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REVIEW

Development and modification of child food preferences and eating patterns: behavior genetics strategies

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Behavioral genetics (BG) designs can offer useful strategies for studying the development of child food preferences and eating patterns. This review summarizes BG designs that tested familial influences on child eating behavior and implicated both genetic and home environmental factors. A range of BG strategies, including family and pseudo-family designs, classic twins designs, discordant sibling designs, cotwin control designs, and high-risk designs, have provided information on child development that could not have been obtained from the analysis of singletons. BG designs can provide can powerful tools for testing environmental theories of child nutrition and, potentially, for better understanding between-child variability in response to dietary interventions for overweight. The term BG may misleadingly imply only the classic twin design or just heritability estimation; BG strategies can be adapted creatively to address a range of questions concerning the development of child appetite and eating regulation.
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Introduction

Studying the development of eating patterns is important for understanding energy balance and weight regulation in growing children. Children's food intake is influenced by environmental factors such as role modeling, frequency of food exposure, and portion sizes.[1] These studies generally have investigated unrelated children (ie singletons) to establish causal associations.

A different line of studies has investigated the extent to which child eating patterns 'run in families' and the broader genetic–environmental architecture of child eating behavior. These studies fall in the tradition of behavior genetics (BG) designs that partition genetic and environmental sources of variability in behavioral traits.[2] BG designs have played a critical role in establishing the 'familiality' and 'heritability' of child adiposity.[3,4] Familiality refers to the correlation among family members for a trait, reflecting the net influence of genetic and home environmental influences. Heritability (h^2) refers to the percent phenotypic variation in a trait that is due specifically to genetic variations.[5] BG strategies have been applied extensively to studies of child body composition,[4,6] but less frequently to the development of child food preferences and eating styles. Their use, however, has potential for advancing knowledge in this arena and for pushing the limits of information that can be derived solely from singletons.

The aim of this mini-review is to summarize a range of BG strategies that have been used for studying child food preferences and eating behaviors. These studies sampled child participants and at least one related family member, or recruited children based on their familial history of obesity. In the first section below, representative studies from the literature are presented that used different methodologies: family correlation studies; twin designs; discordant sibling designs; cotwin control designs; and high-risk designs. Table 1 provides an overview of these approaches, including the types of research questions they can address and related methodological comments. The second section reviews studies that examined familial associations for weight loss following childhood obesity treatment.

This report intends to compliment related reviews,[16–18] while invoking a developmental BG framework.[19,20] There may be unique advantages to recruiting twins or family members for better understanding the development of child

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Table 1 Behavioral genetic strategies used in the literature to investigate child food preferences and eating behaviors

