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Calcium homeostasis may influence resting energy expenditure with effects most apparent in early pubertal girls

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Abstract

Aim—Perturbations in dietary and hormonal components of the calciotropic network may be mediated through the influence of calcium homeostasis on resting energy expenditure (REE). We investigated the association of dietary and hormonal factors involved in the regulation of calcium homeostasis with REE in girls.

Methods—Thirty-six girls age 7–11 years participated. REE was assessed by indirect calorimetry, and body composition, dietary intake (calcium, vitamins D and K, phosphorus), and serum hormones (PTH, osteocalcin, 25OHD), were evaluated by DXA, 24h recall and serum assay, respectively.

Results—A positive association between vitamin K and REE and an inverse association of PTH with REE ($p=0.05$) was observed. PTH and REE were positively related in those having normal adiposity ($p=0.03$) and inversely related in those with excess adiposity ($p=0.01$). The association of REE with vitamin K intake was evident in lean individuals ($p=0.001$), but was null in those with excess adiposity.

Conclusion—Decreased calciotropic hormone levels along with increased related nutrient intakes were associated with greater REE, although these relationships differed according to adiposity. The physiologic response to the diet and subsequent energy partitioning needs to be considered in the context of puberty. In particular, regulation and signaling of the calciotropic network during pubertal maturation warrant investigation.

Keywords

Calcium homeostasis; puberty; diet; adiposity; calciotropic

Due to the substantial involvement in overall energy balance, particularly obligatory growth-related processes, it is conceivable that maintenance of calcium homeostasis plays a pivotal role in resting energy expenditure (REE). Beyond the typical osseous function ascribed, calcium is an essential component of cellular metabolism, body tissue anabolism and repair, electro-conduction of the heart and systemic function (1, 2). The intricate involvement of calcium requires various energy- and cofactor-dependent regulators to ensure calcium is kept within a tight range. These include ingestion and subsequent metabolism of other key nutrients, such as phosphorus and vitamins D and K, well as hormonal signals, including

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parathyroid hormone (PTH) and 25, hydroxy D (25OHD), which interpret and respond to limit serum calcium fluctuation. Together these dietary and hormonal components comprise the “calciotropic” network. Despite the involvement in compulsory basal function, to our knowledge, no study has investigated the extent to which components of this network contributes to REE.

Undoubtedly the most important factor influencing calcium homeostasis is diet. Although adequate intake and subsequent hormonal response directing absorption, utilization and storage contributes to maintenance of circulating calcium levels, achieving adequacy in other nutrients involved in the calciotropic network is also essential for optimal capacity of calcium retention. The pubertal growth spurt and corresponding physiologic adaptations represent a sensitive window in which perturbations in calcium homeostasis may have the greatest impact on body composition trajectories. Indeed, skeletal maturation accelerated with pubertal onset represents a highly energy-dependent series of events. Related to the integral participation in both energy partitioning and growth, dysregulation of the calciotropic network conceivably exerts significant influence on body tissue partitioning, particularly the reciprocal relationship between bone and fat mass accrual (3). REE drives the myriad of biochemical processes necessary to maintain developmental processes. The obligatory requirement for calcium at the cellular, tissue and systemic level permit the plausibility that disturbances in the calciotropic network during puberty may alter body composition via effects on REE. Calcium intake, as well as other key dietary nutrients, especially among peri-pubertal girls is commonly inadequate (4). Thus, imbalances in the calciotropic network may alter physiological processes underlying REE and increasing risk of adverse outcomes related to body composition, such as lower bone mass and greater fat mass (5). To this end, the objective of the study was to evaluate relationships of body composition, the calciotropic network, and potential contribution to REE in peri-pubertal girls.

Patients and Methods

Data on 36 girls ages 7–12 years (pubertal stage 3) recruited from the Birmingham, Alabama area as a part of a larger cross-sectional (6) were used for current analyses. Girls were healthy and not on medications known to affect body composition. Parents and girls provided consent/assent, respectively, after reviewing the protocol with study personnel. The protocol was approved by the Institutional Review Board for human participants at the University of Alabama at Birmingham (UAB). All measurements were performed between 2005 and 2008.

