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ARTICLE

# The Relationship between Diet and Porous Cranial Lesions in the Southwest United States: A Review

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**Abstract**
Bioarchaeologists commonly record porous cranial lesions (PCLs). They have varied etiologies but are frequently associated with nutritional anemia without a differential diagnosis. This article provides a literature review, evaluates diet in the US Southwest over time, and identifies issues with associating PCLs with poor diet in this region. Generally, diet was adequate across time and space. Although maize was a dietary staple, other food items such as rabbits and amaranth provided complementary micronutrients. PCLs exhibit varied morphologies, which generally correspond with age: those characterized by fine, scattered porosity are associated with younger ages at death. Variation in PCL morphology indicates different and sometimes unrelated etiologies. Nutritional anemia is an insufficient explanation for PCL frequency in the Southwest, partly because the diet was adequate across time.

**Resumen**
Las lesiones craneales porosas (LCP) son se encuentran comúnmente en análisis bioarqueologicos. LCP tienen variedad de etiologías, pero frecuentemente se asocian con anemia nutricional sin diagnóstico diferencial. Este artículo propone una revisión de la literatura, evaluación la dieta en el suroeste de los Estados Unidos a través del tiempo e identifica problemas con la asociación de las LCP con mala alimentación en esta región. En general, la dieta fue adecuada a través del tiempo y región. Aunque el maíz era considerado como un alimento básico, otros alimentos (por ejemplo, conejos y amaranto) proporcionaban micronutrientes complementarios. Las LCP exhiben variedad en morfologías, que generalmente corresponden con la edad. Las LCP, caracterizadas por una porosidad fina y dispersa, se asocian con la mortalidad en poblaciones jóvenes. La variación en la morfología de LCP indica etiologías diferentes y a veces sin relación. La anemia nutricional es insuficiente para justificar la frecuencia de LCP en el suroeste americano, en parte porque la dieta fue apropiada a lo través del tiempo.

**Keywords:** anemia; stress indicators; iron deficiency; Southwest; cribra orbitalia; porotic hyperostosis

**Palabras clave:** anemia; indicadores de estrés; deficiencia de hierro; suroeste americano; cribra orbitalia; hiperostosis porótica

Skeletal lesions used as indicators of environmental stress in archaeological contexts are often etiologically complex and may be generated through multiple biological pathways. Cribra orbitalia (CO) and porotic hyperostosis (PH) are descriptive terms for porous cranial lesions (PCLs) found on the orbit roofs and cranial vault that cause pitting, porosity, or thickening of the affected bones (Brickley 2018; Lewis 2017:194). Often, they are assumed to have the same etiology: acquired anemia from nutritional deficiencies. The association of PCLs with iron-deficiency anemia in past peoples is attributable to Moseley (1965:141), although he stipulated that "it does not seem necessary . . . to ascribe the same etiology to

all." Despite that caveat, PCLs, regardless of morphology, are consistently attributed to nutritional anemia without differential diagnosis (for more discussion, see Grauer 2019; Mays 2012; Ortner 2012). According to Ortner (2012:251), this association "has resulted in abnormalities being diagnosed as anemia when other diagnostic options are more probable. It also means that much of the literature on the prevalence of anemia . . . is likely to overestimate the true prevalence of the skeletal disorder in antiquity."

Failure to consider the complex biocultural processes that contribute to lesion formation may impede efforts to assess environmental impacts on patterns of morbidity and mortality in archaeological contexts. This article leverages data from an Ancestral Pueblo archaeological assemblage (AD 1100–1400s) and dietary data from a literature review to build on previous research examining relationships among PCLs, diet, and illness (Brickley et al. 2020; Cole and Waldron 2019; Mays 2012; Rivera and Mirazón Lahr 2017; Walker et al. 2009; Wapler et al. 2004).

Much like PCLs, anemia develops in varied conditions, including trauma, infection, renal failure, cancer, and metabolic disorders (Brickley et al. 2020; Ives 2018; Schattmann et al. 2016). This complexity may confound efforts to identify potential environmental factors such as diet and disease that contribute to PCL development. Additionally, studies suggest that CO and PH may have different etiologies (Rivera and Mirazón Lahr 2017; Schultz 2003; Walker et al. 2009), even when observed in the same person, because individuals can have comorbidities (see, e.g., Ives 2018). Recent work indicates different developmental trajectories for CO and PH, which may speak to their varied causes (O'Donnell et al. 2023). Because different biological pathways promoting PCL formation may involve different life-history trade-offs—for example, immune function versus growth (see Stearns 1992)—and associated costs such as constrained growth and elevated morbidity or mortality risks relevant to archaeological research questions, care should be taken in their interpretation.

This article reviews the literature and provides limited analyses to examine the association between PCLs and diet in the US Southwest. We examine bone changes in anemia and the relationship between anemia and PCLs in the Ancestral Pueblo world.

## Background

### *Anemia and PCLs*

Anemia occurs when red blood cells are low in number or dysfunctional, or hemoglobin concentration is lower than normal (Beutler 1988; WHO 2019a). Anemia has many potential causes and manifestations (Grauer 2019), and its presence signals an underlying health condition, but anemia itself is not a disease (Beutler 1988; Shine 1997). See Supplemental Table 1 for terminology and definitions.

PCLs are commonly attributed to acquired anemias, including iron-deficiency anemia (El-Najjar et al. 1975; Fink 1985; Hens et al. 2019; for discussion, see Brickley and Morgan 2023:343–344). Iron-deficiency anemia and anemia of inflammation (AoI) are the most often observed acquired anemias in clinical settings today (Ganz 2019). When AoI develops, it is often through iron sequestration or iron withholding (Spivak 2002), adaptive features of the immune system (Jurado 1997; Zucker et al. 1974).

AoI differs from iron deficiency because it typically does not deplete the body's total iron stores. Instead, serum ferritin—reflective of iron stores—is increased in AoI but decreased in iron-deficiency anemia (Nayak et al. 2018). Despite these differences, it is sometimes difficult for clinicians to differentiate between the two conditions (Schapkaitz et al. 2015), and they often co-occur (Ganz 2019). PCLs similar to those seen in congenital anemias are sometimes reported in individuals with iron-deficiency anemia (Eng 1958; Sheldon 1936), but they are rare compared to postcranial alterations in iron-deficiency anemia (Agarwal et al. 1970).

Vitamin $B_{12}$ deficiency, which can cause megaloblastic anemia, has been suggested as a cause of PCLs (Martinson 2002; Walker et al. 2009). However, $B_{12}$ deficiency in childhood, during which PCLs develop, is extremely rare (Lewis 2017; Mtvarelidze et al. 2009). This alone is inconsistent with the frequency of PCLs in the archaeological record. $B_{12}$ deficiency is not definitively linked with hyperplastic skeletal lesions in the medical literature, although there is some evidence of a relationship to bone loss (Stone et al. 2004); for discussion, see Oxenham and Cavill (2010) and McIlvaine (2015). A recent article by Brickley (2024) provides an extended discussion and critique of the association of anemia with PCLs.

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Although PCL presence alone only serves as a generalized indicator of stress, PCL morphology is likely indicative of its etiology (e.g., Brickley et al. 2020:7; Mays 2018a; Ortner 2003). In congenital anemias, PCLs are often characterized by marrow hyperplasia and pitting/porosity (Chaichun et al. 2021; Cooley and Lee 1925). In infection, PCLs are often superficial to normal bone and not associated with expansion of the marrow space (Grauer 2019:520). Inflammation, which results from immune responses to many conditions, may cause lesions characterized by bone loss (Epsley et al. 2020). Nutrient deficiency can cause superficial new bone and microporosity (scurvy; Ortner 2003; Snoddy et al. 2018) or spiculation of bone surfaces and larger pores (rickets; Mays 2008:185, 2018b).

### Developmental Factors Contributing to Lesion Formation

When observed in adults, PCLs are thought to represent developmental stress that an individual survived (Stuart-Macadam 1985), potentially signaling the initiation of critical physiological trade-offs that influence later morbidity and mortality risks (McPherson 2021; Temple 2019). Recent work indicates that the developmental window for CO closes around eight years of age, but PH may continue to form in later life-history phases (O'Donnell et al. 2023). Limited evidence suggests that pediatric males are more likely to develop CO than females (O'Donnell et al. 2022; Sheridan and Van Gerven 1997). There are also indications that different PCL morphologies may be associated with normal growth and development processes, such as fine porosity (Cole and Waldron 2019).

Early paleopathological studies of PCLs focused on the Southwest identified associations between lesion prevalence and age. In a study examining PH in Ancestral Pueblo skeletal samples, El-Najjar and colleagues (1976) suggested that PH rates were higher in children because of incompletely mineralized, thinner cranial vault bones, which provide limited space for marrow expansion. Lallo (1977:471) cited "synergistic" interactions between developmental physiology and iron deficiency as key factors in the development of PH, hypothesizing that individuals who experienced nutritional stress during the weaning period and those who experienced particularly rapid somatic growth (with its attendant energetic costs) would be more likely to develop lesions.

Although not focused on the Southwest, Stuart-Macadam (1985) used studies of bone marrow physiology to contextualize data on PCLs derived from clinical and anthropological sources. She suggested that greater bone plasticity in conjunction with limited capacity for marrow expansion increased the likelihood of lesion development in anemic juveniles. This study provided strong support for the theory that PCLs are representative of childhood episodes of stress—while calling into question the role of diet in their development.