Design	*Prototypical research question(s) addressed*	*Comments*
Family Correlation ('Familiality') Design	Do family members (eg parent–child; siblings) show resemblance for an eating trait or food preference?	Family correlation provides evidence for genetic and/or home environmental influences on the eating trait under investigation. However, the design cannot statistically disaggregate genetic from home environmental influences on a trait. Therefore, this design is often a useful first step for establishing that a behavioral trait 'runs in families' and merits subsequent genetic investigation
Pseudo-family Design	Is the association among biologically related family members for an eating trait greater than the association among randomly assigned 'family' members for that trait?	This design extends the standard familiality design, while incorporating aspects of an adoption study. The design examines the correlation for a trait among biological family members (eg parent–child pairs). Biological family members pairs are 'decoupled' from each other and randomly reassigned to unrelated individuals from the same cohort to establish a 'pseudo-family.' A significant pseudo-family correlation would imply that factors other than genetics or the home environment are inducing the observed correlations (eg shared cultural or SES preferences for certain foods; exposure to a common treatment intervention). If the biological family correlation exceeds the pseudo-family correlation, this suggests home environmental and/or genetic influences on the eating trait
Classic Twin Design	What is magnitude of genetic, shared environmental, and nonshared environmental influences on a child eating behavior?	Shared environmental influences refer to aspects of the home environmental that are perfectly shared by children in the same household (eg number of snack foods in the cupboards). Nonshared environmental influences refer to aspects of the environment that are not shared by children in the same household (eg differential interactions with parents or peers). There are many variations on the classic twin design, which has been used extensively to study child BMI and body composition.[4] There are many caveats and assumptions when using the classic twin design.[4] Refined environmental measures have been infrequently used in the context of twin studies
Cotwin Control Design	Do MZ twins show different eating patterns when they are exposed to different experimental manipulations or environmental conditions? What environmental or other variables distinguish MZ twin pairs that differ in eating patterns?	This design is not useful for estimating heritability *per se*. However, it can be a powerful strategy for identifying environmental factors that impact on eating patterns. The use of same-sex MZ twin pairs (ie matched for age, gender, ethnicity, and many aspects of the home environment) can make this a powerful design. Articles discussing the power of the cotwin control design can be found elsewhere.[7–9] The cotwin control design has been used to address other issues in adult obesity and weight control[10,11]
Discordant Sibling Designs	What environmental or other variables distinguish siblings that differ in eating patterns or obesity status?	This design may be more feasible than the cotwin control design, for certain research questions. Still, there are important challenges when recruiting siblings that are discordant for weight status. As (discordant) siblings are matched for many aspects of the home environment (eg parental income, SES), this can be a powerful design for testing putative environmental influences on child eating or weight. The design is also useful for studying questions concerning within-family variations in eating behavior or weight. Additional discussions on this topic in the broader child development literature are provided elsewhere[12–15]
High-risk Design	Do children who differ in familial vulnerability to obesity differ in specific eating patterns or food preferences?	In this design, comparisons between 'high-risk' and 'low-risk' children are conducted, with risk status typically defined on the basis of parental (typically mother) obesity status. High-risk children are more likely than low-risk children to have genes and home environments that are permissive to the development of obesity. The design may be more feasible than the discordant sibling design; however, this design may be less powerful (for certain purposes) than the discordant sibling design, which can match siblings for a shared home environment

eating behavior and responses to dietary interventions. It is concluded that BG designs represent more than the classic twin design and can be adapted creatively to the needs of diverse research questions.

Familial influences on child food preferences and eating behavior

Family correlation designs

Children tend to eat foods that they like.[16] Hence, it is possible that genetic influences on child food intake and body weight operate through food preferences. Studies have tested the association between child food preferences and those of other family members, most typically their parents. Child food preferences have been measured using questionnaires completed by parents or experimental procedures in which children taste and rank test foods.[21] On balance, the association between child and parent food preferences appears to be statistically significant but small in magnitude. Borah-Giddens and Falciglia[22] conducted a meta-analysis of five studies testing the association between child and parent food preferences, where 'child' was defined as any age up to age 25 y. Table 2 summarizes the studies and resulting parent–child associations. Their meta-analysis indicated a mean parent–child correlation of $r = 0.19$ for mother–child pairings and $r = 0.14$ for father–child pairings. Additional studies published since this meta-analysis reported comparable findings.[28]

Several investigators compared family correlations for food preferences among *biologically related* family members compared to *randomly assigned* 'pseudo-family' members. Pseudo-families were created by the investigators by 'decoupling' children from their biological-family member and then randomly assigning them a different (ie genetically unrelated) family member from the same cohort. Children's food preferences could then be correlated against biological-family members as well as pseudo-family members. The rationale for testing pseudo-families was to serve as a comparison to control for broader cultural factors (eg SES, geographic similarities) that might operate across families within the same study and induce familial correlations. This strategy bares some similarity to the adoption study methodology.[29]

Results from two studies indicate that family correlations are generally stronger among genetically related family members than pseudo-family members, although the differences are not large.[23,26] Birch's[23] study of 128 preschool children found a statistically significant correlation between mother–child food ratings for 10% of the foods, whereas the percent of statistically significant associations was nearly as high among pseudo-family members (8%). Pliner and Pelchat[26] had 55 Canadian mothers rate their own preferences for 139 foods, as well as those of their husbands and children using a questionnaire that addressed single foods, casseroles or 'mixed' foods, and condiments. Family correlations for food ratings were generated for biological-family members and pseudo-family members. Results indicated that all family correlations were significant, including those for pseudo-families. Associations were significantly greater for biological than pseudo-family pairings, especially when comparing siblings (see Table 3).

