

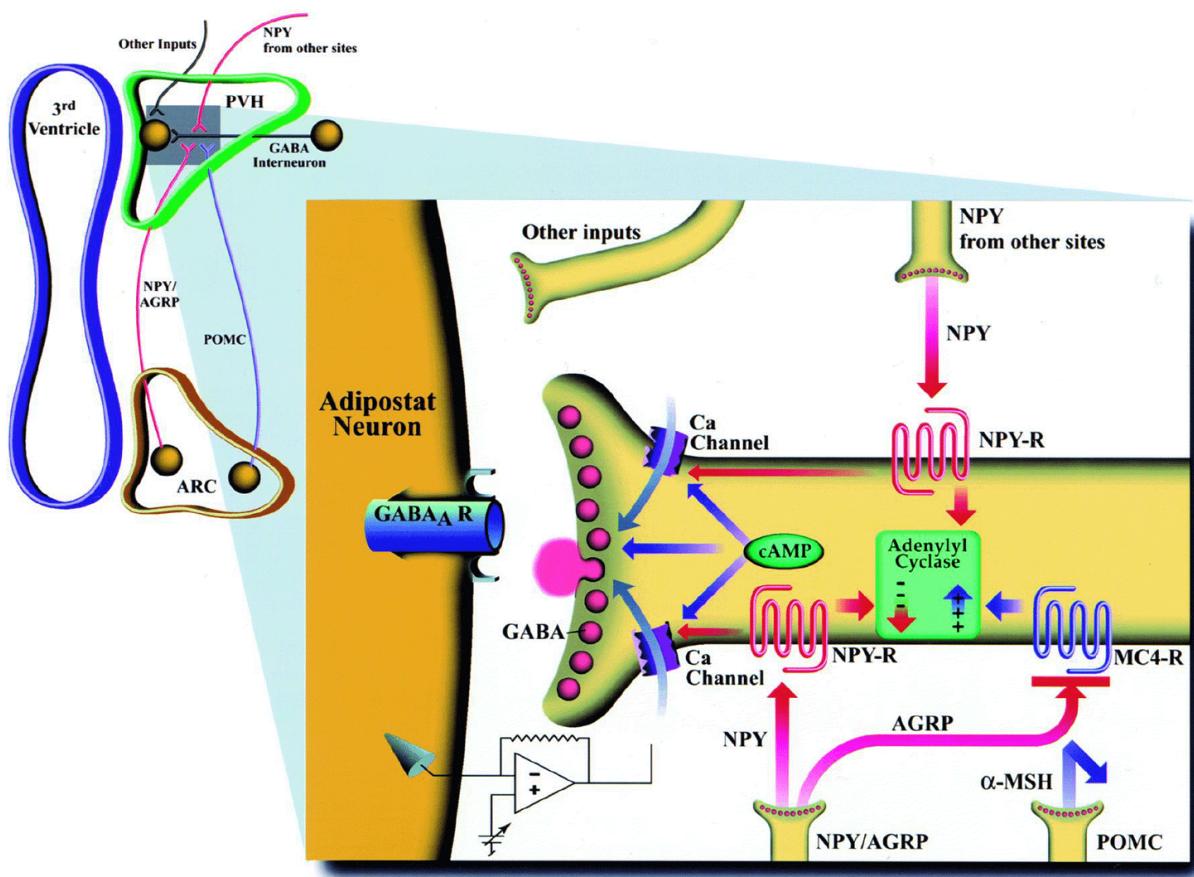
# Post-competition Perils: Hyperphagia, Adipose Overshoot, & Dysphoria

A curioso from the before times.

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Hypothalamic arcuate nucleus-paraventricular nucleus axis (ARC-PVN axis)

*Note to readers: This essay is a piece I wrote from my pre-doctoral days that I thought would be worth sharing to my readership. It was originally published on an old interenet formum for bodybuilding and training known as “Avant Labs” and Google searches may turn up the original essay post or re-post on training or bodybuilding-related web boards. As some of my readers may be aware, physical training and nutrition—and more broadly the field of psychoneuroendocrinology—have always been a core interest of mine as both a scholar and an athlete.*

It is common for bodybuilders and figure competitors to engage in a period of dysregulated behavior characterized centrally by repetitive, uncontrollable hyperphagia and bingeing in the days immediately subsequent to competition or to their lowest achieved adipostatic level. While the eating behavior is by and large the central component of this 'post-competition syndrome', it is part of a larger phenotype which includes both 'bodyfat overshoot' (Dulloo et al., 1997) and a general dysphoria characterized by a multiplex of feeling/behavioral states including emotional and/or mental discomfort, restlessness, malaise, depression, and sleep fractionation.