### Diet and Skeletal Alterations

The association of PCLs with nutrient deficiencies, such as iron, $B_{12}$, and folate, and resulting anemia (El-Najjar et al. 1975; Walker et al. 2009) may arise from a mischaracterization of a population's typical diet; for additional discussion, see Cadwallader and colleagues (2012). In the Southwest, the connection of PCLs to acquired anemia related to maize dependency likely derives from a series of work by El-Najjar and coworkers (1975, 1976). This research analyzed individuals from Basketmaker II–III and Pueblo I–V sites in Arizona and New Mexico, including those from Chaco Canyon (individuals from Chaco Canyon are included in our study).

The 1976 study examined 539 crania for evidence of PCLs and found that rates varied with ecological context: populations living in canyon bottoms exhibited higher rates of PCLs than those living on sage plains. The authors attributed this result to dietary variation, hypothesizing that maize-dependent populations with less access to faunal sources of iron were more likely to develop PCLs (El-Najjar et al. 1976). The subsequent observation that PCL frequencies increased with the introduction of maize agriculture in the Southwest (Berry 1984:264–265, citing Lallo et al. 1977) further established the relationship between maize consumption, iron-deficiency anemia, and PCLs in the archaeological literature.

Using El-Najjar and colleague's work as a reference, Ferguson (1980) concluded that iron-deficiency anemia was common in those who lived in Tijeras in New Mexico and that diarrheal disease or prolonged breastfeeding without iron supplementation were likely factors in its development. Merbs and Miller's (1985) collection of research papers includes four chapters devoted to PCLs, in which

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iron-deficient maize-based diets are consistently cited as contributors to lesion development. For example, Walker (1985) examined PCLs in samples from sites also represented in our study: Chaco Canyon and Tijeras. In addition to iron-deficient diets, Walker hypothesized that other factors, including breastfeeding, parasitism, and other nutritional deficiencies, may have contributed to the development of PCLs in Southwest archaeological contexts. These works were at the forefront of research at the time they were written and were designed to identify stressed individuals within populations, the underlying environmental factors that contributed to PCL formation, or both.

Although PCL frequencies are thought to increase with the introduction of maize agriculture (Berry 1984:264–265, citing Lallo et al. 1977), their presence and prevalence do not necessarily have a relationship with maize consumption or reliance (Reinhard 1988; Stodder 1989; Ubelaker 1992; Walker 1986; see also Rothschild 2012). In addition to the dietary implications of maize agriculture, other changes occur with its adoption, including population growth and population aggregation.

Maize was a dietary staple for many Southwest groups (Brand 1994; Hard et al. 1996; Holliday 1996; Mays 2008; Minnis 1989). Although there are many problems with a diet solely dependent on maize (if such a diet exists), the nutritional-deficiency argument centers on its iron content. Maize is low in iron and can limit iron bioavailability—the body's ability to absorb iron. A study by Moore (1968) estimates iron absorption from maize at 5%. The presence of phytic acid content in maize can also limit iron bioavailability by acting as a chelating agent. Iron bioavailability differs based on the type of iron in food sources. Heme iron from meat, fish, and other seafood is the most bioavailable. Nonheme iron is available in grains, seeds, nuts, and dark leafy greens.

Even when maize is a dietary staple, other food items are incorporated into the diet (Cadwallader et al. 2012), as was the case in the ancient Southwest (Adams 2008:Table 4.3; Huckell and Toll 2004: Tables 3.1–3.5). Southwest groups ground maize using limestone or treated it with lime or ash, enhancing its mineral and nutritional content (Beck 2001:190; Ezzo 1994:271; Huss-Ashmore et al. 1982:400; Snow 1990:293). The nutrient content of maize is also enhanced by foods consumed alongside it, including beans, squash, tubers, prickly pear, goosefoot, yucca, pine nuts, amaranth, and beeweed (Anschuetz 2006; Kellner and Schoeninger 2007; Kinder et al. 2017; Reinhard 1992; Reinhard and Danielson 2005; Watson 2008). The diet also included meats and animal proteins from turkeys, rabbits, prairie dogs, artiodactyls, and fish (Badenhorst et al. 2019; Dombrosky et al. 2020; McKusick 1982; Rawlings and Driver 2010; Snow 2002).

## Methods and Case Study

We examined the distribution of PCLs by individual age and the percent frequency of PCLs alongside evidence for diet from multiple Southwest sites and locales. We expected that PCL types would show patterning by age and that evidence for micronutrient deficiency (scurvy or rickets) would be lacking from skeletal remains, as supported by the literature and by faunal, coprolite (including paleoethnobotanical), and other dietary data.

We included data from 86 Ancestral Pueblo individuals under age 16 who lived in what is now New Mexico between AD 1000 and 1400. We selected this age-restricted assemblage because PCLs develop in children, although adults may maintain evidence of PCLs throughout their lives (Brickley 2018; O'Donnell et al. 2022; Stuart-Macadam 1985), and PCLs in children are unlikely to have undergone extensive remodeling. It is worth noting, however, that remodeled and remodeling PCLs are found in children, including those Ancestral Pueblo individuals in our study (O'Donnell 2019). Lallo (1977) argued that skeletal remodeling is a methodological problem in any study of PCLs, and the rate and extent to which they remodel under varied environmental conditions remain uncertain. In addition to influencing assessments of PCL presence and prevalence, remodeling may affect assessments of the morphological characteristics of lesions.

The Museum of Indian Arts and Culture (MIAC) staff conducted consultation on O'Donnell's behalf. Permission was granted for all data included here; see O'Donnell, Valesca Meyer, and Ragsdale (2020) for a detailed explanation of the process. All methods used here are nondestructive. The case study is subject to archaeological issues, including small assemblage size, but power analysis indicates a sufficient (0.83; alpha = 0.05) sample for logistic regression estimates.

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### Cultural Context

Most individuals included in the case study lived between AD 1100 and 1400, which saw significant demographic and environmental disturbances, large-scale migrations, regional depopulation, and aggregation (Cordell 1995; Kohler et al. 2010). In the late 1200s, the Four Corners region experienced depopulation, often attributed to the "Great Drought" (Schlanger and Wilshusen 1993), although this explanation is likely too simplistic (Crown et al. 1996; Kohler et al. 2010; Ortman 2010; Roney 1995; Schillaci and Lakatos 2016; Varien 2010). The population had survived severe droughts equivalent in intensity or worse than the Great Drought, and some areas were buffered from the drought's effects (Benson et al. 2007).

There is ethnographic, archaeological, and bioarchaeological evidence for inequity and unequal distribution of resources in Puebloan society (Eggan 1950; Levy 1992; Martin and Osterholtz 2016). None of the individuals in this sample with associated burial information had grave goods or burial treatment indicative of elevated social status (O'Donnell and Moes 2021). However, some individuals and groups likely experienced heightened stress related to social status, sex, or age (see Table 1 for assemblage information).

### Skeletal Analysis and Data Collection

Skeletal analyses were done using standard methods (Buikstra and Ubelaker 1994; Ortner 2003; see O'Donnell 2019). Age was estimated using dental and skeletal development (AlQahtani et al. 2010; Baker et al. 2005; Buikstra and Ubelaker 1994; Scheuer and Black 2000, 2004). Sex estimates are not possible in prepubertal children without doing destructive analyses (O'Donnell et al. 2017; Scheuer and Black 2000), so no attempts at sex estimation were made.

Each individual included had at least one observable eye orbit; PCL morphology scoring followed Stuart-Macadam (1991a:Figures 9.3a, 9.3b). Table 2 and Figure 1 provide descriptions and depictions of morphology, respectively.

Expansion of the diploic space, identified in dry bone when the marrow spaces are enlarged, was recorded. Expansion was observable in the orbit roof of six of 32 individuals and in the vault bones of 10 of 62 individuals; for additional depictions, see Figure 2, Grauer (2019:Figure 14.24), and Brickley and coauthors (2020:Figure 9-2). Because expansion was recorded in bones broken postdeposition, there may be some comparability issues. However, marrow hyperplasia (thickening of dry bone) is observable at postdepositional breaks (Mays 2012:291), and expansion outside the norm is identifiable (see O'Donnell et al. 2023:Figure 1B–1D).

### Assessing Diet in the Southwest

In addition to the literature review, we aggregated information from paleofeces (coprolite)/paleoethnobotanical studies to characterize the Ancestral Pueblo diet (see Table 3). The dates for coprolites range from 2900 BC to AD 1450, with the majority of coprolites being from PII (AD 900–1150) and PIII (AD 1150–1325). Not all dietary components are equally represented in coprolites. Some food items, such as beans, squash, and meat, are wholly digested or otherwise difficult to identify in coprolites (Stiger 1977:16). In contrast, maize is not completely digested and may be overrepresented in coprolites (Reinhard 1988:144, 147). However, coprolites enable the direct assessment of human diet and parasitism via the presence of macroscopic (e.g., animal bones, seeds) and microscopic inclusions (e.g., pollen, phytoliths; Reinhard and Bryant 1992). Small animal bone fragments in coprolites provide evidence for meat consumption (Clary 1983; Gillespie 1981; Reinhard and Bryant 1992). The accuracy of the frequencies of coprolites is dependent on the number of observations (presented in Supplemental Table 2).

Data from multiple studies (Table 3; Supplemental Tables 2 and 3) were used to plot temporal patterns of PCL presence/absence. Analyses of lesion morphology were conducted on the 86 individuals in the case study.