Family correlations have also been reported for child food intake measures. Laskarzewski *et al*[30] tested parent–child correlations for reported dietary intake measures among 294 families, in which children were 6 to 19 y of age. Families included 60 African-American and 234 Caucasian families; food intake was assessed by 24-h dietary recalls. Pearson's correlation coefficients tested parent–child associations for

Table 3 Mean associations for food preferences among a target child and other biological-family members or pseudo-family members, as quantified by the $\varnothing$ statistic

	Family member		
Family status	*Mother*	*Father*	*Sibling*
Biological family	0.20	0.20	0.50
Pseudo-family	0.09	0.11	0.18

Note: Adapted from Pliner and Pelchat.[26]

Table 2 Summary of studies examining food preferences within families

Study	*Age of children*	*Food measured*	*# Parent–child comparisons*	*Correlation coefficient*
Birch[23]	2 y 11 mo–5 y 7 mo	8 items. Generally accepted fruits, vegetables, sandwiches, snacks	126 M/C	0.14
			120 F/C	0.07
Logue *et al* (1988)[24]	Mean = 15.7 y	55 items. Representing seven major food groups	68 M/C	0.16
			54 F/C	0.19
Pliner (1983)[25]	19–24 y	47 items. Representing four major food groups	105 M/C	0.25
			105 F/C	0.25
Pliner and Pelchat[26]	2 y–6 y 11 mo	139 items. Most likely to be served to Canadian children	55 M/C	0.20
			55 F/C	0.20
Rozin *et al* (1984)[27]	17–23 y	22 items. Not readily accepted foods	34 M/C	0.19
			34 F/C	0.04

y = year; mo = months; M/C = mother–child comparison; F/C = father–child comparison. *Note*: Adapted from Borah-Giddens and Falciglia.[22]

Table 4 Sibling associations for eating measures, as quantified by an intraclass correlation coefficient (ρ) and 95% confidence intervals

Measure	*Unadjusted*[a] ρ	*Adjusted*[b] ρ	*Adjusted*[c] ρ
COMPX (%)[d]	0.10 (−0.25, 0.43)	−0.13 (−0.49, 0.26)	−0.26 (−0.64, 0.22)
Total energy intake (kJ)	0.39* (0.05, 0.65)	0.70** (0.43, 0.85)	0.49* (0.05, 0.77)
Fat intake (%)	0.66** (0.42, 0.82)	0.66** (0.38, 0.84)	0.53** (0.10, 0.79)
Carbohydrate intake (%)	0.67** (0.42, 0.82)	0.66** (0.39, 0.84)	0.57** (0.16, 0.82)
Protein intake (%)	0.61** (0.33, 0.79)	0.58** (0.25, 0.79)	0.66** (0.29, 0.89)

[a]$N = 32$ sibling pairs for these analyses. [b]Adjusted for age, gender, and ethnicity. $N = 26$ sibling pairs for these analyses. [c]Adjusted for age, gender, ethnicity, and BMI. $N = 18$ sibling pairs for these analyses. [d]COMPX = Compensation Index.[31,32] COMPX is defined as the defined as the difference in test meal intake, following low- and high-calorie preloads, divided by the difference in preload intake, all multiplied by 100%. COMPX is scaled such that 100% represents perfect compensation, scores that are progressively higher than 100% represent overcompensation, and scores that are progressively lower than 100% represent undercompensation. *$P < 0.05$, **$P \leq 0.001$. From Faith *et al.*[31]

energy intake, expressed per kg of total body weight. Results indicated significant associations of $r = 0.21$ (ie for families in which there was one parent and one child), $r = 0.31$ (ie for associations between the oldest parents and/or the oldest child), and $r = 0.25$ (ie for associations between all parents and all children).

Faith *et al*[31] studied 32 pairs of 3- to 7-y-old siblings whose food intake was measured in a child feeding laboratory. Children participated in an energy compensation study, whereby their *ad libitum* energy intake was measured at a multi-item lunch ~20 min following a low-energy (3 kcal) or high-energy (150 kcal) cherry-flavored liquid preload on two laboratory visits. The *ad libitum* lunch included macaroni and cheese, carrots, string cheese, grapes, and string beans. By pre- and postweighing foods at lunch, total energy intake following the two respective preloads was measured and converted to an index of energy compensation ability, the 'COMPX' (Compensation Index).[32] COMPX is computed as the difference in child intake of the two respective test meals divided by the difference in child intake of the two preloads, all multiplied by 100 (see Faith *et al*[31] for details). We also computed average total energy intake and average percent macronutrient intake across the two visits (disregarding the preloads). Results indicated significant sibling associations for total energy intake and macronutrient intake even when adjusting for covariates including BMI (see Table 4). There were no significant familial correlations for COMPX scores.

