

# Cooking Increases Net Energy Gain From a Lipid-Rich Food

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**ABSTRACT** Starch, protein, and lipid are three major sources of calories in the human diet. The unique and universal human practice of cooking has been demonstrated to increase the energy gained from foods rich in starch or protein. Yet no studies have tested whether cooking has equivalent effects on the energy gained from lipid-rich foods. Using mice as a model, we addressed this question by examining the impact of cooking on the energy gained from peanuts, a lipid-rich oilseed, and compared this impact against that of nonthermal processing (blending). We found that cooking consistently increased the energy gained per calorie, whereas blend-

ing had no detectable energetic benefits. Assessment of fecal fat excretion showed increases in lipid digestibility when peanuts were cooked, and examination of diet microstructure revealed concomitant alterations to the integrity of cell walls and the oleosin layer of proteins that otherwise shield lipids from digestive lipases. Both effects were consistent with the greater energy gain observed with cooking. Our findings highlight the importance of cooking in increasing dietary energy returns for humans, both past and present. *Am J Phys Anthropol* 156:11–18, 2015. © 2014 Wiley Periodicals, Inc.

Cooking is a universal human behavior that has been proposed to function partly as a mechanism for increasing dietary net energy gain (Wrangham, 2009). Previous studies have shown cooking to increase the energy gained from carbohydrate-rich and protein-rich foods (Carmody and Wrangham, 2009; Carmody et al., 2011). Yet, to our knowledge no studies have examined whether routine cooking has equivalent effects on lipid-rich foods, despite the physiological importance of fat in ancestral and modern human diets (Speth and Spielmann, 1983; Leonard et al., 2010; Speth, 2010). Nor has any study compared the effects of cooking lipids against those of alternate, nonthermal modes of processing, which is a key to isolating the energetic consequences of heat. To investigate these questions, we assessed the effects of cooking and/or blending on energy gain from peanuts (*Arachis hypogaea*), a lipid-rich oilseed. Nuts and other oilseeds have long served as important sources of dietary lipid (Peters, 1987; Dreher et al., 1996; Goren-Inbar et al., 2002), and both ancestral (Robbins and Campbell, 1990; Alperson-Afil et al., 2009; Hosoya, 2011) and modern humans (Sabate et al., 2006; Ros, 2010) have routinely cooked these items.

The standard, Atwater system of nutritional assessment does not take the effects of food processing into account as long as macronutrient composition remains unaltered. Thus it assumes that a given lipid contributes about 9 calories per gram, regardless of how it is processed (Merrill and Watt, 1973). Consequently, Atwater-based assays report that cooking has little impact on the energy value of peanuts (Table S1, Supporting Information). This conclusion fails to acknowledge the possibility that the cellular structure of oilseeds such as peanuts constrains their digestibility. Oilseeds have cell walls composed mainly of indigestible non-starch polysaccharides (Ellis et al., 2004) and store their lipids in oil bodies, intracellular, spherical organelles coated by oleo-

sin proteins (Huang, 1992; Murphy, 1993). These features hinder digestive lipases from accessing the encapsulated lipids (Beisson et al., 2001; Ellis, 2004; Mandalari et al., 2008; Gallier and Singh, 2012), which may explain why unprocessed (raw/whole; RW) nuts and other oilseeds have high measured lipid and energy content, but display lower digestibility than predicted by the Atwater system in vivo (Novotny et al., 2012). Cooking and/or mechanical processing tears cell walls and disrupts oil bodies, promoting lipid release (Schadel et al., 1983; Young and Schadel, 1990; Ellis et al., 2004; Altan et al., 2011). This suggests that processing could increase lipid digestibility, because, unguarded by cell walls and oleosins, the freed lipids are likely more accessible to lipases (Kennelly, 1996; Ellis et al., 2004). Alternatively, it is possible that the efficiency of lipase is reduced by the aggregation of such freed lipid, as lipase activity is known to decrease with increasing particle

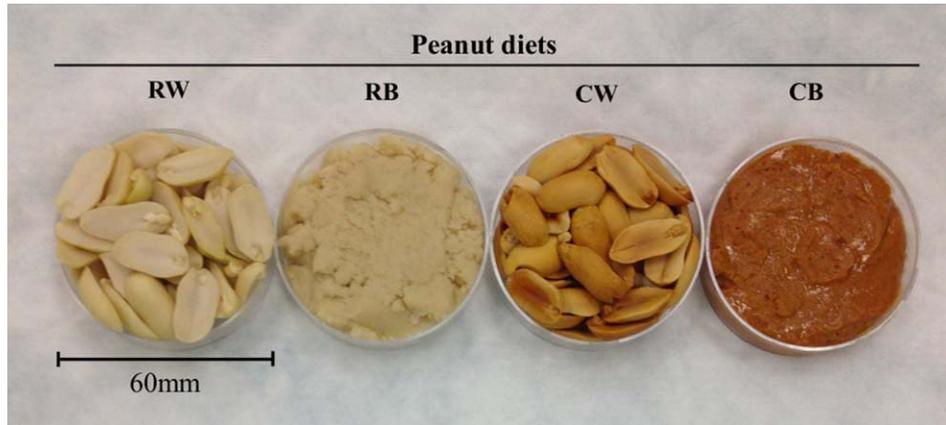
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**Fig. 1.** Typical appearance of prepared peanut diets. RW: raw/whole; RB: raw/blended; CW: cooked/whole; CB: cooked/blended. [Color figure can be viewed in the online issue, which is available at [wileyonlinelibrary.com](http://wileyonlinelibrary.com).]

size (Armand et al., 1992; Borel et al., 1994). How these structural alterations affect the overall energy available from oilseeds is, therefore, unknown, and can only be resolved by examination *in vivo*.