### Analytical Methods

Logistic regression in Stata 18 (StataCorp 2017) was used to estimate odds ratios (OR) for the associations between PCLs and estimated age at death. OR are commonly used in clinical research to

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**Table 1.** Individuals Included in This Study; Counts of Individuals by Site and Age Group (One-Year Intervals).

| Region | Dates | Midpoint | Number of Individuals | Age Range and Number of Individuals |
|---|---|---|---|---|
| East Mesa Verde [also called Totah] | 1000–1300 | 1150 | 11 | 2–2.9 years: 4<br>5–5.9 years: 2<br>7–7.9 years: 1<br>9–9.9 years: 2<br>10–10.9 years: 1<br>11–11.9 years: 1 |
| San Juan Basin | 800–1325 | 1063 | 10 | 6 months–11.9 months: 2<br>1–1.9 years: 1<br>2–2.9 years: 1<br>3–3.9 years: 3<br>4–4.9 years: 1<br>5–5.9 years: 1<br>9–9.9 years: 1 |
| Northern Rio Grande | 1050–1600 | 1325 | 13 | 6 months–11.9 months: 1<br>1–1.9 years: 1<br>2–2.9 years: 1<br>3–3.9 years: 1<br>4–4.9 years: 2<br>5–5.9 years: 3<br>6–6.9 years: 1<br>8–8.9 years: 3 |
| Gallina District | 900–1300 | 1100 | 7 | 6 months–11.9 months: 1<br>1–1.9 years: 1<br>4–4.9 years: 1<br>5–5.9 years: 1<br>8–8.9 years: 1<br>11–11.9 years: 1<br>13–13.9 years: 1 |
| Middle Rio Grande | 1262–1600 | 1431 | 38 | 6 months–11.9 months: 11<br>1–1.9 years: 5<br>2–2.9 years: 8<br>3–3.9 years: 6<br>4–4.9 years: 2<br>7–7.9 years: 2<br>8–8.9 years: 2<br>13–13.9 years: 2 |
| Mogollon (Jornada) | 700–1450 | 1075 | 4 | 6 months–11.9 months: 2<br>4–4.9 years: 1<br>6–6.9 years: 1 |
| Mogollon (Mimbres) | 1000–1450 | 1225 | 2 | 7–7.9 years: 1<br>9–9.9 years: 1 |
| Rio Abajo | 1250–1450 | 1350 | 1 | 6 months–11.9 months: 1 |
| Total Individuals | | | 86 | 6 months–11.9 months: 18<br>1–1.9 years: 8<br>2–2.9 years: 14<br>3–3.9 years: 10<br>4–4.9 years: 7<br>5–5.9 years: 7<br>6–6.9 years: 2<br>7–7.9 years: 4<br>8–8.9 years: 6<br>9–9.9 years: 4<br>10–10.9 years: 1<br>11–11.9 years: 2<br>13–13.9 years: 3 |

*Note:* All individuals included in this study are from pre-Spanish contact site components.

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**Table 2.** Lesion Types (Morphology) and Definitions Used in the Study.

| Score | Descriptions |
|---|---|
| 0 | Normal bone (no lesion) |
| 1 | Capillary-like impressions |
| 2 | Scattered, small foramina (1 mm or less) |
| 3 | Large and small scattered foramina (1 mm or greater) |
| 4 | Foramina linked in trabecular structure (coral-like) |
| 5 | Outgrowth in trabecular form from the outer table (coral-like) |
| 6 | New bone formation on outer cortex |
| *Cranial Vault and Orbit – Thickening, Thinning, Marrow Hyperplasia* | |
| Normal | Normal diploë (marrow spaces) and thickness |
| Marrow Hyperplasia | Characterized by enlargement of spaces in the diploë (expansion of marrow space), which would have been caused by marrow expansion. The enlarged spaces would have been occupied by bone marrow in life (see also Brickley et al. 2020:Figure 3–6c). |
| Lamination | Characterized by multiple, thin layers of bone that is "compact" between the diploë; can be accompanied by expansion of the bone or not (vault only). |
| Thickening | Thickening of the orbit roof or vault bones out of character with the age of the individual, which could be due to hyperplasia, new bone, or another cause. |
| Thinning | Can refer to cortical thinning, caused by marrow hyperplasia or thinning of the diploë (see also Brickley et al. 2020:Figure 9-2b). No thinning was observed in broken cross sections in this study. |

*Note:* See Figures 1 and 2 for illustrations of lesion types and bone cross sections. These scores were also used for porotic hyperostosis (see Stuart-Macadam 1991a:Figure 9.3a, 9.3b; orbits schematics follow Figure 9.3b).

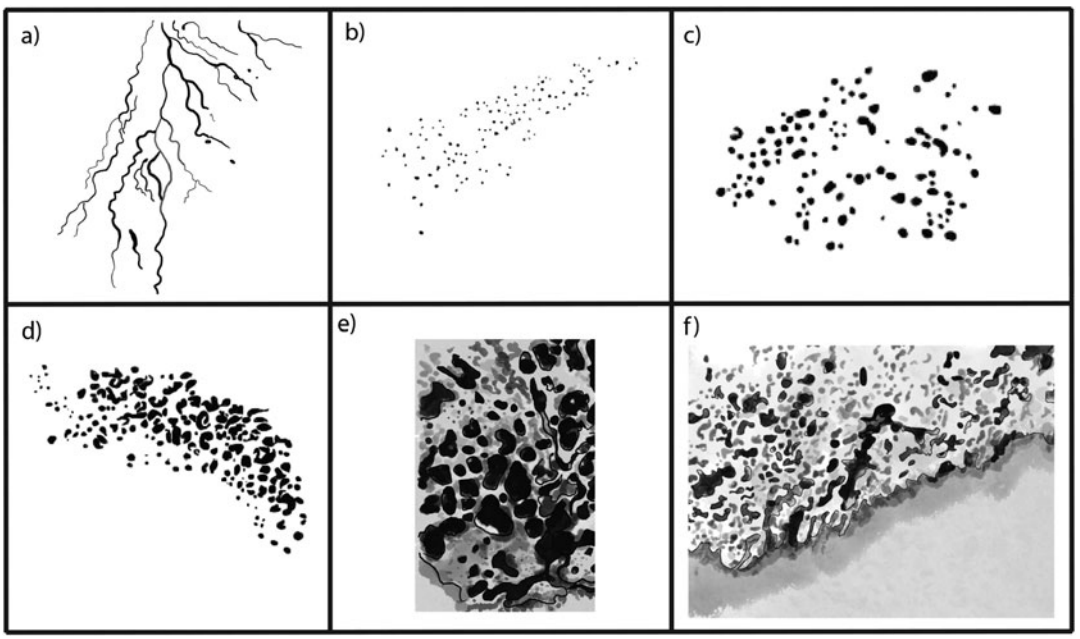


**Figure 1a.** Illustrations of CO and PH morphology (Types 1–6) described in Table 2. 1a-a Type 1, capillary-like impressions; 1a-b Type 2, scattered, small foramina; 1a-c Type 3, large and small scattered foramina; 1a-d Type 4, foramina linked in a trabecular structure; 1a-e Type 5, trabecular outgrowth from the outer table; 1a-f Type 6, new bone deposited on outer cortex. All illustrations by the first author.

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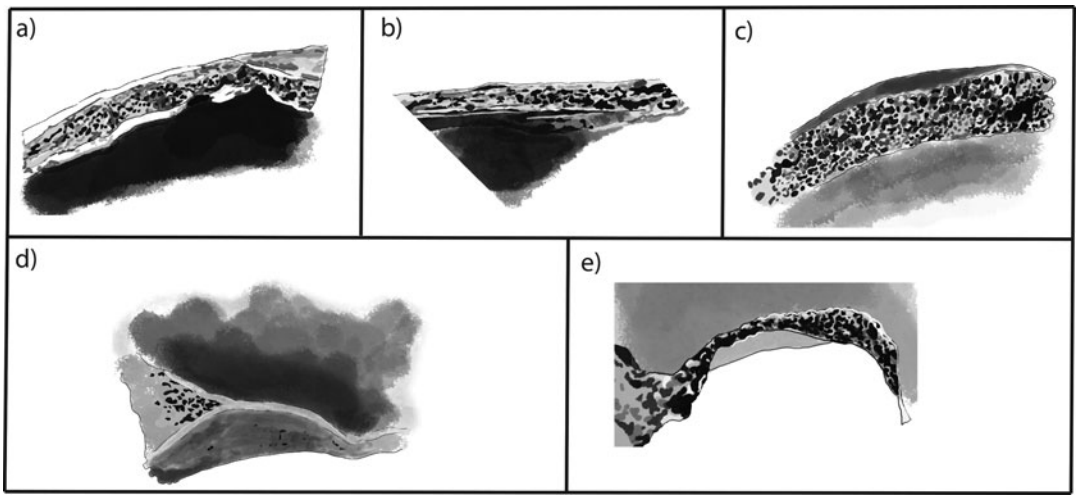


**Figure 1b.** Illustrations of cross sections of vault (Figure 1b-a and 1b-c) and orbit bones (1b-d, 1b-e). (1b-a) Normal vault cross section; (1b-b) lamination of diploë without expansion of the marrow space; (2c) expansion of the marrow space and thickening, coupled with thinned outer/inner cortices; (1b-d) normal orbit; (1b-e) orbit with expansion of the marrow space accompanied by thickening of the bone. Orbit illustrations are from the posterior (looking into the endocranium). All illustrations by the first author. (See also Brickley and colleagues 2020:Figure 9-2; O'Donnell et al. 2023:Figure 1).

examine the odds of developing an outcome following an exposure (e.g., lung cancer in those who smoke). Here, we use OR to examine the odds of having a lesion characterized by certain morphology, such as Type 6 (outcome), based on individual age at death (exposure). Logistic regression was done with regions pooled by age at death, rather than focusing on individual regions. For region- and site-level analyses, see O'Donnell (2019:207–214).

An OR > 1 indicates greater odds of having a lesion, an OR < 1 indicates lower odds of having a lesion, and an OR = 1 indicates no difference in odds of having a lesion. OR are presented in forest plots (coefplot; Jann 2013), which provide visual representations of the analysis results. A red line is placed at 1. Significant results to the right of the line indicate increased odds of having an outcome, whereas those to the left of the line indicate decreased odds of having an outcome.