In sum, family correlation designs are useful for establishing that certain food preferences or eating styles are associated among family members. A significant family correlation may be due to genetic and/or home environmental factors that cannot be readily disentangled. Hence, this design provides limited support for a genetic effect *per se*, as much as a broader familial influence. On the other hand, given the paucity of data concerning genetic influences on child eating behavior, important information may be gleaned from preliminary studies that test familial correlations for eating traits such as energy compensation ability,[32] responsiveness to portion size manipulations,[1] eating in the absence of hunger,[33] and the reinforcing value of food.[34,35]

Twin designs

Plomin and Rowe[36] studied 91 twin pairs, mean age = 3.6 y, whose parents evaluated them on the Colorado Childhood Temperament Inventory (CCTI). The CCTI measures child sociability, emotionality, activity, attention span persistence, soothability, and reaction to food. 'Reaction to food' represented a child's temperamental reaction to different foods (eg 'Child consistently dislikes many kinds of foods.'). The investigators reported a significant heritable component to all CCTI subscales, except reaction to food. The intraclass correlation for reaction to food did not significantly differ from between monozygotic (MZ) and dizygotic (DZ) twin pairs ($r = 0.43$ and 0.49 for MZ twin pairs and DZ twin pairs, respectively).

Facliglia and Norton[37] studied 14 MZ twin pairs and 21 DZ twin pairs, 9–18 y old, to estimate the heritability of intake of 17 foods. Participants rated the frequency with which they ate each food during the week using a nine-point rating scale. Results indicated that three foods had heritability estimates that were significantly different from zero: sweetened cereal ($h^2 = 0.62$), cottage cheese ($h^2 = 0.63$), and orange juice ($h^2 = 0.70$).

Keski-Rahkonen *et al*[38] studied 5250 male and female twin pairs, 16 y of age, in terms of the heritability of reported breakfast eating patterns. Participants were enrolled in 'Finn Twin 16', a population-based study of five consecutive nationwide birth cohorts of Finnish twins. Twins were mailed a comprehensive questionnaire within 2 weeks of their 16th birthday, which included the following question: 'How often do you eat breakfast (eg sandwiches, milk, hot cereal, other similar foods) before going to school or going to work?' Response options were 'every morning', '3–4 times a week', and 'about once a week or less often'. Results indicated a heritable component to breakfast intake frequency among adolescent females ($h^2 = 0.31$–0.72) and males ($h^2 = 0.47$–0.63) across models. There was also evidence for shared (home) environmental influences on breakfast intake frequency in both sexes and that these effects were consistently greater among females than males. Keski-Rahkonen *et al* concluded 'Breakfast eating is moderated differently in adolescent boys and girls. Unlike boys, girls are much influenced by the family and pair-specific

environment. In girls, environmental influences may override genetically driven factors' (p 512).

There is also evidence from adult twin studies that certain eating patterns may be more 'environmentally loaded' among women than men. van den Bree *et al*[39] examined the genetic–environmental architecture of 'high fat–sugar–salt intake' and 'healthful eating habits' in 4500 adult twin pairs who completed a 24-h food frequency questionnaire. Results indicated that the magnitude of genetic and environmental influences on eating patterns differed significantly by gender. Heritability estimates for these traits were consistently larger for men, while estimates of home environmental influence were significant only among women.

In sum, the classic design has been used in a handful of studies to provide estimates of genetic, shared (home) environmental, and nonshared environmental influences on child eating traits. These estimates extend the information about genetic and environmental influences beyond a family correlation coefficient. An intriguing finding from existing studies is the apparent evidence for shared environmental influences on eating behavior, which implicates the home environment vis-à-vis the development of child eating patterns. This is in contrast to most twin studies of BMI, which generally fail to find a significant effect of the home environment (although some studies do report home environmental influences on BMI in pediatric samples[40,41]). If additional studies document home environmental influences on child eating behavior, this would raise a number of interesting questions concerning the nature of environmental influences on child eating patterns.