Applying an established protocol (Carmody et al., 2011), we fed laboratory mice (*Mus musculus*) diets of peanuts in four conditions: unprocessed (RW), processed mechanically (raw and blended; RB), processed thermally (cooked and whole; CW), or processed both mechanically and thermally (cooked and blended; CB) (Fig. 1). Mice are a model omnivorous mammal (Latham and Mason, 2004) known to exploit lipid-rich nuts and seeds in natural contexts (Pritchett-Corning et al., 2013). Mice were given each experimental diet *ad libitum* for 5 d, with a 6 d washout period of *ad libitum* chow between consecutive diets. The net energy gained or lost was indexed by change in body mass accounting for gross energy intake (food energy density multiplied by grams food intake) and activity level (wheel running). In addition, we examined fecal fat excretion and peanut diet microstructure to investigate the potential mechanisms responsible.

## MATERIAL AND METHODS

### Research models

Male C57BL/6 mice (*Mus musculus*) were acquired at 21 days of age (Model 027; Charles River). From delivery until 26 weeks of age, mice cohoused with littermates under standard conditions ( $21 \pm 1^\circ\text{C}$ ; 30–50% humidity; 12 h light/dark cycle) at the Biological Research Infrastructure barrier facility at Harvard University. At 26 weeks of age, mice were recruited into the study. Adults were used to minimize change in body mass between trials, and treatment diets were given in a counter-balanced order to control for residual growth. Males were used to eliminate the potential confound of individual variation in energy expenditure due to the ovarian cycle (Curtis et al., 1996). All procedures were approved by the Institutional Animal Care and Use Committee at Harvard University (Protocol 12–17).

### Experimental cage setup

The experimental cage setup was as described previously in Carmody et al. (2011). Briefly, mice were housed individually in standard non-ventilated cages fitted with

a wire mesh floor to minimize coprophagy and facilitate daily collection of food refusals. To minimize contamination and loss of diet beneath the mesh floor, diets were given in Petri dishes fixed to the mesh floor with a pair of sterile plastic-coated neodymium magnets (Model D84PC-WHT; KJ Magnetics). Cage tops were equipped with an exercise wheel (Model 61390; SuperPet) to which a neodymium magnet (Model D42; KJ Magnetics) was attached. A magnetic bicycle counter (BC 500 or BC 506; Sigma) was attached to the ceiling, directly above the orbit of the magnet, such that the magnet would pass by upon each revolution of the wheel. The number of daily wheel rotations recorded by the bicycle counter served as an index of activity level. Cages were sterilized, and fresh liners, cotton nestlet, and water were given daily. Mice were given 6 d to acclimate to this experimental cage setup prior to the start of feeding trials, during which time they were fed chow (IsoPro RMH 3000; PMI Nutrition) *ad libitum*.

### Diet preparation

Experimental diets consisted of peanuts (*Arachis hypogaea*) fed in four preparations: (1) RW, (2) RB, (3) CW, and (4) CB (Fig. 1; Table S2, Supporting Information). Peanuts were purchased raw and in-shell (Wakefield Peanut, VA) to eliminate the confound of pre-experimental processing and minimize the risk of lipid oxidation (Davidson et al., 1982). Diets were prepared under sterile conditions and within 3 h of use. To maximize consistency across trials, all diets were processed in batches of uniform size and by the same researcher (E.E.G.). For all diets, peanuts were shelled immediately prior to use. For RW, peanuts were skinned by gentle rubbing, separated into their two cotyledons, and weighed into *ad libitum* rations ( $22.5 \pm 0.05$  g). For CW, shelled whole, raw peanuts were placed in a single layer on a baking sheet lined with aluminum foil and roasted for 17 min in a  $167^\circ\text{C}$  oven. This time/temperature combination yields peanuts of a Medium roast color (McDaniel et al., 2012), which are used as the reference standard in USDA investigations involving roasted peanuts (McNeill and Sanders, 1998). Following roasting, peanuts were allowed to cool to room temperature, and weighed into *ad libitum* rations. For each of the blended diets (RB, CB), peanuts were prepared as for RW or CW, blended using a food processor (DLC-2A Mini Prep Plus;

Cuisinart) until they reached a consistency resembling “smooth-style” commercial peanut butter (RB: 75–90 s; CB: 45–60 s), and weighed into ad libitum rations. Blending times were experimentally determined to not appreciably increase diet temperature (RB: pre-blending: 22.1°C, post-blending: 22.4°C; CB: pre-blending: 22.7°C; post-blending: 22.9°C). To determine gross dietary energy density, replicates of each diet were analyzed for macronutrient content using standard biochemical assays (Table S2, Supporting Information).

