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Con: Live High–Train Low Does Not Improve Sea-Level Performance Beyond that Achieved with the Equivalent Living and Training at Sea Level

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The live-high train-low (LHTL) model is postulated to improve sea-level performance beyond that which can be achieved by reciprocal training near sea level alone (live low–train low, LLTL) by residing at moderate/high altitude and training at low altitude (Levine and Stray-Gundersen, 1997). The assumptions inherent to the LHTL postulate are: 1) Physiological adaptations to moderate/high altitude are beneficial to sea-level performance; 2) Hypoxic training-induced adaptations to sea-level performance are subordinate to those obtained with normoxic training; and 3) The desired physiological adaptations specific to acclimatization can and do reliably occur in elite athletes. These inherent assumptions of the LHTL model are either unsubstantiated or prove invalid.

1) The "live-high" leg of LHTL is designed to induce specific adaptations to hypoxia that would improve sea-level exercise performance (Levine and Stray-Gundersen, 1997). Adaptations to hypoxic exposure and/or acclimatization, however, are widespread and may result in depressed immune function and increased risk of infection (Bailey et al., 1998; Brugniaux et al., 2006a; Tiollier et al., 2005), lower quality of sleep (Kinsman et al., 2005a, 2005b; Pedlar et al., 2005), and facilitate a loss of skeletal muscle oxidative capacity (Hoppeler and Vogt, 2001), specifically oxidative phosphorylation capacity (Jacobs et al., 2012), which has been identified as a primary determinant of endurance performance near sea level in elite athletes (Jacobs et al., 2011). These adaptations to hypoxic environments, which by no means represent an inclusive list, all threaten sea-level performance. The detrimental effect of acclimatization on sea-level exercise performance is clearly demonstrated in the seminal LHTL study (Levine and Stray-Gundersen, 1997) where the subjects that both lived and trained at moderate altitude (2500–2700 m) had an increase in red cell volume (RCV; ml/kg) and an improved sea-level Vo_{2max} however failed to improve sea-level 5k performance. These observations were interpreted as a side effect from the inability to maintain sufficiently high work rates and training velocities to maintain competitive fitness (Levine and Stray-Gundersen, 1997). Next, however, I demonstrate how hypoxic training *per se*, even with depressed work rates and/or training velocity, fails to impair sea-level performance. Alternatively, these observations demonstrate the detrimental effects of acclimatization on sea-level performance, even despite an increase in RCV (Levine and Stray-Gundersen, 1997).

2.) The "live-low" leg of LHTL is designed to counter the alleged the inability to maintain sufficiently high work rates and training velocities at altitude to maintain competitive fitness (Levine and Stray-Gundersen, 1997). Hypoxic training, however, does not consistently demonstrate an adverse effect on sea-level performance (Dufour et al., 2006; Gore et al., 1998; Hoppeler et al., 2008; Neya et al., 2007). When controlling for a potential placebo/nocebo effect of hypoxic training by blinding two-groups of elite athletes, one that trained in hypoxia and the other strictly normoxia, there was no difference in normoxic performance despite a reduction in absolute training intensity specific for those subjects that trained in hypoxia (Truijens et al., 2003). Thus, hypoxic training *per se* does not necessarily hinder sea-level performance.

3.) The primary reductionistic mechanisms put forth to explain any purported improvement in sea-level performance with LHTL have been attributed either to an increase in oxygen carrying capacity via an increase in total hemoglobin mass (nHb) (Levine and Stray-Gundersen, 2001; Wilber et al., 2007) or to an improvement in mechanical efficiency during exercise (Gore et al., 2007). Group mean increases in nHb with LHTL have been the more consistent adaptation with LHTL (Robach and Lundby, 2012; Robertson et al., 2010), although there is marked individual variation (Chapman et al., 1998; Robertson et al., 2010; Siebenmann et al., 2012), and measurable increases are not always apparent (Neya et al., 2007; Siebenmann et al., 2012). Even a longer duration of chronic terrestrial hypoxic exposure to a higher elevation than was initially presented (Levine and Stray-Gundersen, 1997) failed to increase nHb in elite cyclists (Gore et al., 1998). A recent meta-analysis revealed that hypoxic-mediated erythropoiesis occurs slowly with the time dependency of erythropoeisis firmly reliant on i) the elevation of exposure, and ii) initial nHb (Rasmussen et al., 2013). Accordingly, elite athletes, a population with an already elevated nHb (Robach and Lundby 2012), would likely need to spend even more time at a respective elevation to successfully expand nHb (Rasmussen

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et al., 2013). Thus, the potential for LHTL to improve oxygen carrying capacity in elite athletes lacks probability (Rasmussen et al., 2013; Robach and Lundby 2012) and reliability/consistency (Gore et al., 1998; Neya et al., 2007; Siebenmann et al., 2012). In regards to enhanced mechanical efficiency during exercise with LHTL, while there is evidence of improved electron coupling efficiency in skeletal muscle with high-altitude acclimatization in untrained individuals (Jacobs et al., 2012), the possible extrapolation of that data into functional alterations in whole body mechanical efficiency has been invalidated (Lundby et al., 2007; Siebenmann et al., 2012).

Finally, and most importantly, the literature studying LHTL does little to support its claim of improving sea-level performance above that obtained with equivalent LLTL. Studies that all together lack a proper LLTL control group regarding exercise performance (Clark et al., 2009; Garvican et al., 2011; Stray-Gundersen et al., 2001; Wehrlin et al., 2006) are unable to confirm any benefit of LHTL over LLTL. Hitherto, primarily one study reports data that could reasonably be interpreted as an improvement in sea-level performance with LHTL compared to a complementary sea-level group of athletes (Levine and Stray-Gundersen. 1997). This study, unfortunately, did not blind either the researchers or the subjects and therefore cannot discount potential biases nor can they account for confounding effects of either a placebo or nocebo effect on the outcome of the study. Alternatively, there are multiple other nonblinded studies across various laboratories that have been unable to verify any actual improvement in sea-level exercise performance following LHTL over the complementary LLTL control groups (Brugniaux et al., 2006b; Neya et al., 2007; Robertson et al., 2010; Saunders et al., 2010). Additionally, the only study to date that has examined the efficacy of LHTL using a placebo-controlled, double-blind experimental design in elite athletes found that 4 weeks of 16 h/day spent in hypoxia corresponding to an altitude of 3000 m, well within the recommended dose of LHTL (Wilber et al., 2007), not only failed to improve exercise performance in the LHTL group but also failed to disseminate any difference from the control group that lived and trained at low altitude (Siebenmann et al., 2012). Moreover, one potential limitation of the Siebenmann study (2012) may be the modest statistical power achieved. This power could have silenced some of the smaller effects observed, such as the 3% greater improvement in time trial performance measured in the placebo versus the LHTL group (a statistically nonsignificant difference) over the 4 week span. The possibility of these type II errors should be acknowledged.

In summary, the basic premise of LHTL is flawed, the primary mechanisms proposed to improve sea-level performance repeatedly fail to demonstrate any reliability, and, most importantly, the empirical evidence of LHTL as an effective training modality to sea-level performance beyond that achieved with the equivalent living and training at sea-level alone is at best equivocal.

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