Extending my work on dynamical systems and more generally adaptation at lowered adipostatic states, this article is a specific contribution discussing a more acute time-period (post-competition) and highlighting a subtle paradox in that the worst symptoms of severely reduced adipostatic states are not necessarily during them, but rather on the *slippery slope* out of them.

It can be argued that most of the bodybuilding community, while noticing the symptoms noted above during repeated competition cycles, are unaware that they are in fact physiologically rooted adaptive processes (from a purely physiological standpoint). In addition, the claim is advanced here that training and supplementation post-competition may be even more important than training pre-competition since it is during this post-competition period that significant declines in physique and mental resilience occur *beyond* where they stood before pre-competition preparation was fully set in motion.

Originally noted by Keys et al. (1950) in the infamous Minnesota Starvation experiment and subsequently corroborated by the Dulloo lab in a number of papers that re-analyzed the original Minnesota data, there was a marked tendency for individuals who underwent the starvation period (or a protracted period of hypoenergetic caloric intake) to engage in voracious hyperphagia and demonstrate significant body fat gain following release from the starvation requirements. Indeed, because the body-fat gain following starvation exceeded the absolute adipostatic levels prior to the deficit (starvation) period, Key's et al. (1950) termed the phenomenon 'post-starvation obesity' while the Dulloo group (Dulloo, et al., 1997) termed the phenomenon "post-starvation hyperphagia and body-fat overshoot." If we consider the bodybuilder or fitness enthusiast's 'pre-contest' dieting akin to that of the 'starvation period' of Key's et al. (albeit with some notable dissimilarities including resistance training and extensive supplementation use) we can begin to understand the 'post-contest' period, and the normative metabolic and behavioral-psychological reactions that often cause great distress for the passionate bodybuilder (natural or not) or fitness enthusiast.

An account retold from one of Keys' original participants in the Minnesota experiment reflect what has been related to me time and again in numerous private messages as well observed myself in various individuals who I have trained with (both male and female) and echo reports of 'post-competition binges':

**"Although they were warned to be careful not to overeat on d 1 [following the experiment], they were free to eat as they wished. H.S. remembered**

**being taken to the hospital to have his stomach pumped because he ‘just simply overdid’” (Kalm & Semb, 1997, p.1351).**

As a group, the Minnesota subjects as well as others who have undergone significant body weight reductions during extended periods of energetic deficit (cf. St. Pierre et al., 1996) show persistence in both appetite and hyperphagia *long after refeeding commences*. This combination of both increased appetite and subsequent hyperphagia was termed ‘integrated hyperphagia’ by Dulloo et al. (1997). Once again, persistence in binge behavior and the associated mental dysphoria with losing the contest physique is a common phenotype of bodybuilders and fitness (figure) competitors. One personal recount told to me indicated that one such person both put on substantial fat (past pre-competition prep) following competition but also suffered from notable socioemotional disturbances, including, but not limited to, depression and social anxiety, which ultimately lead to the inability to maintain a romantic relationship.