### Feeding protocol

Mice were fed for 5 consecutive days on each of the experimental diets, using a counterbalanced, within-subjects design. Diets were assigned symmetrically, with five mice consuming each diet at any point in time. To prevent carry-over effects across treatments, mice were reared on chow ad libitum for 6 d between each feeding trial. All diets were given at the same time each day to ensure a standardized, 24 h data collection cycle. During this daily intervention, mice were weighed in sterile paper weighing containers, and body mass was recorded ( $\pm 0.1$  g) during a period of inactivity. Fresh fecal samples were collected non-invasively with sterile forceps, flash-frozen in liquid nitrogen, and stored at  $-20^{\circ}\text{C}$  until analysis of fecal fat excretion. Food refusals from the previous 24 h were also collected and weighed fresh to determine fresh-weight intake (defined as the difference between initial diet ration and refusal weights). Refusals were stored at  $-20^{\circ}\text{C}$  and later freeze-dried to constant mass to determine dry-weight intake. To measure daily activity level, wheel-running data from the previous 24 h were recorded, and the bicycle counter was reset.

### Fecal fat excretion

Feces collected during Days 2–5 of feeding trials were assessed for total lipid content to compare lipid digestibility across the peanut diets. Samples were ground under liquid nitrogen to pass through a #40 mesh (420  $\mu\text{m}$ ) filter. Total fecal lipid content was determined by petroleum ether extraction (Conklin-Brittain et al., 2006).

### Diet microstructure

Treatment diets were examined using light and confocal laser scanning microscopy (CSLM) to assess the effects of mechanical and/or thermal processing on peanuts' cellular structure.

**Light microscopy.** Light microscopy was used to examine cell walls. Samples were fixed in 4% paraformaldehyde at room temperature for 4 d and embedded in paraffin. Three 5  $\mu\text{m}$  thick cross-sections of each diet were cut using a Cryostat, mounted on VWR Superfrost Plus Slides (VWR Cat# 48311-703), and stained with hematoxylin (carbohydrate; purple) and eosin (protein; red/pink). Each diet sample was examined qualitatively using an Olympus BX41 Histology Microscope with DP25 Camera & Side-by-Side Viewing (Olympus Corp., Tokyo, Japan) at 100x magnification using CELLSSENS imaging software (Olympus Corp., Tokyo, Japan).

**CSLM.** CSLM was used to assess the state of oil bodies. Samples of whole diets (RW, CW) were prepared according to the method of Altan et al. (2011), which validated

CSLM for whole almonds subjected to different modes of thermal processing. Five peanut halves were randomly selected (one half from each of the five samples), and 500  $\mu\text{m}$  thick sections were cut using a razor blade from the middle of the nut to obtain the parenchyma tissue, where the majority of nutrients are stored. For blended diets (RB, CB), samples were cut into 500  $\mu\text{m}$  thick sections. All samples were subsequently stained with a solution of Nile Red (non-polar lipids, including triacylglycerols; 1 mg/mL acetone, 1:100, v/v) and Fast Green FCF (protein; 1 mg/mL Milli-Q water, 1:100, v/v), and mounted on MatTek dishes (Model P35G-1.5–14-C; MatTek Co) using glycerol. They were then viewed under CSLM (Zeiss LSM 700 Inverted Confocal, Oberkochen, Germany) with a 63 mm oil immersion lens. Nile Red was excited with the 488 nm line from the Ar+ laser and the filters were set to collect emitted light between 500 and 600 nm. Fast Green FCF was excited with the 633 nm line from the HeNe laser and the filters collected emitted light between 650 and 750 nm.

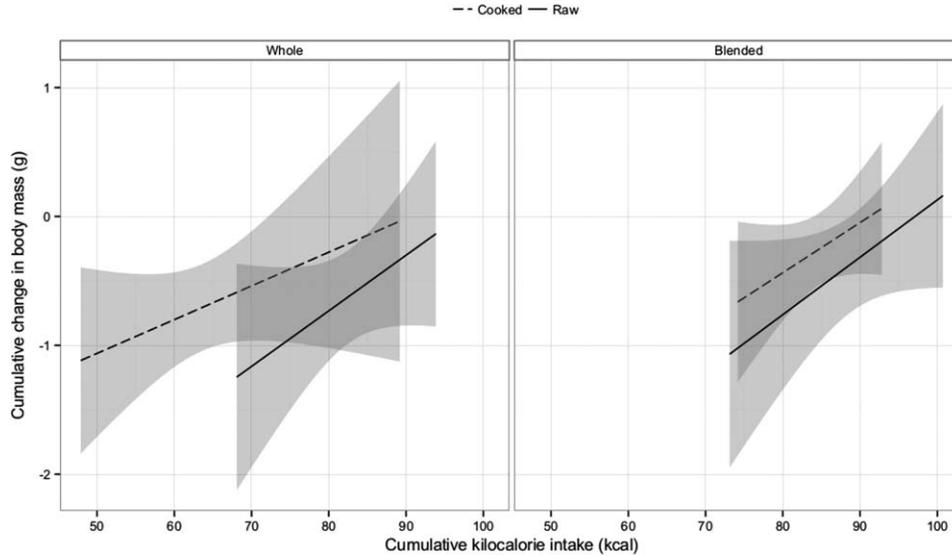
### Data analysis

Net energy gain was assessed by running linear mixed effects models (LMEs) to examine the effects of cooking and blending on body mass independent of gross energy intake and physical activity. The best model was selected by restricted maximum likelihood, which penalizes appropriately for the number of model parameters when sample size is small (Schaeffer et al., 1978; Bolker et al., 2009). Significance was set at an alpha level of 0.05. Two-way repeated measures ANOVA ( $2 \times 2$  RM ANOVA) was used to examine the association between fecal fat excretion and the factors cooking, blending, and their interaction. Two-sample *t*-tests (two-tailed) were used to identify pairwise differences between all six possible diet combinations. To correct for the increased probability of false positives due to multiple independent comparisons, a Bonferroni correction was used to readjust the alpha level from 0.05 to 0.0083. The effects of processing on relative dietary preferences were examined using chi-square goodness of fit tests. All analyses were conducted in R (R Core Team, 2012). LMEs were performed using the *lme* function of the *nlme* package, and the  $2 \times 2$  RM ANOVA using the *Anova* function of the *car* package. Chi-square tests and *t*-tests were performed using the chi-square test and *t*-test functions of the *stats* package, respectively.