The regression formula is

$$\Pr\left(Lesion\ Type_i = 1\right) = \frac{1}{1 + e^{-(\beta_0 + \beta_1 age_i + u_i)}}$$

Index $i$ is an individual observation, $\beta$ is the coefficient estimated using regression, *age* is individual age at death, and $u$ is the error term. *Lesion Type* is the outcome of interest (*Lesion Type* = 1 if the outcome is observed; Lesion Type= 0 if the outcome is not observed). For example, Lesion Type 5 can be "0" absent or "1" present. The variable $age^2$ was included in the logistic regression analyses to account for nonlinear relationships between age at death and PCL presence/absence but was insignificant for all analyses, so results are provided for age alone.

Balloon plots showing the frequency of dietary inclusions and PCLs (size and color of the "balloon") were made using RStudio's ggplot (RStudio Team 2016). The ggplot package was used to plot density and smoothed percent frequency curves.

## Results

### *Diet in the Southwest*

Maize was ubiquitous throughout time, but there was substantial dietary variety (Figure 2a). Squash and beans are found at lower frequencies than maize, likely due to complete digestion. Chenopods and amaranth (cheno-ams) are in several categories in Figure 2a (cheno-ams, goosefoot). Other

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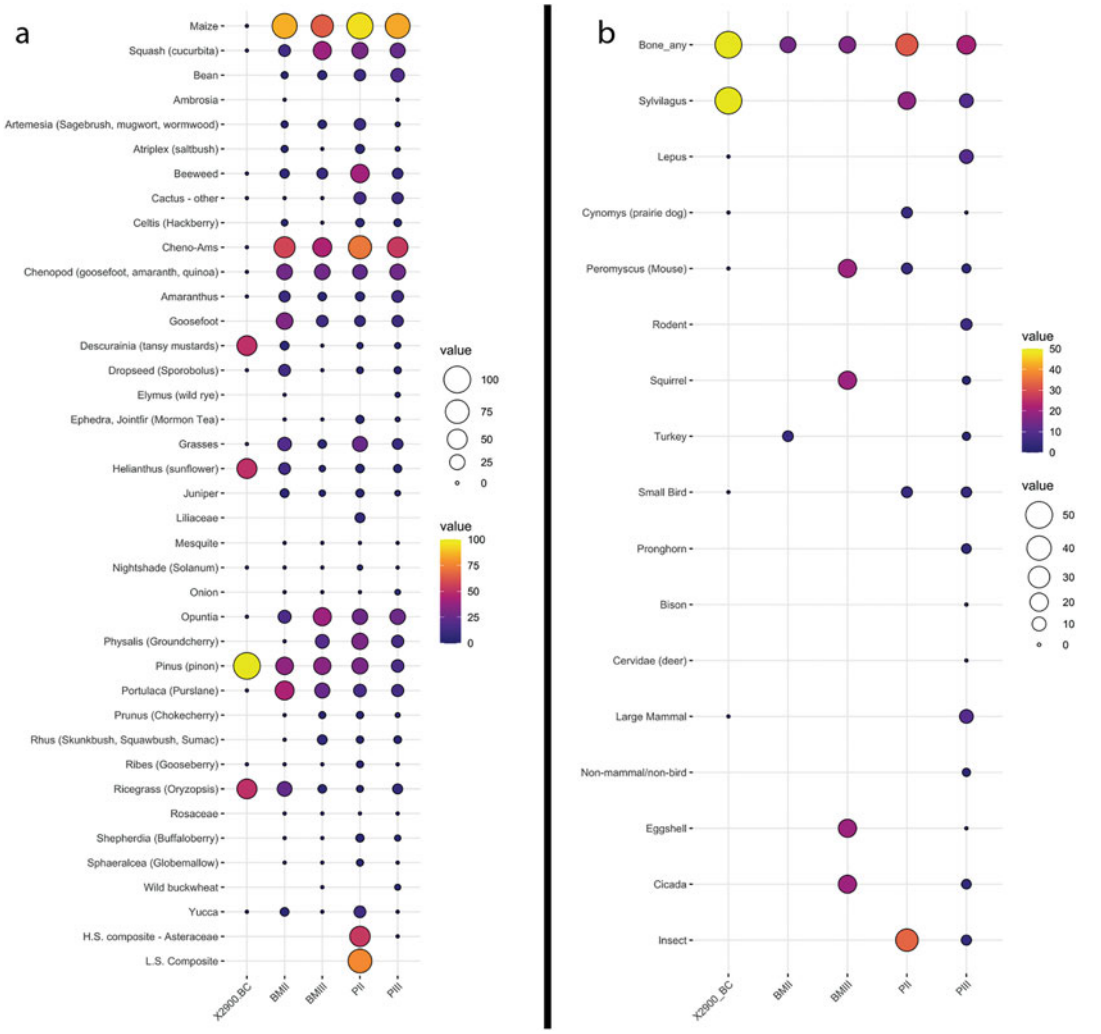


**Figures 2a–2b.** Balloon plots show the percent frequency of coprolites with pollen or plant (2a) / animal remains (2b) across time. The "balloon" increases in size with increasing frequency, and the color increases in temperature following that same pattern. For the number of coprolites per site, see also Supplemental Figure 1 and Supplemental Table 2; references for data are shown in Table 3. Abbreviations: BMII (1500 BC–AD 50), BMIII (AD 500–750), PI (AD 750–900), PII (AD 900–1150), PIII (AD 1150–1325), PIV (AD 1325–1550). (Color online)

common food items are pine nuts (*Pinus*), grasses, beeweed (*Cleome*), sunflowers (*Helianthus*), purslane (*Portulaca*), and prickly pear (*Opuntia*). Figure 2b shows that large and small game animals, including rabbits, were part of the diet across time.

## *PCL Presence and Morphology, Age at Death, and Diet*

The most common PCL morphology is Type 2. Type 4 morphology in the orbit and on the parietal bones shows a bimodal distribution (Figures 3a, 3c). The frontal bone is the least likely to exhibit lesions (Figure 3b). Figures 3 and 4 demonstrate that the probability of having PCLs decreases with age: their presence is associated with younger ages at death in this sample (Figure 4a). CO characterized by "simple porosity" (Type 2) is associated with younger ages at death (Figure 4b, purple circle). PH characterized by Type 6 on any vault bone is associated with younger ages at death (Figure 4b, open blue circle). In analyses examining the relationship between PCL morphology and age in individuals under 10 years old (Figure 4c), we find similar results. There is one notable difference: in

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**Table 3.** Sources of Coprolite Dietary Data.

| Site or Region | State | Dates* | Data Sources |
|---|---|---|---|
| *Coprolite Studies* | | | |
| Antelope House | AZ | PII (1000)<br>PII/PIII (1090–1300)<br>PIII (1150–1300) | Fry and Hall 1986; Stiger 1977; Reinhard 1988: Table 5; Sutton and Reinhard 1995; Williams-Dean and Bryant 1975 |
| Inscription House | AZ | PIII (1150–1300) | Brand 1994; Reinhard 1988:Tables 9 and 22; Stiger 1977 |
| Ventana Cave | AZ | PII/PIII (1000–1450) | Reinhard and Hevly 1991 |
| Mesa Verde | CO | BMIII (1–750)<br>PIII? | Stiger 1979 |
| Step House | CO | BMIII (500–750)<br>PIII (1150–1300) | Brand 1994; Reinhard 1988:Table 5; Stiger 1977 |
| Hoy House | CO | PII (1090–1150)<br>PIII (1250) | Reinhard 1988:Table 5; Scott 1979; Stiger 1977 |
| Lion House | CO | PIII (1240) | Stiger 1977:26, Table III |
| Atlatl Cave | NM | 2900 BC | Clary 1983 |
| Bat Cave | NM | BMII-PII (200–1000) | Trigg et al. 2000 |
| Pueblo Alto | NM | PII (1020–1100) | Clary 1983, 1987 |
| Pueblo Bonito | NM | PII (850s–1100s) | Clary 1983 |
| Kin Kletso | NM | PIII (1125–1300) | Clary 1983 |
| Chaco Canyon | NM | | Clary 1983 |
| Aztec Ruins – West Ruin | NM | PII (1090–1105) | Cummings et al. 2009; Stiger 1977 |
| Salmon Ruin | NM | PII (1090–1150) | Reinhard 1988:Table 5, 2006 |
| Turkey Pen Ruin | UT | BMII (1–400) | Battillo 2017, 2019 |
| *CO and PH Studies* | | | |
| Antelope House | AZ | BMII–BMIII (400–700); PII/PIII (900–1300) | El-Najjar 1986 |
| Canyon de Chelly | AZ | BM (300–700)<br>PII/PIII (700–1300) | El-Najjar et al. 1976 |
| Canyon del Muerto | AZ | Early BM (700–1500 BC) | Zaino 1967 |

(*Continued*)