Discordant sibling designs

Young siblings and twins who are discordant for obesity status can provide valuable insights into the development of child eating patterns, although there can be important recruitment challenges.[12] Waxman and Stunkard[42] compared the home and school eating patterns of four pairs of brothers who were discordant for obesity status. Obese brothers ate significantly more than nonobese brothers at home (766 ± 290 *vs* 505 ± 183 kcal) and at school (901 ± 217 *vs* 500 ± 386 kcal) by direct observation. Mothers served obese children greater amounts of food than their nonobese children at home meals. Saelens *et al*[43] compared 18 pairs of obese and nonobese siblings with respect to maternal feeding practices on the original Child Feeding Questionnaire.[32] Results indicated that mothers showed greater weight concern towards the obese than the nonobese child; however, the degree of maternal feeding control was not related to child obesity status.

In sum, siblings who are discordant for weight can be compared on a host of behavioral or environmental measures to see which ones significantly discriminate the two groups (ie nonoverweight *vs* overweight siblings). As young siblings share the same home, this design can better control for environmental factors that may impact on child eating behavior or weight status (eg family SES, parental education, snack foods in the house). Discordant siblings designs therefore can enhance power to test putative environmental influences on child eating behaviors. These studies are typically less focused on estimating heritability *per se*. Dick *et al*[44] provide a thoughtful discussion on the use of discordant sibling designs for testing within-family variations in behavioral traits.

Cotwin control designs

The use of cotwin control designs for testing the development of child eating patterns is virtually nonexistent. This design capitalizes on the power of MZ twin pairs, who are perfectly matched for genotype, gender, age, and shared home environmental experiences. The cotwin control design, when used experimentally, randomly assigns one MZ twin to one condition/treatment and his or her cotwin to another condition/treatment. A useful example from the field of learning and child aggression comes from Diener *et al*.[45] In principle, this approach could be used to test powerfully the effects of environmental manipulations (eg portion sizes, food availability) on child eating behavior. We used the cotwin control design to test the effects of reinforcing fruit and vegetable intake on children's food selections and energy intake in the laboratory.[46]

In sum, the cotwin control design may offer an elegant strategy for testing gene-by-environment interactions vis-à-vis child eating behavior. It has not been used to date for these purposes, although it has been used to address some issues pertinent to adult obesity.[10,11] Pike *et al*[47] describe the use of MZ twins pairs to test the effects of differential parenting styles on child behaviors in the 'Nonshared Environment And Adolescent Development' project. Useful discussions on the power of the cotwin control design are provided by Carr and colleagues.[7–9]

'High-risk' designs

Another strategy used in the literature has been to compare the eating traits of children of obese parents *vs* nonobese parents. Parental obesity status, especially maternal weight, is a strong risk factor for childhood obesity.[48] Hence, comparing the eating patterns of children whose parents differ in obesity status can be a useful strategy for testing whether genetic vulnerability for obesity expresses itself through food intake. Wardle *et al*[49] examined the eating patterns of 428 4- to 5-y-old twins. In all, 200 children were from families with overweight/obese parents and 228 children were from families with normal-weight/lean parents. Families were recruited from the Twins Early Development Study (TEDS), a cohort of 10 000 twin pairs born in England and Wales in 1994 and 1995. Compared to children of normal-weight/lean parents, children of overweight/obese parents had significantly lower preference

ratings for vegetables, were more responsive to food cues, and had higher desire for drinks (all based on the maternal report). The two groups of children did not differ significantly with respect to food intake as measured by a food frequency questionnaire.

In another study, Stunkard *et al*[50] compared the behavior and metabolic profile of 3-month-old infants who were born at high risk ($N=40$) or low risk ($N=38$) for overweight on the basis of maternal prepregnancy body weight. Infants were measured on 3-day energy intake, nutritive sucking rate measured in the laboratory, total energy expenditure, sleeping energy expenditure, and other measures. Results indicated that the only variable discriminating the two groups at 3 months of age was rate of sucking, with high-risk children demonstrating a greater sucking rate than low-risk children (0.75 ± 0.25 *vs* 0.59 ± 0.26 sucks/s). Moreover, 3-month sucking rate but none of the energy expenditure measures predicted infant weight status at 12 months.