## RESULTS

### Net energy gain

Cooking consistently increased net energy gain. LME model analysis showed that gross energy intake (calculated on a dry-weight basis) was a significant predictor of change in body mass ( $B = 0.046$ ,  $SE = 0.012$ ,  $P < 0.001$ ), but activity level was not ( $B = 0.001$ ,  $SE = 0.004$ ,  $P = 0.775$ ). Controlling for these factors, cooking had a positive effect on change in body mass (LME model;  $B = 0.589$ ,  $SE = 0.209$ ,  $P = 0.007$ ), indicating that it increased net energy gain (Fig. 2; Table 1). By contrast neither blending ( $B = -0.051$ ,  $SE = 0.227$ ,  $P = 0.825$ ) nor the interaction between blending and cooking ( $B = -0.049$ ,  $SE = 0.223$ ,  $P = 0.827$ ) affected change in body mass independently. While insignificant overall, these effects were both weakly negative. Consequently, cooking significantly increased energy gain from



**Fig. 2.** Impact of dietary processing on net energy gain. Cumulative change in body mass (g) and cumulative kilocalorie intake (kcal consumed; dry-weight basis) are over the 5 d feeding trial. Shaded regions represent 95% CI regions for the relevant regression line.

**TABLE 1.** LME model output for net energy gain (dry-weight basis values)

Factor	B	SE	P-value
<b>Main effects</b>			
(Intercept)	-4.203	0.956	<0.001
Physical activity (km)	0.001	0.004	0.775
Gross energy intake (kcal) <sup>a</sup>	0.046	0.012	<0.001
Cooking	0.589	0.209	0.007
Blending	-0.051	0.227	0.825
Cooking*Blending	-0.049	0.223	0.827
<b>Simple Effects</b>			
RW-CW	1.080	0.522	0.043
RB-CB	1.276	0.689	0.070
RW-RB	-0.003	0.569	0.995
CW-CB	-0.199	0.697	0.776

B = unstandardized effect size. This is the amount that the dependent variable (net change in body mass) changes for a 1-unit change in the given factor. For categorical factors, this one-unit change refers to moving from one state to another— e.g., for “cooking,” the effect size represents the change in body mass when moving from a raw to a cooked diet, while keeping all other factors in the model constant.

<sup>a</sup>Gross energy intake was calculated on a dry-weight basis: dry mass food intake (g) × gross dietary energy density (kcal/g dry mass).

a whole diet (RW-CW:  $B = 1.080$ ,  $SE = 0.522$ ,  $P = 0.043$ ) but not a blended one (RB-CB:  $B = 1.276$ ,  $SE = 0.689$ ,  $P = 0.070$ ). Similarly, blending a cooked diet did not increase net energy gain (CW-CB:  $B = -0.199$ ,  $SE = 0.697$ ,  $P = 0.776$ ). Results were similar when fresh-weight basis values were used (Fig. S1, Table S3, Supporting Information).

**Fecal fat excretion**

Patterns of fecal fat excretion (Table 2) corresponded to those observed for net energy gain. Thus, only cooking significantly influenced fecal fat loss ( $2 \times 2$  RM ANOVA; cooking:  $P = 0.001$ , blending:  $P = 0.741$ , cooking × blend-

**TABLE 2.** Fecal fat excretion on peanut diets

Diet	Percent fat excreted
RW	11.63 ± 1.03 <sup>a</sup>
RB	11.14 ± 0.91
CW	7.92 ± 0.97 <sup>a</sup>
CB	7.72 ± 0.89

Percent fat excreted was calculated as: total fecal lipid content (g)/wet feces (g), times 100. Values are means ± SE, for feces samples collected over 4d from mice ( $n = 20$ ).

Diet abbreviations are: RW: raw/whole; RB: raw/blended; CW: cooked/whole; and CB: cooked/blended.

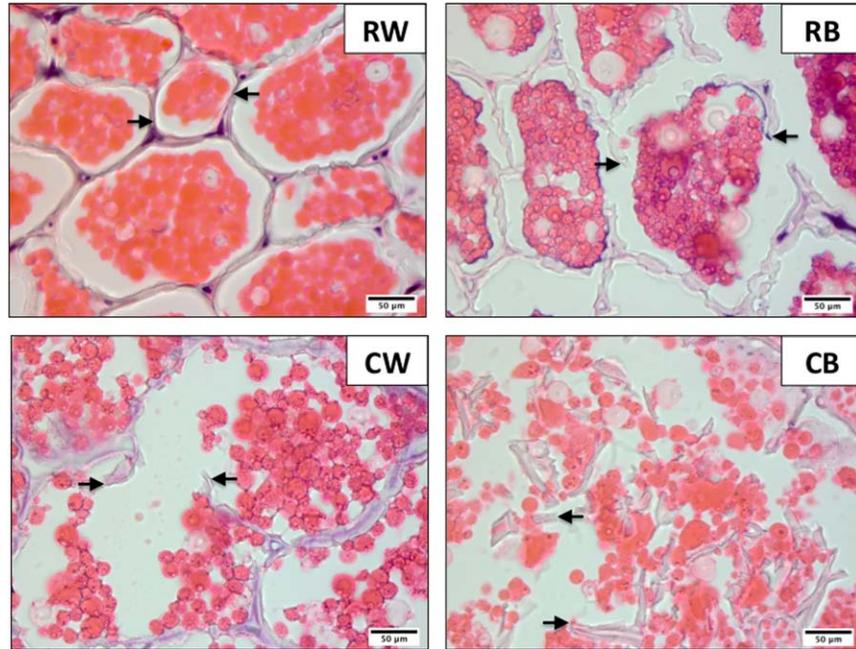
<sup>a</sup> Values that significantly differed from each other (paired  $t$  test with Bonferroni correction,  $P \leq 0.0083$ ).