Protocol

Participation required two visits within thirty days of each other. On the first visit, reproductive status, anthropometric assessment and body composition were measured, and a 24-hour dietary recall was obtained. On the second visit, participants were admitted to the General Clinical Research Center for an overnight stay (ensuring ~10-hour fast) and a second 24-hour dietary recall was obtained. Upon completion of the overnight fast, REE assessment and metabolic testing was performed as described elsewhere (6).

REE

REE was measured via computerized, open-circuit, indirect calorimetry system with a ventilated canopy (Delta Trac II; Sensor Medics, Yorba Linda, CA). One-minute average intervals of oxygen uptake (VO_2) and carbon dioxide production (CO_2) were measured continuously for 30 minutes, in which the last 20 were used to calculate energy expenditure.

Body Composition Assessment

Whole-body dual energy x-ray absorptiometry (DXA; GE Lunar Radiation corp., Madison, WI) with pediatric software (version 1.5e) was used to assess body composition variables.

Serum Assays

PTH, 25OHD and OC were obtained from fasting sera drawn and assayed in the UAB Core Laboratory. Serum PTH was assessed by a two-site immunoradiometric assay, 25OHD with liquid chromatography/ tandem mass spectrometry technique and total osteocalcin (OC) using radioimmunoassay. The intra-assay c.v.'s for the analysis of PTH, 25OHD and OC were 7.76, 4.83, and 5.36, and the mean inter-assay c.v.'s were 2.07, 4.94, and 5.76%, respectively.

Dietary Assessment

Total energy (kcal/d), calcium (mg/d), vitamin D (mcg/d), vitamin K (mcg/d) and phosphorus (mg/d) intake were assessed using the average of the two 24-hour dietary recalls, conducted using the “multiple pass” method. Cup and bowl sizes were provided to help estimate portion sizes. Each recall was performed in the presence of at least one parent. A registered dietitian coded and entered the data into Nutrition Data System for Research version 2006 (Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN).

Reproductive Status

Pubertal progression is associated with characteristic influences on body composition and energy metabolism. The one-to-five staging of Marshall and Tanner, based on examination of both breast and pubic hair development, was used for pubertal stage assessment, with one composite number representing the higher of the two assigned values (7).

Anthropometric Measures

Anthropometric measures were obtained by the same registered dietitian. Height (Heightronic 235; Measurement Concepts, Snoqualmie, WA) and weight (Scale-tronix 6702W; Scale-tronix, Carol Stream IL) were obtained in minimal clothing without shoes. BMI percentile was calculated using age-specific growth charts.

Genetic Admixture Analysis

Inherent inter-population differences in calcium utilization, body composition and REE may confound data interpretation. Accordingly, genetic admixture was included as a genomic control variable representing population-based differences for statistical analysis. Briefly, a panel of 140 ancestry informative markers was used to estimate the genetic admixture proportion of each subject, using maximum likelihood estimation based on an algorithm described by Hanis et al (8). European genetic admixture, the most widely distributed component across racial/ethnic groups in this sample was used in analyses.

Physical Activity (PA)

The MTI Actigraph accelerometer (Actigraph GT1M – Standard Model 198-0100-02, ActiGraph LLC, Pensacola, FL, and accompanying software) was used to measure PA level and pattern for seven days prior to admission to the GCRC. Epoch length was set at one minute and data was expressed as counts per minute. Children were instructed to wear the monitor at the waist above the right hip, allowing removal for only sleeping, bathing and swimming. Daily and total counts per minute were summed and averaged.