**Table 3.** Sources of Coprolite Dietary Data. (*Continued.*)

| Site or Region | State | Dates* | Data Sources |
|---|---|---|---|
| Carter Ranch | AZ | PIII (1100–1225) | Danforth et al. 1994 |
| Grasshopper Pueblo | AZ | PIII/PIV (1225–1450) | East 2008:Table 100 |
| Houck | AZ | PI/PII (900–1350) | Zaino 1967 |
| Inscription House | AZ | PIII (1250–1300) | El-Najjar et al. 1976 |
| Kayenta | AZ | PII; PIII | Stodder 1989:Table 42 |
| Navajo Reservoir | AZ | PI–PIII (700–1100) | El-Najjar et al. 1976 |
| Oak Creek Pueblo | AZ | PII– PIV (1125–1400) | Taylor 1985 |
| Point of Pines | AZ | PII–PIV (1240–1450) | East 2008:Table 100 |
| Turkey Creek Pueblo | AZ | PIII (1225–1286) | East 2008:Table 100 |
| Animas La–Plata | CO | PI (750–900) | Perry et al. 2010 |
| Black Mesa | CO | BM–PII (700–1100) | Stodder and Martin 1992 |
| Mesa Verde | CO | PI–PIII | Stodder 1989, 1996; Stodder and Martin 1992:Tables 2 and 3 |
| Sand Canyon Pueblo | CO | PIII (1240s–1280s) | Kuckelman and Martin 2007 |
| Chaco Canyon | NM | PII (700–1100) | El-Najjar et al. 1976 |
| Pueblo Bonito | NM | PII (850–1100) | Harrod 2012; Marden 2011 |
| Chaco Small Sites | NM | PII/PIII (900–1178) | O'Donnell 2019 |
| Kin Kletso | NM | PIII (1125–1300) | O'Donnell 2019 |
| Pueblo Alto | NM | PII (1020–1100) | Akins 1987 |
| Kin Bineola | NM | PII (940–1120) | Harrod 2012 |
| Salmon Ruin | NM | PII (900–1100);<br>PII (1090–1150) | Doebley and Bohrer 1983; Shipman 2006; Stodder and Martin 1992:Table 5 |
| Aztec Ruin | NM | PII (1090–1105)<br>(West Ruin) | Harrod 2012 |
| Arroyo Hondo | NM | PIII–PV (1300–1600) | Palkovich 1980, 1985 |

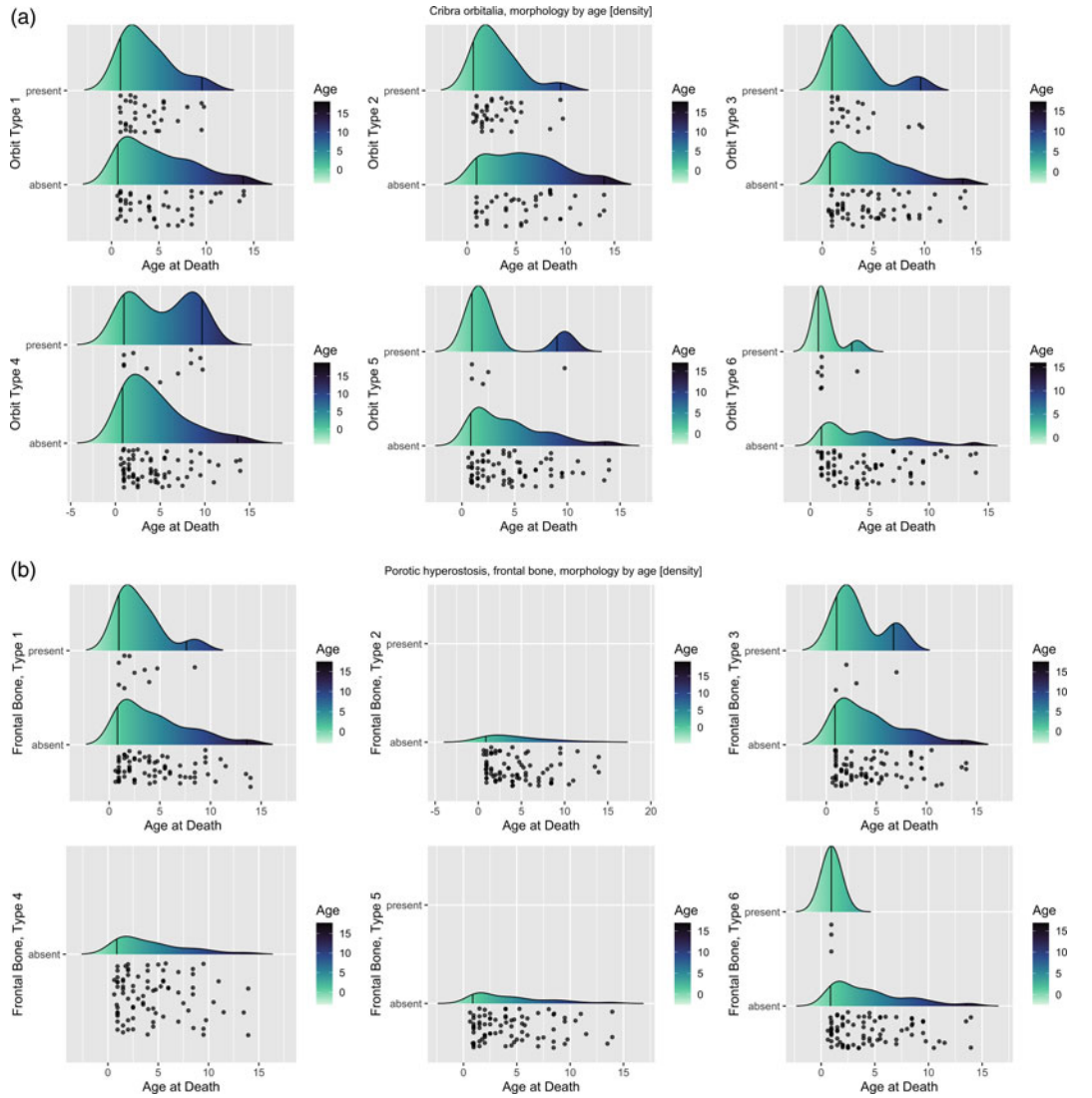
(*Continued*)

**Table 3.** Sources of Coprolite Dietary Data. (*Continued.*)

| Site or Region | State | Dates* | Data Sources |
|---|---|---|---|
| Casamero | NM | PII (900–1150) | O'Donnell 2019 |
| Gran Quivira | NM | PIII–PV (1300–1600) | El-Najjar et al. 1975; Stodder and Martin 1992 |
| Hawikku | NM | PIII–PV (1300–1600) | Stodder 1996:Table 5.2 |
| La Plata Highway | NM | PII/PIII (1000–1300) | O'Donnell 2019 |
| Pa'ako | NM | PII–PV (1100–1600) | Ferguson 1980 |
| San Cristobal | NM | PIII–PV (1300–1600) | Stodder 1996:Table 5.2 |
| Northern Rio Grande | NM | PII–PV (1050–Historic) | O'Donnell 2019 |
| Middle Rio Grande | NM | PIII/PIV (1260–1400) | O'Donnell 2019 |
| Gallina District | NM | PII/PIII (900–1300) | O'Donnell 2019 |
| Jornada Mogollon | NM | BM–PIV (700–1375) | O'Donnell 2019 |
| Mimbres Mogollon | NM | PII/PIII (1000–1325) | O'Donnell 2019 |
| Time Period | *Midpoint* | *Date Range* | |
| Basketmaker (BM) II | 775 BC | 1500 BC–AD 50 | |
| BMIII | AD 625 | AD 500–750 | |
| PI | AD 825 | AD 750–900 | |
| PII | AD 1025 | AD 900–1150 | |
| PIII | AD 1238 | AD 1150–1325 | |
| PIV | AD 1438 | AD 1325–1550 | |

*Notes:* These data are used in Figure 2; pollen studies generally required >=200 grains; CO and PH data are used in Figure 6. The bottom portion of this table provides the date ranges used along with midpoints. Refer to Supplemental Tables 2 and 3 for counts.

* Although there are some difficulties with using the Pecos Classification to compare areas throughout the Southwest (not all sites or groups fit neatly into the categories), it is used here to generalize trends across time and space.




**Figures 3a–3b.** These figures provide density curves by age at death for lesion Types 1–6. (3a) CO, orbit; (3b) PH, frontal bone; (3c) PH, parietal bones; (3d) PH, occipital bone. Along the x-axis at the bottom of each figure are points indicating each individual in the assemblage. (Color online)

the younger age group, Type 3 PH on the parietal bone is associated with younger ages at death (Figure 4c, orange triangle).

CO follows no discernible pattern across time, and PH increases as time progresses (Figure 5). However, the CO or PH category in this figure represents instances where authors pooled the lesions (implying an inferred shared etiology). If CO and PH had been broken into two categories, the frequency patterns observed might have been altered.

Few individuals in the case study had a suite of skeletal changes consistent with scurvy ($n = 3$; 3.5%) or rickets (no identified cases), and neither produced pathognomic skeletal alterations (Brickley et al. 2020:53, 95).

