ml \cdot min^{-1} \cdot kg^{-1}$). The results of these two cross-sectional studies support the hypothesis, that very high volume endurance based very high intensity cycling training will enhance the lactate transport capacity significantly leading to excellent performances in sprint cycling (Burgomaster et. al. 2005) of up to one kilometre. The possible mechanism may be increase in lactate and proton efflux from working myocytes and consequent decline in intracellular $p^H$. Also, the proton coupled lactate efflux occurring at type II fibers, and effective transfer of this lactate and uptake by the adjacent type I fibers and concomitant decrease in both interstitium and plasma $p^H$. Different phenomena of the oxidative phosphorylation, plasma membrane potential and proton motive force are influenced by the endurance training of sufficiently long periods of time. This would lead to favourable conditions for higher ATP turnover and substrate oxidation effecting enhanced aerobic status.

A high volume of endurance training with sufficient intensity coupled with high intensity intermittent cycling sprinting extending to competition time will elevate the lactate transfer capacity and this is optimally observed only those who worked out with high volumes of endurance cycling (minimum of $VO_{2\,max} < 68\ ml^{-1} \cdot kg^{-1} \cdot min^{-1}$) had an elevated lactate and not among those whose $VO2_{\,max}$ is less than $68\ ml^{-1} \cdot kg^{-1} \cdot min^{-1}$ supports this.

## Conclusions

Sprint cycling for one kilometer is an activity which could last for about one minute and a few seconds at world class performances. This high intensity or supra maximal cycling effort requires energy contribution from both anaerobic phosphorylation and aerobic phosphorylation, though during the first forty seconds or so, the main contribution is anaerobic and during the last twenty or twenty five seconds aerobic metabolic pathway also contributes energy. Hence, trianing should aim to optimise both the metabolic systems and the emphasis must be on enhancing the resting muscle PCr reserves, resting muscle glycogen reserves to enhance the anaerobic metabolism and optimising the enzymatic turnover for better aerobic performance. Simultaneously, it is also essential to enhance the lactate/proton bidirectional cotransport capacity of the cyclist to resist fatigue, which is highly essential during the last stages of the sprint time trial for one kilometer. It has been observed, that postponement of fatigue is more important, which has much impact over conserving the muscle contraction power output, thereby recording excellent timings.

## Recommendations

Sprint cyclists, need to train on roads more extensively to build up an aerobic endurance base, which will enhance the MCT 1 optimally, along with MCT 4 in palpable quantities, which will make individuals more fatigue resistant during the later stages of the sprint cycling especially during the last few seconds of the time trial. Since, MCT 4 content can be significantly improved with high intensity to very high intensity bouts of cycling, it is essential to include supra maximal bouts of sprint cycling of smaller durations like fifteen to thirty seconds in repetitions of complete rest. Sub maximal sprint cycling for longer distances than kilometer enhances both aerobic and anaerobic glycolysis pathways. Sub maximal sprint cycling over one kilometer but not more than two kilometers would enhance the GLUT 4 translocation more vigorously leading to enhanced glycolysis with better glucose availability to muscles, also this kind of sustained sprinting repetitions with incomplete recovery would also super compensate both the PCr and muscle Glycogen reserves, leading to further enhancement in the glycolytic phosphorylation. Hence, even during the competition period, sprint cyclists of one kilometer time trial need to perform road cycling of sufficiently long distances like twenty five to forty kilometers with sub maximal intensity frequently to keep the lactate transport capacity at very high to resist fatigue during the last stages of the sprint cycling time trial. One session of cycling targeting the glycolytic phosphorylation enhancement needs to be included, like 1.5 kms to 2 kms of sub maximal sprint cycling repetitions with insufficient recovery. One session of supra maximal intensity of sprint cycling covering distances of only 400 meters to 600 meters with complete recovery targeting the MCT 4 content.

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