Statistical Approach

Mean descriptive values for the overall sample and adiposity level (using body fat percent cut-off of 30, the standard value for females at which it is considered to be in excess (6, 9) were analyzed and differences were investigated using ANOVA. Multiple linear regression was used to evaluate the relationships between REE, body composition and dietary and hormonal components of the calciotropic network. The early pubertal period (Tanner 3) in girls represents a period of simultaneous fat and bone mass accrual. As bone and fat emanate from the same pluripotent stem cell lineage, allocation of resources (calcium and energy) during this period is critical (10, 11). Accordingly, level of adiposity (based on percent fat cut-points) may be relevant. Analysis was therefore conducted by percent fat-cut points. Although these values represent arbitrary values, this level of adiposity has been reported to be associated with adverse health outcomes in this population. Models investigating the contribution of nutrient intake on REE were additionally stratified by adequacy according to DRI recommendations, using 1300mg for calcium, 5 mcg for vitamin D, 60 mcg for vitamin K and 1250 mg for phosphorus (12). Inclusion of total energy intake as a control variable served to account for the correlation between micronutrient and overall caloric intake. All models were adjusted for European admixture, pubertal stage, fat and lean mass. The nominal variable pubertal stage was orthogonally coded for regression analysis. Contribution of PA to REE was investigated, but was not identified as significant so was excluded from final models.

Results

Sample characteristics are displayed in Table 1. There were no differences by adiposity level beyond that of weight/body composition variables, including weight, BMI percentile, and total percent fat. Evaluation of the contribution of calciotropic hormone analysis to REE revealed an inverse association of REE with PTH ($p=0.05$) and a trend for an inverse relationship of REE with OC ($p=0.09$). Investigation of associations between calciotropic dietary variables and REE revealed a positive relationship between vitamin K intake and REE ($p=0.0001$).

Although there were no differences in either mean calciotropic variables by adiposity level, multiple regression analysis using stratification by percent fat cut-offs revealed PTH and REE were positively related in those having normal adiposity ($p=0.03$) and inversely related in those with excess adiposity ($p=0.01$) (Table 2; supp). The positive association of REE with vitamin K intake remained in lean individuals ($p=0.001$), but was no longer significant in those with excess adiposity. A trend towards a positive association between phosphorus and REE was observed only in those with excess adiposity ($p=0.09$).

Subsequently, stratification of the overall sample by dietary adequacy revealed a positive association with REE and vitamin K irrespective of dietary adequacy ($p=0.001$, adequate; $p=0.05$, inadequate). ANOVA analysis (Figure 1) revealed marginal associations between REE and calcium in those meeting or exceeding recommendations ($p=0.08$) and vitamin K ($p=0.07$) intake compared to those who did not.

Discussion

REE accounts for approximately 65% of total energy expenditure and body composition is the main driver of REE. At an average intake of 1800 kcal/day, particularly in the circumstance of low physical activity, low REE during growth would tilt the energy fulcrum towards storage in the form of adipose tissue. Thus, trajectories established during puberty have profound impact on long-term energy balance. Calciotropic factors are integral in metabolic pathways directing fuel utilization and body tissue partitioning (e.g. fat vs. bone),

and therefore ultimately energy balance. The skeleton, as the body's calcium reservoir, requires substantial energy allotment vital for optimal physiologic function (13–15). We hypothesized that dietary intake and hormonal regulation by the calciotropic network influences energy balance through its effects on REE. Analysis of the contribution of individual calciotropic components revealed vitamin K and PTH as contributors to REE. However, level of adiposity exerted a differential impact upon the relationships. It is plausible that adequate intake and absorption of dietary nutrients involved in calcium maintenance may optimize mechanistic pathways involved in REE. Further, the maintaining calcium homeostasis and consequent energy resource partitioning, particularly during puberty, represents a potential strategy for optimizing body composition trajectory.

The relationship between vitamin K and REE is not surprising given the role of vitamin K in a number of physiologic processes including bone remodeling. A consistent line of evidence in human and animal studies clearly demonstrates that vitamin K influences bone health (16–18). Acting as a coenzyme, vitamin K mediates the conversion of glutamate to gamma-carboxyglutamate which is essential to facilitate calcium incorporation into hydroxyapatite crystals, thus mineralizing bone (19). Particularly salient during skeletal maturation, vitamin K activity involves an increase in both bone deposition and resorption to ensure structural integrity (20). As an anabolic process directing calcium deposition into bone, we hypothesized that during skeletal maturation, higher vitamin K intake would lead to an up-regulation of REE for bone growth.