ing:  $P = 0.880$ ). Mice excreted less fat on cooked peanuts (paired  $t$ -test; CW < RW;  $P = 0.008$ ; CB < RB;  $P = 0.017$ ), indicating that cooking increased total-tract lipid digestibility.

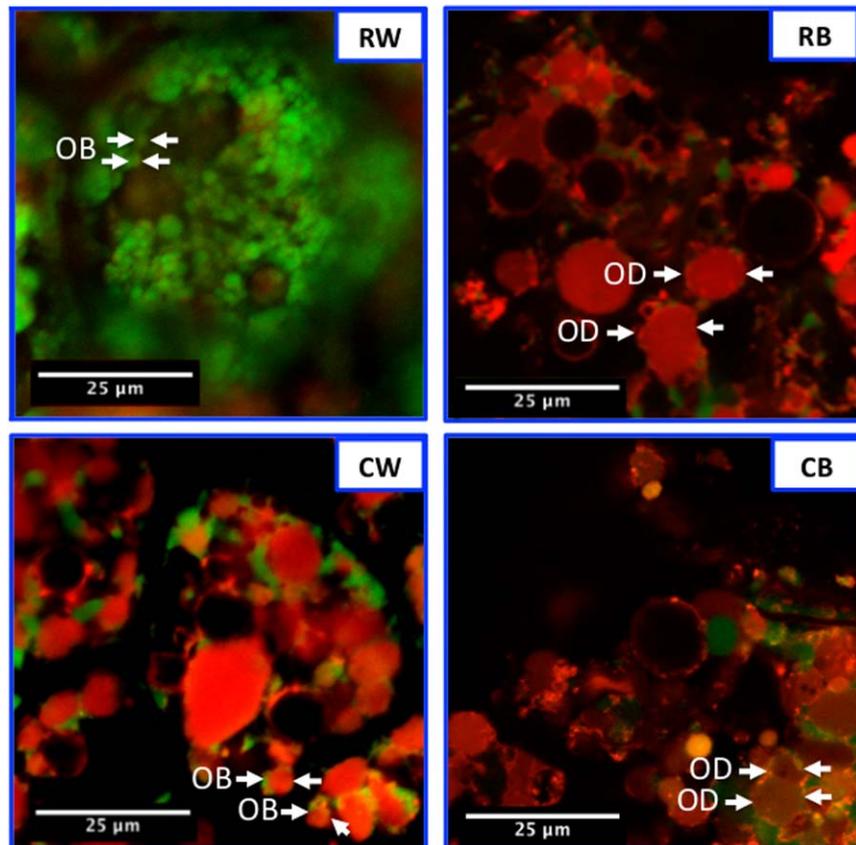
**Peanut diet microstructure**

**Cell walls.** As observed previously (Schadel et al., 1983; Young and Schadel, 1990; Ellis et al., 2004; Altan et al., 2011), both cooking and blending damaged cell walls (Fig. 3). While blending alone had no other obvious disruptive effects, cells in CW peanuts were substantially larger than those in the RW samples and experienced scattering of their contents, indicating that heating had promoted cellular swelling and rupture. Cooking also appeared to facilitate subsequent mechanical processing: the CB sample displayed the most cell damage, with few intact cells present and the greatest release of intracellular contents.

**Oil bodies.** While cooking and/or mechanical processing damaged the exterior oleosin layer present in RW peanut oil bodies (Fig. S2, Supporting Information), oil bodies in the CW and blended (RB, CB) diets had distinct structures (Fig. 4). In the CW diet, discrete lipid



**Fig. 3.** Effects of processing on cell walls in peanut treatment diets (RW: raw/whole, RB: raw/blended, CW: cooked/whole, CB: cooked/blended). Black arrows point to cell walls: intact, in the (unprocessed) RW diet, or torn, in the (processed) cooked and/or blended diets. Samples were stained with hematoxylin (carbohydrate; purple) and eosin (protein; red/pink). Scale bar is 50  $\mu\text{m}$ .



**Fig. 4.** Effects of processing on oil bodies in peanut treatment diets (RW: raw/whole, RB: raw/blended, CW: cooked/whole, CB: cooked/blended). Samples were stained with Nile Red (lipid) and Fast Green (protein). Note that oil bodies (OB) are intact in RW peanuts. Oil bodies are still identifiable in CW peanuts, but are present as (aggregated) flocs. In the blended diets, oil bodies have coalesced to form oil droplets (OD). Contrast was adjusted for the RW image to facilitate identification of the oil bodies. Scale bar is 25  $\mu\text{m}$ .

bodies, henceforth denoted as “cooked oil bodies,” were largely present as aggregated flocs with associated surface peptides. These peptides were likely oleosin fragments, as they were highly co-localized to the exterior of lipid bodies (Fig. S2, Supporting Information). In contrast, oil bodies in blended peanuts had largely coalesced into oil droplets (Fig. 4).