## Discussion

PCL morphology should be reflective of lesion etiology (e.g., Brickley 2024; O'Donnell, Hill, et al. 2020). We found that expansion of the diploic space (which would be reflective of anemia) is rare,

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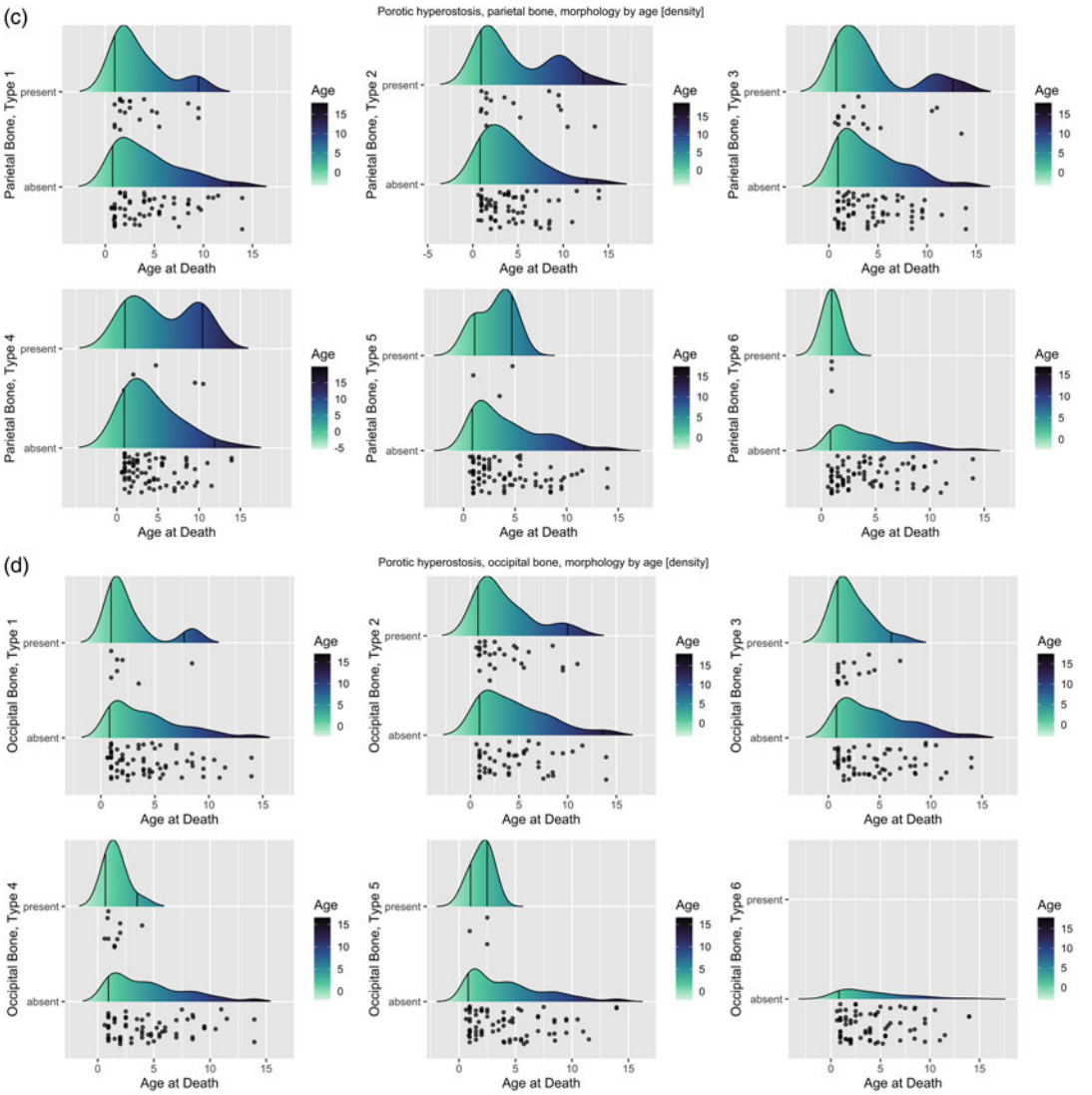


**Figures 3c–3d.** These figures provide density curves by age at death for lesion Types 1–6. (3c) PH, parietal bones; (3d) PH, occipital bone. Along the x-axis at the bottom of each figure are points indicating each individual in the assemblage. (Color online)

whereas lesions characterized by fine porosity are more common. The diversity of PCL morphology implicates varied processes in their formation. These could include immune responses such as inflammation, infections, neoplasia, nutrient deficiencies, and trauma, although no skeletal injuries were identified in our study. Considering morphology in concert with archaeological evidence for diet can help researchers identify and rule out potential diagnoses and underlying biological mechanisms that promote lesion formation (Brickley et al. 2020:249; Mays 2018a).

Early studies of PH in the Southwest, which largely attributed lesion formation to maize-dependent diets, noted that ecological factors and dietary variation likely influenced lesion rates (see El-Najjar et al. 1976; Walker 1985). We highlight these studies because the present sample overlaps with theirs. Walker (1985) and El-Najjar and colleagues (1976) considered the potential role played by other conditions—parasitism and, for adults, breastfeeding—in the development of anemia, but limited available evidence led El-Najjar and coauthors to conclude that dietary factors were a more likely cause of lesion formation in the Southwest. More recent ethnobotanical and coprolite studies provide a more nuanced

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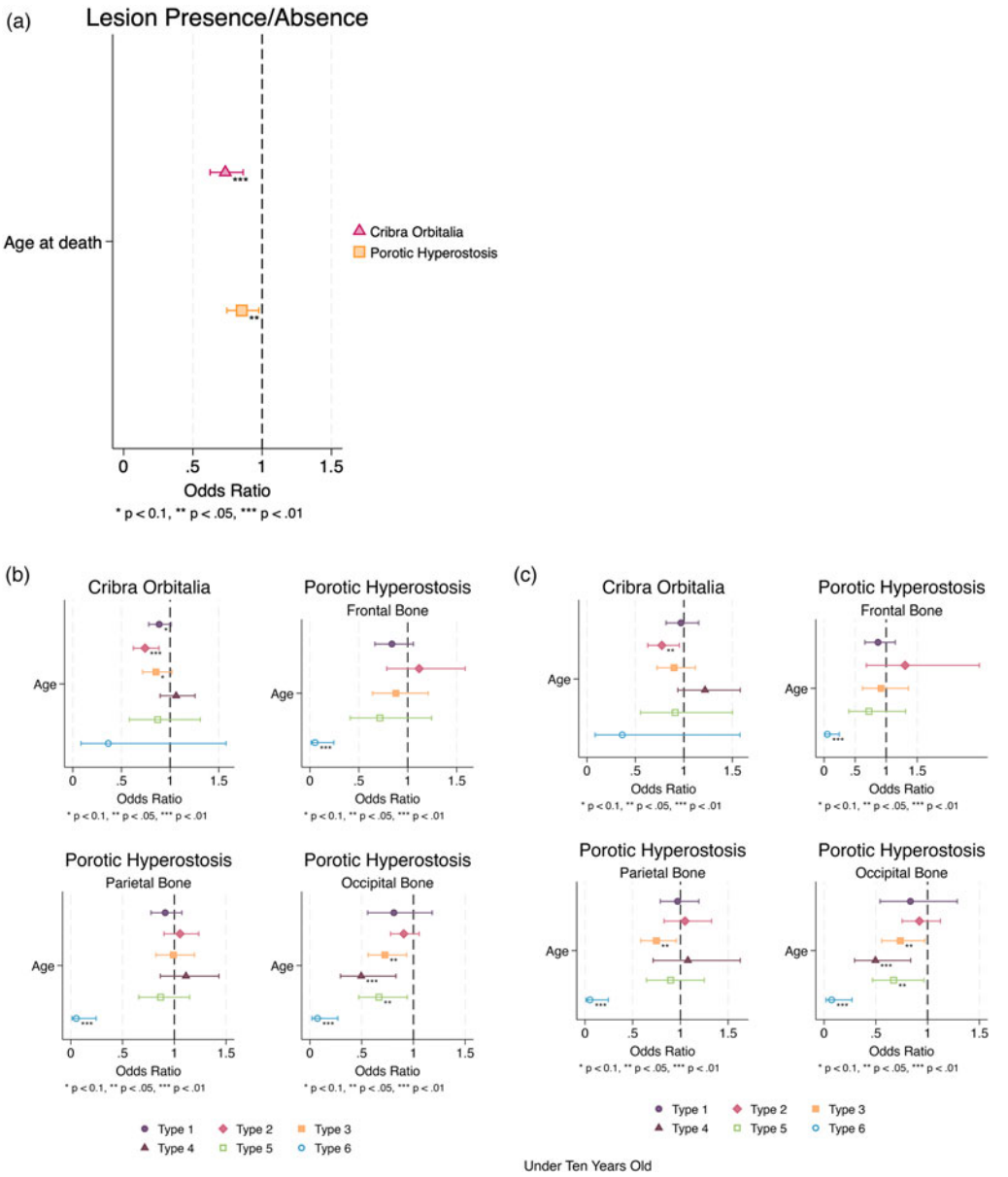


**Figures 4a–4c.** Each figure presents odds ratios depicting the relationship between PCL location, type, and age at death in individuals 6 months to 15 years; Figure 4a presents results for CO (triangle) and PH (square) presence, regardless of morphology, by age at death. Figures 4b and 4c present the same analyses, but Figure 4c provides results for individuals under ten years of age. These analyses focus on lesion types and age at death. Type 1 is represented with a filled circle (purple), Type 2 with a red diamond, Type 3 with an orange square, Type 4 with a purple triangle, Type 5 with an open green square, and Type 6 with an open blue circle. Significance values are provided for "$p$" at three levels: 0.01, 0.05 (threshold for statistical significance) and 0.1. $p \leq 0.1$ is provided for the reader as some of these values are $p < 0.07$ but $p > 0.05$. Lines represent 95% confidence intervals. Of note, no individuals with type 4 PH were observed for the frontal bone. (Color Online)

picture of Southwest dietary variation and risks posed by soil and water-borne parasites, contextualizing observed PCL rates and morphological variation.

Although this article focuses on the US Southwest, its findings are of relevance to those who study human health and stress in other temporal and geographic contexts. For example, other studies such as that of Cadwallader and colleagues (2012) indicated that the results of paleodietary reconstructions should be interpreted with care. The presence and meaning of PCLs should be interpreted similarly.