It is well-established that serum calcium levels are inversely associated with PTH (2), a hormone which may influence energy requirements directly and indirectly through body composition alterations. The energy requirement of skeletal maturation is regulated by bone turnover. Optimization of skeletal integrity necessitates a coupling between deposition and Resorption. Consistent with existing literature, PTH was inversely associated with REE ($p < 0.05$) in this cohort. Serum PTH increases in circulation secondary to dietary insufficiency of calcium and related nutrients, serving to regulate calcium (2). Whereas chronic elevation of PTH inhibits balance in the bone modeling process, thereby down-regulating REE and increasing adipogenic pathways intermittent PTH, characteristic of calcium homeostasis is associated with increased bone remodeling and consequent attenuated adipose tissue accrual (21).

Adiposity may alter the contribution of dietary adequacy to resource allocation between tissue compartments, highlighting the importance of body composition in terms of both fat and bone in the metabolic regulation of calcium homeostasis. Interestingly, after stratification by percent fat cut-offs, relationships between vitamin K and PTH with REE varied. Such stratification indicated a positive relationship of dietary vitamin K and REE in lean individuals, yet null in those with excess adiposity. In subjects with excess adiposity, the attenuation of this relationship suggests the mechanism by which vitamin K influences energy expenditure. Indeed, adiposity has been reported to interfere with bone metabolism in children via secondary low bone turnover and reduced skeletal utilization of calcium (22). Though causality cannot be inferred identified relationships between both vitamin K and PTH with REE support the possibility that reciprocity between bone and fat may be mediated by REE (22).

Although statistical significance was not reached for each dietary nutrient's association with REE, it is important to note an observation, warranting further investigation. Phosphorus metabolism in many respects parallels that of calcium (23). As calcium is liberated from bone, so too is phosphorus. In addition, PTH-stimulated enhancement of intestinal absorption of calcium extends to that of phosphorus. Evidently, alterations of PTH levels

become concordant with phosphorus metabolism, thereby playing a role in calcium homeostasis and resultant energy resource disposal (21, 23–25).

In the context of our observed relationship between vitamin K and REE, the observed trend for an association of the hormone OC with REE ($p<0.10$) may also be noteworthy. Although physiologic implications of the bone marker OC are not fully understood, circulating levels are to an extent indicative of energy utilization. Discordant with adult studies (15, 26), we observed an inverse relationship between OC and REE. The discrepancy is not entirely inexplicable, but may rely on complexity of the hormone itself, as action relies on its bioactivity. In its vitamin K-required carboxylated form, OC confers calcium-binding properties of bone (17). In its circulating undercarboxylated form, OC has been shown to be positively related to energy expenditure (15). Whereas total OC, as assessed in this sample, is dependent on bone turnover, the ratio of carboxylated to undercarboxylated OC, an unavailable measure, is dependent on vitamin K intake. Future investigation regarding the relationship of OC with energy metabolism is warranted, particularly in those undergoing skeletal growth.

Major strengths of this study were use of objectively assessed data (in addition to self-reported intake) and robust measurement of body composition. Because of the interrelationships among the variables, the relatively small sample size may have precluded the detection of significant relationships. The cross-sectional nature of the study limits ability to infer causation; thus findings serve as observational data for future expansion. As after REE, PA represents the next greatest contributor to energy expenditure for most, the lack of association between PA and REE was surprising. It is likely that the inability to detect a relationship was grounded in the low levels of PA measured in this group. Though PA was not informative in this analysis, absence of a relationship may guide intervention efforts among this population. Interestingly, the effects of inactivity on fuel utilization are increasingly becoming characterized. However, the mechanisms underlying down-regulation in REE induced by physical inactivity have not yet been clearly defined. Finally, the analysis is also limited by the modest sample size. Although, a larger cohort would provide further support for the findings, it was determined that a minimum of 27 participants was necessary for an analysis providing 80% power with a corresponding effect size=0.25 at $p=0.05$, suggesting adequate power for the performed analyses.