## DISCUSSION

Our findings indicate that cooking increases the fraction of lipid available and the net energy gained from peanuts, a lipid-rich oilseed. Analysis of diet microstructure provides a strong candidate mechanism by which cooking increased the energetic value of peanuts. Cooked oil bodies were present as flocs resembling those formed by almond (Gallier and Singh, 2012) and walnut (Gallier et al., 2013) oil bodies after 60 min of *in vitro* gastric digestion. This close resemblance suggests that heat mimicked the disruptive effects of pepsin on oleosins. The fact that peanut oleosins denature between 50°C and 59°C (Cabanos et al., 2011), which is well below the 167°C roasting temperature used, further supports this hypothesis. Thus, cooking may “pre-digest” oil bodies, promoting subsequent lipolysis *in vivo*. As observed *in vitro*, disruption of the oleosin layer could facilitate lipase adsorption onto the oil droplet surface (Gallier and Singh, 2012; Gallier et al., 2013) and stimulate gastric fat emulsification, as peptides are known to help emulsify lipid droplets in the stomach (Carey et al., 1983).

Prior studies have noted that human subjects given peanut butter (peanuts that have been roasted, and then ground) excreted less fat than those given whole roasted peanuts. This implies that mechanical processing of cooked peanuts increases the digestibility of their lipid, and presumably the energy gained from them (Levine and Silvis, 1980; Traoret et al., 2008). However, in our study the greater lipid release observed microscopically in blended diets did not increase lipid digestibility or net energy gain. Interspecies differences in mastication offer one possible explanation for this discrepancy. In mice and other rodents, the masseter is extended, such that it inserts onto the medial, rather than the lateral, surface of the mandible (Druzinsky et al., 2011). This masseteric extension helps rodents produce large bite forces that favor high chewing efficiency (Baverstock et al., 2013). Thus, one possibility is that mice masticated the whole peanuts so completely that all diets were effectively blended upon reaching the stomach and small intestine, the predominant sites of lipid digestion in both species (DeNigris et al., 1988; Mu and Hoy, 2004). Further study on the effects of processing on mastication and digestion in mice, humans and other species could help resolve the energetic significance of blending for lipid-rich foods.

Given the importance of lipids as a uniquely dense source of energy, our results have significant implications for both ancestral and modern human nutrition. Despite being costly in time and energy, cooking is a cultural universal (Wrangham, 2009). A functional perspective suggests that cooking, therefore, should increase overall fitness, and a proposed mechanism is through increasing net dietary energy gain (Wrangham, 2009; Wrangham and Carmody, 2009). However, prior research on the energetic significance of cooking for human evolution has considered only two of the three major macro-

nutrients. Thermal processing has been previously observed to increase the energy gained from carbohydrate-rich tubers and protein-rich lean meat (Carmody et al., 2011); our data show that it increases the energy gained from lipid-rich nuts as well. Tubers, meat, and nuts are thought to have been major components of ancestral human diets (Peters, 1987; Ungar, 2007). Our findings, therefore, imply that cooking these foods would have raised the human energy budget, helping fuel expensive increases in body mass, brain size, locomotor activity and other costly physiological traits (Aiello and Wells, 2002).

Today, energy deficiency and energy excess are both major public health concerns, with many nations facing the “double burden” of obesity and malnutrition (FAO, 2006). Lipids are highly relevant to understanding and addressing these modern epidemics (FAO, 2010). In the United States, Atwater-based calculations of food energy content advise national dietary guidelines (Bliss, 2012). Such calculations (Table S1, Supporting Information) indicate that raw and roasted peanuts offer equivalent amounts of lipid and metabolizable energy once corrected for differences in water content (dry-weight basis). In contrast, our data show that lipid digestibility and net energy gain from cooked peanuts significantly exceeded that from raw peanuts on a dry-weight basis. This discrepancy between measured calorie content and *in vivo* energy returns—previously shown for foods rich in carbohydrate or protein (Carmody et al., 2011), and now for those rich in lipid—limits our ability to effectively address the epidemics resulting from caloric insufficiency or excess. Such research calls new attention to the need for finding appropriate ways to interpret the existing Atwater-based system of nutritional assessment.

Our findings also offer promising avenues to optimize dietary energy intake. For example they suggest that individuals who eat raw lipid-rich foods would excrete a greater fraction of the lipid, enabling them to limit weight gain despite the high measured calorie content of these items. Conversely, consuming cooked lipid-rich foods could help increase net energy gain and body mass in cases of malnutrition. Recent observations support the potential value of such strategies: (1) adults consuming diets supplemented with raw, whole nuts achieve similar weight loss outcomes as those consuming non-supplemented control diets, despite the additional calories (Casas-Agustench et al., 2011); and (2) consumption of a roasted peanut paste promotes rapid weight restoration in malnourished children (Ashworth, 2006). Further studies of the energetic consequences of cooking lipid-rich foods, including quantification of these effects in humans, will help to better understand the adaptive significance of cooking and more effectively address the burdens of obesity and malnutrition.

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## LITERATURE CITED

Aiello LC, Wells JCK. 2002. Energetics and the evolution of the genus *Homo*. *Annu Rev Anthropol* 31:323–338.