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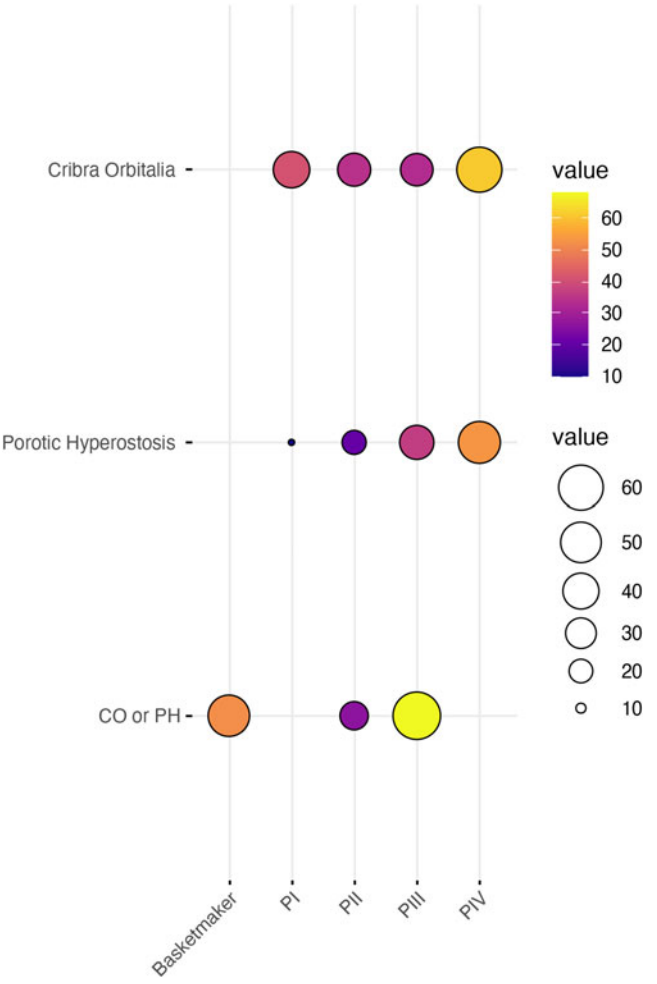


**Figure 5.** Balloon plot shows the percent frequency of CO and PH across time. The "balloon" increases in size with increasing frequency, and the color increases in temperature following the same pattern. References for data are shown in Table 3. Abbreviations: BMII (1500 BC–AD 50), BMIII (AD 500–750), PI (AD 750–900), PII (AD 900–1150), PIII (AD 1150–1325), and PIV (AD 1325–1550). (Color online)

PCLs are found around the globe in varied archaeological settings (Carlson et al. 1974; Jatautis et al. 2011; Keenleyside and Panayotova 2006; Obertová and Thurzo 2004; Schats 2023; Smith-Guzman 2015; Zaino and Zaino 1975). They have also been identified in contemporary populations (Anderson 2022; Beatrice and Soler 2016; Beatrice et al. 2021; O'Donnell, Hill, et al. 2020) and are emerging as markers of generalized stress in public health contexts (O'Donnell et al. 2024). If their presence is assumed to derive from a single cause, then we may overestimate the frequency of conditions historically implicated in PCL development—for example, iron-deficient diets—while failing to recognize the multifactorial nature of PCL development and the role of other potential causative factors: dietary, immunological, and social. PCLs may be interpreted as indicators of stress suffered by a developing individual, but unless their morphology is used to diagnose the cause, they should not be seen as hallmarks of specific conditions.

### *Reconstructed Southwest Diet*

Coprolite, paleoethnobotanical, and faunal studies demonstrate a varied and, in general, nutritionally adequate diet in the Southwest, with a variety of cultivated and wild foods supplementing maize (Durand and Durand 2008; Huckell and Toll 2004; Woosley 1980). Maize on its own is nutritionally incomplete, and the consumption of wild food sources provides critical micronutrients. For example, rabbits, an essential and ubiquitous dietary component (Brand 1994:39; Coltrain and Janetski 2013; Durand and Durand 2008:103; Holliday 1996), provide vitamin $B_{12}$ and other nutrients, including

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heme iron (Nistor et al. 2013; USDA 2021). Fish, which were also eaten, provide heme iron and also increase the nonheme absorption of iron from maize (Hutchinson et al. 2007; Layrisse et al. 1968).

Many wild plants are higher in iron, protein, and other micronutrients than maize. Pine nuts are a seasonally available source of protein, folate, and lysine. Yucca, juniper berries, opuntia, and wild mustard are also sources of vitamin C (Barriada-Bernal et al. 2018; Truesdale 1993). Cheno-ams were noted at high frequencies in our survey, rivaling those of maize (Figure 2a). Amaranth is high in protein and contains higher levels of calcium, iron (29% of the recommended daily intake in one cup), magnesium, and sodium than other cereal grains (Ajmera 2018; Stallknecht and Schulz-Schaeffer 1993). It is also a good source of carotenoids (Duya et al. 2018), which increase iron uptake and ferritin synthesis (García-Casal 2006). The fungus *Ustilago maydis* (*huitlacoche*, or corn smut), which has higher protein content than maize, may have also been consumed (Battillo 2018).

Infants and children are more likely to develop PCLs than adults, but their diets are more challenging to assess in Southwest archaeological contexts. Coprolite studies do not specify whether adults or children deposited them. Isotopic studies involving infants and children are limited in number and sample size and largely restricted to the Basketmaker II and III periods. These suggest that the timing of weaning was highly variable and that the diets of nursing infants were likely supplemented with weaning food such as maize gruel (Coltrain and Janetski 2013).

Ethnographic research provides further clues to Puebloan infant and child diets. Cahuilla (Southern California) babies were fed piñon nuts ground up and mixed with water (Moerman 2010:184). The Haudenosaunee (Iroquois) did something similar with butternut (Moerman 2010:131), and Hopi (Pueblo) children were given small bits of food, including corn, mutton, and fruit (Eggan 1943:363). These studies suggest that supplemental foods provided to infants were varied and likely extended beyond maize gruel. In some cases, Pueblo babies were nursed for up to three years (Gonzalez 1974). In Ancestral Pueblo archaeological contexts, prolonged weaning, diarrheal diseases, and resultant iron deficiency have been implicated in the elevated PCL rates observed in infants (Kunitz and Euler 1972). However, maternal buffering via nursing promotes the delivery of key nutrients and immune factors during episodes of nutritional stress and may limit the development of dietary deficiencies and diarrheal disease—if mothers' micronutrient stores are not substantially depleted (Thayer et al. 2020).

It is impossible to know whether the ethnographic literature accurately reflects infant and child diets. However, we might infer that children in the Southwest were fed an adequate diet that included food items consumed by adults. This varied diet and the impact of maternal buffering render it unlikely that diets deficient in iron or vitamins ($B_{12}$, C, D, and others) are causes of all or even many of the lesions recorded and reported in the Southwest.

Of course, there were times of dietary, environmental, or social stress, which may have led to increased biological stress and potentially to PCL formation. During the mid- to late 1200s and early 1300s, there was significant demographic upheaval in northern New Mexico and the Mesa Verde region that caused environmental and social stresses, including migrations (Crown et al. 1996; Kohler et al. 2010). Psychosocial stress may contribute to PCL development (Beatrice et al. 2021), but studies of its impacts on PCL formation need to be conducted in children to address this hypothesis fully. Social and environmental stress might lead to dietary deficiencies, because maize production is reduced in periods of low rainfall or during years with reduced growing seasons.

Although some evidence of scurvy in the Southwest is provided by this study and others (Crandall 2014; Ortner et al. 2001), definitive skeletal evidence for nutrient deficiency is rare in the Southwest across time (East 2008; Ortner et al. 2001; Stodder et al. 2010:96) and in our analysis.

### *Bone Changes Seen in Anemia*

Because anemias cause inconsistencies in hematopoiesis and hypoxia (WHO 2019a), they can result in alterations of bone. Hypoxia is associated with increased osteoclastic activity, suppressed osteoblastic activity, and bone loss (Hannah et al. 2021), and congenital anemias are associated with marrow hyperplasia. However, clinically identified bone changes in anemias and related diseases are rare (Aksoy et al. 1966; Lanzkowsky 1968; Sebes and Diggs 1979). Skeletal alterations, including PCLs,

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are not typically considered by clinicians when diagnosing anemia (Brickley and Morgan 2023:343). Furthermore, the severity of anemia is not a factor in lesion formation (Grauer 2019:517; Lanzkowsky 1968:25; Stuart-Macadam 1992:44). Not everyone who is anemic has skeletal involvement, and not everyone who has PCLs has anemia (Diggs et al. 1937; Lanzkowsky 1968; Wapler et al. 2004).

Wapler and coworkers (2004) found no histological indications of anemia in at least 56.5% ($n$ = 333) of CO cases. Congenital anemias are more likely to induce marked skeletal alterations than acquired anemias (Lewis 2012; Brickley and Morgan 2023) but still do not produce alterations in all those afflicted (see also Brahmbhatt et al. 2017). PCLs are rarely observed in iron-deficiency anemia, with an incidence of 0.68; postcranial alterations occur more frequently (Agarwal et al. 1970).

A clinical connection between AoI and PCLs is uncertain, which has led to some disagreement in the bioarchaeological literature. Stuart-Macadam (1991b, 1992) is a proponent of the idea that infection, via AoI, caused many PCLs observed in bioarchaeological settings; see also Kent (2000). Other researchers found that AoI restricts erythropoiesis, so it should lead to bone loss, not marrow hyperplasia (Oxenham and Cavill 2010).

In our study, expansion was noted in 18.8% of individuals with postdepositional breakage of the orbit roof and 16.1% of individuals with postdepositional breakage of the vault bones. In a contemporary New Mexico mortality sample, 20.8% (133/475) had expansive lesions with pitting/porosity (orbit), but only 1.38% (5/362) were diagnosed with anemia, and none had iron-deficiency anemia (O'Donnell et al. 2023: Table 3). If expansive lesions are taken as evidence of iron-deficiency anemia, which would be ill-advised, then a small number of individuals included here may have suffered from iron deficiency.