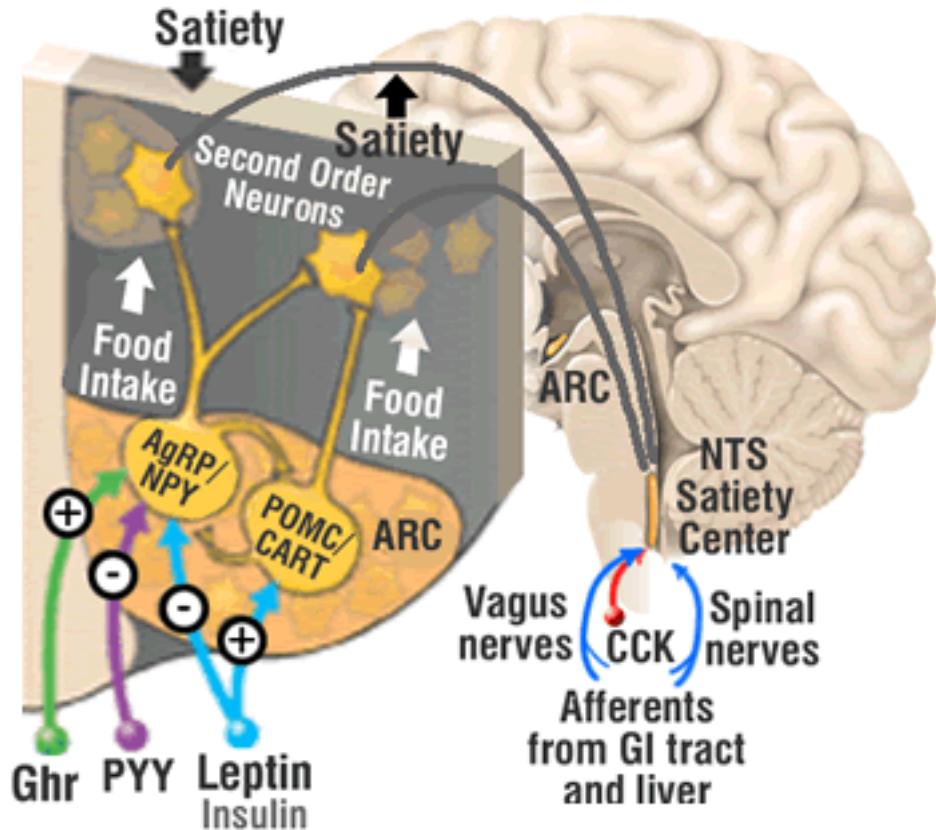
Taken together, the elegant work of Keys and colleagues as well as Dulloo and colleagues should clue the training community in that following bouts of contest dieting, counterregulatory processes will soon ensue that lead to behavior which can seriously threaten maintenance of even pre-competition dieting conditioning levels. As such, the post-competition period becomes a very critical time when, I argue, that training and supplementation usage must be dialed in. Before I offer a few brief, flexible recommendations, there are a few extremely important details that must be noted with regard to the re-analysis of the Minnesota starvation data by the Dulloo group.

In re-analyzing the Minnesota starvation data, Dulloo’s group found at least two important variables in the characterization of the post-starvation integrated hyperphagia. The first being that even in spite of increased dietary induced thermogenesis (DIT) due to the sympathomimetic actions of excessive carbohydrate loading (i.e., bingeing) there remains a specific metabolic component favoring fat storage (1990). Thus, even in the presence of increased DIT (due to metabolic normalization as weight is gained) adipose tissue can be laid down simultaneously. Dulloo and Girardier (1990) noted that the metabolic component represented a net 15% reduction in energy expenditure during refeeding. Dulloo (1997) further notes that the phenomenon of fat increase occurring more rapidly than that of active tissues has been noted since the “turn of the century in adults recovering body weight after diseases or famines” (p. 30) and cites both Jamin & Muller (1931) and Kornfeld & Schuller (1931). While a more comprehensive overview of their models, including that of the P-ratio (Dulloo & Jacquet, 1999) are outside the scope of this article, the key point of the foregoing discussion is that *fat overshooting* is in large part determined by delayed protein repletion (loss of lean-tissue), which in turn results from a suppression of thermogenesis favoring the ‘replenishment’ of adipose reserves (Dulloo, 1997).

The second variable involves the predictive ability of both adipose and lean tissue loss to promote the post-starvation integrated hyperphagia. In an elegant set of regressions, Dulloo et al., 1997) showed that while adipose loss was the strongest predictor of integrated hyperphagia, lean tissue loss also added unique variance (albeit of small magnitude). These correlations were still present even after controlling for degree of prior energy deficit.

Considering the foregoing, it is quite clear that the post-competition phenomenon of integrated hyperphagia is a relatively ubiquitous phenomenon among those who have lost substantial body mass. However, the component of mental dysphoria is an addition this author has contributed independently (as a result of both personal and vicarious experience), and we can conceptualize both the integrated hyperphagia and the mental dysphoria as the “post-competition syndrome.” I have written previously on psychoneuroendocrinological contributions to the post-competition phenotype (cf. Adipose Reduction and Bodyfat Setpoint: A Dev. Reg. Model) and will elaborate on this component of the post-competition syndrome in future work. Suffice it to say that included in the dysphoric component are depression, restlessness, fatigue, sleeplessness, and most importantly social withdrawal. So, from a training standpoint, how can the trainee deal with the ‘post-competition syndrome’?