- Alperson-Afil N, Sharon G, Kislev M, Melamed Y, Zohar I, Ashkenazi S, Rabinovich R, Biton R, Werker E, Hartman G, Feibel C, Goren-Inbar N. 2009. Spatial organization of hominin activities at Gesher Benot Ya'aqov, Israel. *Science* 326: 1677–1680.
- Altan A, McCarthy KL, Tikekar R, McCarthy MJ, Nitin N. 2011. Image analysis of microstructural changes in almond cotyledon as a result of processing. *J Food Sci* 76: E212–E221.
- Armand M, Borel P, Ythier P, Dutot G, Melin C, Senft M, Lafont H, Lairon D. 1992. Effects of droplet size, triacylglycerol composition, and calcium on the hydrolysis of complex emulsions by pancreatic lipase: an in vitro study. *J Nutr Biochem* 3:333–341.
- Ashworth A. 2006. Efficacy and effectiveness of community-based treatment of severe malnutrition. *Food Nutr Bull* 27: S24–S48.
- Baverstock H, Jeffery NS, Cobb SN. 2013. The morphology of the mouse masticatory musculature. *J Anat* 223:46–60.
- Beisson F, Ferte N, Bruley S, Vouloutoury R, Verger R, Arondel V. 2001. Oil-bodies as substrates for lipolytic enzymes. *Biochim Biophys Acta* 1531:47–58.
- Bliss RM. 2012. Monitoring food-supply nutrients: the second step - conservators of the National Nutrient Database. *Agric Res* 60:8–15.
- Bolker BM, Brooks ME, Clark CJ, Geange SW, Poulsen JR, Stevens HH, White JS. 2009. Generalized linear mixed models: a practical guide for ecology and evolution. *Trends Ecol Evol* 24:127–135.
- Borel P, Armand M, Ythier P, Dutot G, Melion C, Senft M, Lafont H, Lairon D. 1994. Hydrolysis of emulsions with different triglycerides and droplet sizes by gastric lipase in vitro. Effect on pancreatic lipase activity. *J Nutr Biochem* 5:124–133.
- Cabanos C, Katayama H, Tanaka A, Utsumi S, Maruyama N. 2011. Expression and purification of peanut oleosins in insect cells. *Protein J* 30:457–463.
- Carey MC, Small DM, Bliss CM. 1983. Lipid digestion and absorption. *Annu Rev Physiol* 45:651–677.
- Carmody RN, Weintraub GS, Wrangham RW. 2011. Energetic consequences of thermal and nonthermal food processing. *Proc Natl Acad Sci USA* 108:19199–19203.
- Carmody RN, Wrangham RW. 2009. The energetic significance of cooking. *J Hum Evol* 57:379–391.
- Casas-Augstench P, López-Uriarte, Bulló, Ros E, Cabré-Vila JJ, Salas-Salvado J. 2011. Effects of one serving of mixed nuts on serum lipids, insulin resistance and inflammatory markers in patients with the metabolic syndrome. *Nutr Metab Cardiovasc* 21:126–135.
- Conklin-Brittain NL, Knott CD, Wrangham RW. 2006. Energy intake by wild chimpanzees and orangutans: methodological considerations and a preliminary comparison. In: Hohmann G, Robbins M, Boesch C, editors. *Feeding ecology in apes and other primates: ecological, physical and behavioral aspects*. Cambridge, UK: Cambridge University Press. p 445–471.
- Curtis V, Henry CJK, Birch E, Ghousain-Choueiri A. 1996. Intra-individual variation in the basal metabolic rate of women: effect of the menstrual cycle. *Am J Hum Biol* 8:631–639.
- Davidson JI, Whitaker TB, Dickens JW. 1982. Grading, cleaning, storage, shelling, and marketing of peanuts in the United States. In: Pattee HE, Young CT, editors. *Peanut science and technology*. Yoakum, TX: Amer. Peanut Res. Ed. Soc. p 571–623.
- DeNigris SJ, Hamosh M, Kasbekar DK, Lee TC, Hamosh P. 1988. Lingual and gastric lipases: species differences in the origin of prepancreatic digestive lipases and in the localization of gastric lipase. *Biochim Biophys Acta* 959:38–45.
- Dreher ML, Maher CV, Kearney P. 1996. The traditional and emerging role of nuts in healthful diets. *Nutr Rev* 54:241–245.
- Druzinsky RE, Doherty AH, De Vree FL. 2011. Mammalian masticatory muscles: homology, nomenclature, and diversification. *Integr Comp Biol* 51:224–234.
- Ellis PR, Kendall CW, Ren Y, Parker C, Pacy JF, Waldron, KW, Jenkins DJ. 2004. Role of cell walls in the bioaccessibility of lipids in almond seeds. *Am J Clin Nutr* 80:604–613.
- [FAO] Food and Agriculture Organization. 2006. *The double burden of malnutrition: case studies from six developing countries*. FAO Food and Nutrition Paper 84. Rome: Food and Agriculture Organization.
- [FAO] Food and Agriculture Organization. 2010. *Fats and fatty acids in human nutrition: report of an expert consultation*. FAO Food and Nutrition Paper 91. Rome: Food and Agriculture Organization.
- Gallier S, Singh H. 2012. Behavior of almond oil bodies during in vitro gastric and intestinal digestion. *Food Funct* 3:547–555.
- Gallier S, Tate H, Singh H. 2013. In vitro gastric and intestinal digestion of a walnut oil body dispersion. *J Agric Food Chem* 61:410–417.
- Goren-Inbar N, Sharon G, Melamed Y, Kislev M. 2002. Nuts, nut cracking, and pitted stones at Gesher Benot Ya'aqov, Israel. *Proc Natl Acad Sci USA* 99:2455–2460.
- Hosoya LA. 2011. Staple or famine food? Ethnographic and archaeological approaches to nut processing in East Asian prehistory. *Archaeol Anthropol Sci* 3:7–17.
- Huang AH. 1992. Oil Bodies and oleosins in seeds. *Annu Rev Plant Physiol Mol Biol* 43:177–200.
- Kennelly JJ. 1996. The fatty acid composition of milk fat as influenced by feeding oilseeds. *Anim Feed Sci Technol* 60: 137–152.
- Latham N, Mason G. 2004. From house mouse to mouse house: the behavioural biology of free-living *Mus musculus* and its implications in the laboratory. *Appl Anim Behav Sci* 86:261–289.
- Levine AS, Silvis SE. 1980. Absorption of whole peanuts, peanut oil, and peanut butter. *N Engl J Med* 303:917–918.
- Leonard WR, Snodgrass JJ, Robertson ML. 2010. Evolutionary perspectives on fat ingestion and metabolism in humans. In: Montmayeur JP, le Coutre J, editors. *Fat detection: taste, texture, and post ingestive effects*. Boca Raton, FL: CRC Press. Chapter 1. Available at: <http://www.ncbi.nlm.nih.gov/books/NBK53561/>. Accessed on July 27, 2014.
- Mandalari G, Faulks RM, Rich GT, Lo Turco V, Picout DR, Lo Curto RB, Bisignano G, Dugo P, Dugo G, Waldron KW, Ellis PR, Wickham MS. 2008. Release of protein, lipid, and vitamin E from almond seeds during digestion. *J Agric Food Chem* 56:3409–3416.
- McDaniel KA, White BL, Dean LL, Sanders TH, Davis JP. 2012. Compositional and mechanical properties of peanuts roasted to equivalent colors using different time/temperature combinations. *J Food Sci* 77:C1292–C1298.
- McNeill KL, Sanders TH. 1998. Maturity effects on sensory and storage quality of roasted Virginia-type peanuts. *J Food Sci* 63:366–369.
- Merrill AL, Watt BK. 1973. *Energy value of foods: basis and derivation*. Washington, DC: United States Department of Agriculture, Agricultural Research Service.
- Mu H, Hoy CE. 2004. The digestion of dietary triacylglycerols. *Prog Lipid Res* 43:105–133.
- Murphy DJ. 1993. Structure, function and biogenesis of storage lipid bodies and oleosins in plants. *Prog Lipid Res* 32:247–280.
- Novotny JA, Gebauer SK, Baer DJ. 2012. Discrepancy between the Atwater factor predicted and empirically measured energy values of almonds in human diets. *Am J Clin Nutr* 96:296–301.
- Oka K, Sakurarae A, Fujise T, Yoshimatsu H, Sakata T, Nakata M. 2003. Food texture differences affect energy metabolism in rats. *J Dent Res* 82:491–494.
- Peters CR. 1987. Nut-like oil seeds - food for monkeys, chimpanzees, humans, and probably ape-men. *Am J Phys Anthropol* 73:333–363.
- Pritchett-Corning KR, Keefe R, Garner JP, Gaskill BN. 2013. Can seeds help mice with the daily grind? *Lab Anim* 47:312–315.
- R Core Team. 2012. *R: a language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical

- Computing. ISBN 3-900051-07-0. Available at: <http://www.R-project.org/>. Accessed on July 27, 2014.
- Robbins LH, Campbell AC. 1990. Prehistory of Mongongo nut exploitation in the Western Kalahari Desert. *Botsw Notes Rec* 22:37-42.
- Ros E. 2010. Health benefits of nut consumption. *Nutrients* 2: 652-682.
- Sabate J, Ros E, Salas-Salvado J. 2006. Nuts: nutrition and health outcomes. *Br J Nutr* 96:S1-S2.
- Schadel WE, Walter WM, Young CT. 1983. Cotyledon structure of resting peanut (*Arachis Hypogaea* L. cv. Florunner) seed before and after hydraulic pressing. *J Food Sci* 48:1405-1407.
- Schaeffer LR, Wilton JW, Thompson R. 1978. Simultaneous estimation of variance and covariance components from multi-trait mixed model equations. *Biometrics* 34:199-208.
- Speth JD. 2010. The paleoanthropology and archaeology of big-game hunting: protein, fat, or politics? New York: Springer.
- Speth JD, Spielmann KA. 1983. Energy source, protein metabolism, and hunter-gatherer subsistence strategies. *J Anthropol Archaeol* 2: 1-31.
- Traoret CJ, Lokko P, Cruz AC, Oliveira CG, Costa NM, Bressan J, Alfenas RC, Mattes RD. 2008. Peanut digestion and energy balance. *Int J Obes (Lond)* 32:322-328.
- Ungar PS. 2007. *Evolution of the Human Diet: the known, the unknown, and the unknowable*. New York: Oxford University Press.
- Wrangham R. 2009. *Catching fire: how cooking made us human*. New York: Basic Books.
- Young CT, Schadel WE. 1990. Light and scanning electron-microscopy of the peanut (*Arachis Hypogaea* L. cv Florunner) cotyledon after roasting. *Food Struct* 9:69-73.