The rarity of PCLs in clinical patients with iron-deficiency anemia (Agarwal et al. 1970), coupled with an adequate diet, renders it highly unlikely that it is responsible for most cases of PCLs observed in the Southwest. This does not discount a relationship between PCLs and nutrient deficiency/acquired anemias in all cases, but anemias should not be diagnosed solely through the presence of PCLs.

### *If Not Diet (in All Cases), What Explains PCLs?*

Ancestral Puebloans and other Southwest groups likely experienced various health insults. Kent (1986:607) found that "maize, and diet in general, are at most only incidental causes of PH in the Southwest region." Instead, Kent (1986, 2000) argued that exposure to parasites, streptococcal infections, or other viral and bacterial agents could have caused PCLs, claims echoed by Stuart-Macadam (1992). Individuals sick for prolonged periods (a month or longer) have heightened odds of exhibiting PCLs (O'Donnell et al. 2023).

### *Respiratory Infection*

Before 1900, pneumonia and gastrointestinal disease were common causes of child death (Mulholland 2007). Pneumonia can be sustained for a prolonged period (Haines et al. 2013), allowing time for skeletal lesions to form (Lindaman 2001; O'Donnell, Hill, et al. 2020). Kunitz (1970) suggested that respiratory infections were important contributors to patterns of morbidity and mortality observed in Ancestral Pueblo populations. Some environmental conditions that existed in the past increase the risk of pneumonia and respiratory illness. These include indoor air pollution caused by wood and dung burning known to occur in the Southwest (Jorgensen 1975; Windes and Ford 1996), crowded living conditions, parental smoking (WHO 2019b), and exposure to dust and pollen (Lambert 2002).

There is growing support for an association between PCLs and respiratory illness, although mechanisms underlying this relationship remain unclear. O'Donnell, Hill, and coworkers (2020) associate PCLs with respiratory illness in a contemporary mortality sample but stress that not every PCL is evidence of respiratory infection. Other studies found similar results; Gomes and colleagues (2022) and Anderson (2022) found associations between symptomatic tuberculosis and CO in a contemporary Bolivian population. Respiratory infections, including tuberculosis, bronchitis, and pneumonia, likely occurred in the Southwest. Although rare, tuberculosis was reported in several Southwest locales (East 2008; Lambert 2002; Ortner 2003; Stodder and Martin 1992). The reported cases likely represent a minimum number of infections (Roberts and Buikstra 2019).

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When taken in conjunction with dietary data, some PCL morphology may support assertions that infection and associated inflammation (perhaps AoI) are responsible for PCL development (Kent 1986, 2000; Stuart-Macadam 1991b). Although AoI is likely not responsible for expansive lesions (Oxenham and Cavill 2010), it could cause porosity, because inflammatory processes are linked to bone loss (Epsley et al. 2020).

### Parasitism and Gastrointestinal Disease

There are no diagnostic criteria for identifying parasitism or gastrointestinal disorders in a skeleton. However, there is direct evidence for intestinal parasites in the Southwest from coprolite studies (Fry and Hall 1986; Reinhard 2008a, 2008b; Siqueira et al. 2010). Pinworm (*Enterobius vermicularis*) and human whipworm (*Trichuris trichiura*) eggs have been identified in coprolites recovered from Ancestral Pueblo sites in Chaco Canyon (Paseka et al. 2018). Parasitic infection was likely common among Ancestral Puebloans (Reinhard 2008a, 2008b) and may have been a consequence of cultural development related to agriculture (Paseka et al. 2018). Parasites can cause chronic inflammation and may result in AoI (Glinz et al. 2015). Helminths cause malabsorption of nutrients and diarrhea (Genta 1993). Other gastrointestinal diseases, such as gastroenteritis—acute or chronic inflammation (Merriman 2014)—likely caused issues for people in the past but are not associated with PCLs in other studies (e.g., Gomes et al. 2022; O'Donnell, Hill, et al. 2020).

Malnutrition and gastrointestinal infections may contribute to diarrheal diseases, which are a significant contributor to morbidity and mortality in children under five years of age today and likely shaped patterns of growth and mortality risk in Ancestral Puebloan populations (Schillaci et al. 2011). Gastroenteritis can quickly lead to dehydration and death; several routes to infection exist, including bacteria and parasites, but rotavirus is the most common agent (Siqueira et al. 2010). Because of the rapidity with which gastroenteritis kills, it is unlikely that it causes PCLs, given that prolonged illness seems to be a prerequisite (O'Donnell et al. 2023).

However, *H. pylori* infection often occurs in early childhood and was likely present in the Americas before Spanish arrival (Darling and Donoghue 2014). Hosts may experience chronic inflammation in the intestinal tract and reductions of micronutrient bioavailability over extended durations, which can result in iron loss and stunted growth (Muhsen and Cohen 2008). Although parasitism could possibly lead to PCL formation, partly because of its prolonged nature, this would need to be directly tested in living subjects.

### A Note on Development

Cole and Waldron (2019) suggested that "simple porosity" of the orbit may represent normal developmental variation. Here, the probability of having PCLs drops with increasing age (Figure 4). Perhaps, due partly to developmental factors, PCLs are more likely to form in the young (see also Brickley 2018). Research findings may also indicate the resilience or robusticity of individuals who either never developed lesions or survived long enough for them to fully remodel.

Type 2 CO is significantly associated with younger ages at death (Figure 5), an association not noted for the vault bones. This relationship underscores the importance of recording and considering CO and PH morphology and location *before* diagnosing the presence of any disease. If Type 2 CO is *not* pathological, it should not be included in *any* analyses of past health and certainly should not be considered diagnostic of "anemia."

Conversely, when Type 2 CO is found alongside other PCL types, it may indicate pathological origins. In our study, two individuals with probable scurvy had Type 2 CO alongside Types 3 and 4. It would be prudent in any study of PCLs to examine whether there is an association between CO characterized *only by* fine, scattered foramina and young age in the pediatric population and what that association might mean.

### Recommendations for Researchers

PCLs should be analyzed without assuming that they were caused by anemia or nutrient deficiency. Likewise, CO and PH should be considered separately, because they may have disassociated etiologies.

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When possible, PCLs should be evaluated within the context of (1) the entirety of an individual's skeleton; (2) specific PCL morphology; (3) age at PCL formation; that is, because they develop in early childhood, blood loss from menstruation or pregnancy resulting in iron deficiency anemia is unlikely; (4) the geographic and temporal location of the population; and (5) the actual, rather than the assumed, diet. If this evaluation is not possible, PCLs can be used as indicators of stress events of unknown origin.

Researchers need not eliminate use of the indicator—the presence or absence of PCLs—in analysis, but caution should be exercised when this approach is taken. As demonstrated here, this approach has utility. One definite benefit is the increased sample size, but a drawback could be the homogenization of potential causes; that is, all PCLs become signals of "anemia." If only PCL presence/absence is recorded, that data should not be used as equivocal evidence that PCLs signal nutrient deficiency, anemia, or any other condition. The best practice researchers can employ today is setting aside assumptions as to cause. PCLs should be treated as nonspecific indicators of stress until a complete differential diagnosis and analysis of the entire skeleton are undertaken (Grauer 2019:515) or the individual's medical history is known.

Literature that ascribes PCLs to anemia—or to any single cause, for that matter—is still useful for understanding the lived experience of past peoples. However, unless PCL morphology—diploic expansion in concert with pitting/porosity—is used to assess lesion etiology, no diagnosis can be made. PCLs have varied etiologies, and even when they manifest together in a single individual, they may not have the same causes.

## Conclusions

Although PCLs are often associated with nutritional anemia, including that caused by iron deficiency, this relationship is probably only tangential to the primary insult. Anemia is a symptom of an underlying disease or disorder with many potential etiological drivers (see Grauer 2019:Figure 14.20). The problematic implications of associating CO and PH with nutritional anemia, despite their diverse etiologies, have been noted by multiple researchers (Cole and Waldron 2019; Grauer 2019; Mays 2012; Walker et al. 2009; Wapler et al. 2004), but the association continues to be made. Narrowing our lens to just iron deficiency or another specific form of nutritional anemia is an even more reductive approach. Instead of assuming anemia is the source, researchers should set aside preconceived notions of cause.

The cause(s) of PCLs is certainly open for debate in the Southwest—and likely elsewhere—especially when dietary data are considered. Surely, a proportion of PCLs are caused by nutritional anemia. However, human diets vary, even when people lean heavily on a staple grain. Ancestral Puebloans supplemented a maize-intensive diet with hunted and gathered foods. Many of those foods are rich in micronutrients, and some increase iron uptake and ferritin synthesis. So, it is unlikely that nutrient deficiency (iron-deficiency anemia, vitamin $B_{12}$), especially due to a maize-reliant diet, is the cause of most PCLs seen in the Southwest. Findings might differ in studies of different geographic locales or groups with different lifeways. Still, it is advisable to consider archaeological evidence for diet and cultural conditions alongside PCL morphology and frequency when assessing potential causative factors.

**Supplemental Material.** For supplemental material accompanying this article, visit https://doi.org/10.1017/aaq.2024.61.

Supplemental Table 1. Definitions of terminology and different types of anemia.

Supplemental Table 2. Number of coprolites by food type and site; for all references see Table 3; frequencies presented in Figure 4 were estimated using these totals. This data was used in Supplemental Figure 1 and Figure 2 (in this article); for the most part pollen studies required >=200 grains.

Supplemental Table 3. Sites where individuals included in Figure 5 are from. Number of individuals scored for cribra orbitalia (CO) and porotic hyperostosis (PH) here are presented for studies who reported PCLs for children (less than 15 years of age). This table also provides a range for the number of coprolites per site and study, but see Supplemental Table 2 for more detailed sample sizes of coprolites by site and food item. These data are not exhaustive.

Supplemental Figure 1. Frequency of coprolites with pollen/macrofossils by site.

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