In sum, high-risk designs can be a useful strategy when ascertainment of related individuals, including twins, is not feasible. Identifying behavioral traits that discriminate children born at high risk *vs* low risk for overweight can help identify behavioral mechanisms by which genetic vulnerability expresses itself. These designs can be informative for cross-sectional comparison, but are most informative in the context of prospective analyses that test whether behaviors mediate the relationship between risk status and subsequent adiposity gain.

Familial influences on response to childhood obesity treatment

The previous studies addressed the familial bases of child food preferences and eating patterns. A different but related question is whether there is a familial influence on how much weight obese children lose in response to a dietary (including lifestyle) intervention? For example, might genetic factors influence child weight loss in response to the Traffic Light Diet?[51] The Traffic Light Diet is an extensively researched and behaviorally based eating program, prescribing 900–1300 kcals/day of energy. The program breaks food down into three groups based on nutrient density: 'green' foods, which children are encouraged to eat plentifully; 'yellow' foods, which children are encouraged to eat cautiously; and 'red' foods, which children are encouraged to eat infrequently. The diet has been shown to play an integral role in short- and long-term weight loss for obese children.[52]

Although data are limited, several reports document an association between parent and child weight loss following family-based behavioral treatment for childhood obesity. Epstein *et al*[53] tested the association between parent and child reduction in percent overweight among 76 families randomized to one of three behavioral interventions. Pooling across treatments, changes in parent and child percent overweight were significantly associated at 2 months of treatment ($r=0.39$, $P<0.002$) and at 6 months of treatment ($r=0.53$, $P=0.002$), although the association was no longer significant at 21-month follow-up ($r=0.13$, $P>0.10$).

Wrotniak *et al*[54] recently examined the association between change in parent BMI *z*-score and change in child BMI *z*-score among 142 family members who had participated in three randomized clinical trials of family-based behavioral treatments. The studies included a participating overweight child, 8–12 y old, and his/her participating parent. Across all families, the mean$\pm$s.d. reduction in BMI *z* for children and parents over 6 months was -1.3 (0.6) and -0.7 (0.5), respectively; the reduction for children and parents over 24 months were -0.8 (0.7) and -0.3 (0.5), respectively. In hierarchical regression analyses that controlled for sociodemographic variables and treatment conditions, change in parent BMI *z* predicted change in child BMI *z* over 6 months ($r^2=0.21$) and 24 months ($r^2=0.05$).

No pediatric intervention trials have explicitly recruited obese siblings to test family correlations for treatment response. However, reduction in percent overweight among children actively receiving behavioral intervention (ie 'targeted children') was associated with the reduction in percent overweight seen in their untreated siblings (ie 'nontargeted siblings'). Epstein *et al*[55] studied 56 targeted siblings who were randomly assigned to one of two behavioral interventions for increased activity or reduced sedentary behavior, with both groups receiving the Traffic Light Diet. A total of 89 nontargeted siblings were included in the study, with the number of siblings per family ranging from 1 to 4. Regression analyses indicated that change in nontargeted siblings' percent overweight was predicted by change in targeted siblings' percent overweight ($P=0.006$).

In sum, there appears to be a familial correlation for weight change following behavioral intervention for childhood obesity that includes diet modification. The association may dissipate during longer-term maintenance, suggesting that genetic or environmental influences on treatment response may differ for acute weight loss phase *vs* maintenance phase. No twin study to date has attempted formally to disaggregate genetic and environmental influences on obese children's response to dietary modifications for weight loss. Despite the recruitment challenges, such a study could provide fundamental insights into the role of genes and environment on dietary interventions during childhood.

Conclusions

A range of BG designs can provide insights into familial influences on child eating behavior and response to dietary interventions. These studies also set a strong foundation for

molecular studies that examine specific genes in relation to child eating patterns. A handful of studies have already begun to address these issues with pediatric samples[56,57] and may stimulate innovative BG strategies for testing candidate genes for food intake.[58] BG strategies offer more than the classic twin design and the 'mere' estimation of heritability; they can be adapted creatively to address a range of questions concerning child eating behavior, energy balance, and weight control.

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