It would seem that a simple understanding of the post-competition period, as presented here, will go a long way to treating this period of training more appropriately. More specifically there are at least four specific areas where trainees and competitors can directly address the ‘post-competition syndrome’:



**1) Psychological:** While acute bingeing post-competition is of the involuntary subtype, it is argued that subsequent bingeing (still part of the integrated hyperphagia matrix) is more voluntary in nature and psychologically triggered. Psychological triggers are more likely to become effective at triggering binge patterns in light of the underlying post-starvation vulnerability (cf. Adipose Reduction and Bodyfat Setpoint: A Dev. Reg. Model). In addition,

individual differences in affect regulation (socioemotional adaptation, attachment) also may play a key role (for comparative evidence see Hancock, Menard, & Olmstead, 2005). Awareness of psychological cues that trigger bingeing and insuring that you take proactive steps to re-engage in adaptive, positive social situations (e.g., going out, interacting with others) may short-circuit the feed-forward loop of ‘social isolation-binge alone’.

**2) Training:** At least two prominent considerations come to mind. First, and of obvious import, the more lean mass retained during the dieting period, the more potential for reductions (albeit subtle) in the experience of integrated hyperphagia. It would seem that a strong focus on time under tension (TUT) should be a core component of both the pre and post competition periods. Second, cardio should be maintained or even increased during the post-competition period to both compensate for involuntary bingeing as well as to perhaps inhibit the metabolic component that appears designed to facilitate fat storage (a more detailed discussion will be presented in a separate paper). Finally, an immediate change in lift scheme is advised, as this will ‘refresh’ up the routine and prevent needless iterations of the mental fatigue of the same routine, which can lead to an avoidance of the gym (isolation) and subsequent bingeing as discussed above.

**3) ‘Binge’ Nutrition:** It may be profitable to create a ‘fixed’ binge pattern such that, even during times of involuntary binges, there are only certain foodstuffs that you are allowed (provided they enable the quiescence of the integrated hyperphagia and psychological craving). For example, during times of integrated hyperphagia, this author has been able to fix his foodstuffs at fat-free frozen yogurt and specifically (only) butter popped corn cakes (which contain no HFCS). This may be one factor that has attenuated the consequence of integrated hyperphagia. Often we hear of binges on candy, chocolate, pizza and other calorie dense high-fat, high-carbohydrate foods which most certainly will elevate the energy intake.

**4) Thermogenic Supplementation:** A few brief, non-exhaustive comments here. Thermogenic supplementation, such as the EC(A) stack (and others) should be continued, possibly increased during this phase (consider 40mg ephedrine/day rather than 20mg). The reasoning is the same mentioned above in regards to cardio—that being the potential to short-circuit the metabolic component favoring fat storage in spite of increased DIT. Of course, abuse of any thermogenic during the dieting phase will reduce any gains one might make by increasing the dosage during the post-competition phase. That said much of the diet phase can proceed without the use of thermogenic aids by instantiating and maintaining a modest caloric deficit using both food restriction and increased energy expenditure.

While I am torn on advocating the use of nicotine in this context, I will indicate that its continued usage through both dieting and post-rebound phases may exert favorable body composition changes and maintenance. One study (Schwid, Hirvonen, & Keesey, 1992), albeit in rats, is particularly relevant as it was framed in a *regulatory perspective* and it showed that nicotine treated rats were able to reduce their setpoint, but also that they showed the same hyperphagic phenotype as control rats when they were starved beyond this new ‘setpoint.’ However, rather than ‘binge back up’ to control levels of absolute fat, these rats overshot their new ‘reduced setpoint’ briefly, eventually returning to this new ‘setpoint’ which was lower than their control (pre-nicotine) control values. This provides comparative evidence for the

hyperphagic phenotype discussed here in humans as well as the energy conserving adjustments both during food restriction which spill over into the refeeding period. I would be remiss not to indicate that one must obviously reflect on the method of nicotine administration as well as the noted risks to its usage.

Here I have discussed the normative phenomenon of integrated hyperphagia observed in individuals who have experienced significant and extended periods of caloric deficit resulting in a substantial loss of bodyweight. I then applied this portrait to that of the body-builder or figure competitor following the competition period. I then offered more specific details regarding variability in integrated hyperphagia and created a new term, specific to this broad sport, called the '***post-competition syndrome.***' Finally, I have offered some brief recommendations regarding training, hyperphagic nutrition, and post-competition thermogenic supplementation. I have intentionally made an attempt to keep this contribution less 'academic' in tone while still preserving a highly academic backdrop (i.e., Keys et al., and Dulloo et al.).

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