Beyond commonly regarded pathways, dynamic parameters involved in calcium homeostasis influence energy balance through multiple mechanisms involving REE. Components of the calciotropic network were associated with REE. The relationships differed according to levels of adiposity and nutrient intake, highlighting the importance of resource partitioning and dietary nutrient adequacy. Further, the associations were independent of genetic background and physical activity. These findings stress the importance of achieving dietary adequacy essential for establishing optimal body composition trajectories, particularly through the formative years. The physiologic response to the diet and subsequent body tissue partitioning warrants further investigation, particularly in the context of skeletal maturation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Key Notes

- REE accounts for approximately 65% of total energy expenditure and body composition is the main driver of REE.
- Thus, trajectories established during puberty have profound impact on long-term energy balance.
- Calcitropic factors are integral in metabolic pathways directing fuel utilization and body tissue partitioning (e.g. fat vs. bone), and therefore ultimately energy balance.

Figure 1.

Mean[†] resting energy expenditure by dietary nutrient intake adequacy (determined by Dietary Reference Intake (12)). Dark gray bars represent nutrient intake meeting DRI, light gray bars represent intake lower than DRI. REE=Resting Energy Expenditure; [†]p=0.09, [‡]p=0.08 [†]Adjusted for European admixture, pubertal stage, fat and lean mass, and overall energy intake.

Table 1Sample characteristics (n=36; mean $\pm$ SEM) in total sample and stratified by adiposity[†]

Variables	Total sample	Lean (n=25)	Obese (n=11)
Age (yr)	9.3 $\pm$ 0.3	9.4 $\pm$ 0.3	8.9 $\pm$ 0.4
Pubertal stage [‡]	1.72 $\pm$ 0.15	1.7 $\pm$ 0.2	1.8 $\pm$ 0.3
European admixture	0.50 $\pm$ 0.06	0.47 $\pm$ 0.1	0.56 $\pm$ 0.1
Height (cm)	140.2 $\pm$ 1.7	140 $\pm$ 2	140 $\pm$ 3
Weight (kg)	36.0 $\pm$ 1.2	33.7 $\pm$ 1.2 ^a	41.3 $\pm$ 1.9 ^b
BMI percentile	63.6 $\pm$ 4.8	52 $\pm$ 6 ^a	89 $\pm$ 2 ^b
Total % Fat	25.2 $\pm$ 1.4	20.7 $\pm$ 1.0 ^a	35.4 $\pm$ 1.1 ^b
Dietary Intake (kcal/d)	1,797 $\pm$ 39	1,873 $\pm$ 71	1,773 $\pm$ 120
PA (min/d)	173.3 $\pm$ 17.1	184.4 $\pm$ 21.9	147.8 $\pm$ 25.4
REE (kcal/d)	1,152.8 $\pm$ 33.2	1,134.8 $\pm$ 41.5	1,193.9 $\pm$ 54.2
BMC (g)	1,260.6 $\pm$ 45.0	1,232.2 $\pm$ 52.9	1,325.1 $\pm$ 85.9
PTH (pg/ml)	47.2 $\pm$ 3.0	49.7 $\pm$ 3.8	41.6 $\pm$ 4.6
25OHD (ng/ml)	26.4 $\pm$ 1.1	25.9 $\pm$ 1.5	27.3 $\pm$ 1.6
OC (ng/ml)	12.0 $\pm$ 0.7	12.0 $\pm$ 1.0	11.9 $\pm$ 0.6
Calcium (mg/d)	832 $\pm$ 44	820 $\pm$ 56	859 $\pm$ 70
Vitamin D (mcg/d)	4.9 $\pm$ 0.6	4.9 $\pm$ 0.8	4.9 $\pm$ 0.6
Vitamin K (mcg/d)	64 $\pm$ 14	61 $\pm$ 17	70 $\pm$ 24
Phosphorus (mg/d)	1,099 $\pm$ 49	1,067 $\pm$ 59	1,173 $\pm$ 83

PA=physical activity, REE=resting energy expenditure, BMC=bone mineral content

[†]lean <30% fat, obese $\geq$ 30% fat (6, 9)[‡]On a scale of 1–5, based on the criteria of Marshall and Tanner(7)^{a,b}represents significant difference between groups (p<0.05)