Title: Hidden dangers? Investigating the impact of volcanic eruptions and skeletal fluorosis in medieval Iceland

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Abstract:

Volcanic emissions are known to be a serious source of pollution to humans and animals. This study aimed to examine the possible health burden of fluoride (F) exposure from volcanic eruptions in the past. Osteological analyses were performed on 186 skeletons from seven sites across Iceland with the aim of identifying skeletal changes potentially associated with osteofluorosis. Additionally, ion selective electrode (ISE) was used to investigate possible correlations between skeletal lesions and bone fluoride concentrations in a subset of skeletons (n=50) from two of these sites, Skriðuklaustur in Fljótsdalur and Skeljastaðir in Þjórsárdalur. The results showed that pathological markers or skeletal changes increased according to age across all investigated time periods and geographical regions but likely not due to significant fluoride exposure. The fluoride concentration range was 223-4370 ppm (mean= 2324±1067 ppm) at Skriðuklaustur (n=36) while at Skeljastaðir (n=14) the range was 223-3030 ppm (mean= 1366±937 ppm). The pathological changes noted in this study are thus more likely to relate to the environment, population dynamics, culturally mediated behaviours and increasing urbanisation than serious fluoride contamination. Therefore, volcanic emissions appear to have only substantially affected those living within the closest vicinity of actual eruption events. It may be vital to not only consider the severity of the immediate effects of volcanic pollution but also to reinforce socioeconomic circumstances and disaster preparation in regions with environmental hazards.

Keywords: Palaeopathology, ISE, pollution, volcanic eruption, trace element analysis, fluoride

Introduction

The potential health effects of exposure to volcanic emissions have been widely recognised, however, few primary studies have directly examined the relation between them (Hansell and Oppenheimer 2004; Horwell and Baxter 2006). Past eruptions have triggered severe worldwide environmental changes, such as volcanic winters and summers, characterised by darkness, famine and pestilence. The low irradiance of the sun following 6th century eruptions – that may have occurred in Iceland – is believed to have caused both direct and indirect adverse effects on human health. For example, it has been hypothesized that these eruptions provoked the Justinian Plague (541-544 AD), as the cooling weather provided favourable conditions for disease vectors to spread across the Mediterranean (Loveluck et al. 2018; Helama et al. 2018). Public health has proved to be directly affected by changes in climate, the environment and availability of resources (Semenza and Menne 2009). In the past, natural disasters, such as volcanic eruptions, have caused local environmental changes which have resulted in profound cultural changes and even site abandonment (Black 1981; Grattan 2006).

With its volcanic activity, Iceland provides a unique opportunity to examine correlations between disease prevalence, eruption events and social and environmental change over time (Fig. 1). Recent research using ICP-MS demonstrated that numerous archaeological individuals from two medieval Icelandic sites, Skriðuklaustur and Skeljastaðir, were exposed to mercury. The results indicated that elevated mercury concentrations at the hospital-monastery Skriðuklaustur (AD 1493-1554) were likely associated with the use of mercury to treat treponemal disease, while much higher concentrations found at Skeljastaðir (AD pre-1104) indicated a volcanogenic origin (Walser III et

al., 2019). Likewise, 12 individuals from Skriðuklaustur showed evidence of lead exposure during childhood (Walser III et al., 2020). While these elevated lead concentrations were likely due to anthropogenic exposure, a volcanogenic origin of lead pollution cannot be entirely ruled out. Considering these findings, we sought to further investigate evidence of human exposure to other toxic volcanogenic emissions with a particular emphasis on fluoride. Skeletal fluorosis in humans is characterized by ossification of soft tissues, dental enamel defects, joint ankyloses, osteomalacia, osteosclerosis and bony outgrowths known as exostoses (Littleton 1999). Fluoride toxicity (acute and chronic) is even known to have caused mass fatalities to livestock throughout Icelandic history (Weinstein and Cook 2005). Although a small pilot study was previously conducted (see Gestsdóttir et al. 2006), the overall extent to which the historical human population was subject to fluoride toxicity has yet to be explored.

The aim of this research is thus to examine the possible health burden of fluoride (F) exposure from volcanic eruptions in the past, using osteological and chemical analyses of historic Icelandic human skeletal remains. In order to examine this, osteological data was analysed from 186 skeletons excavated from seven archaeological sites (Skeljastaðir, Haffjarðarey, Skriðuklaustur, Reykholt, Viðey, Bessastaðir, Reykjavík) across Iceland, ranging in date from the 11th to 19th centuries. A range of pathological skeletal changes were recorded with the aim of identifying those potentially associated with skeletal fluorosis. The prevalence of carious lesions was likewise investigated in order to examine the possible relationship between dietary factors and natural water fluoridation. An additional aim was to examine whether skeletal or dental lesions indicative of fluorosis correlated with bone fluoride concentrations. Therefore, a total of 50 bone samples were selected from two of the sites, Skriðuklaustur and Skeljastaðir, for fluoride concentration analysis using ion selective electrode (ISE). Associated soil samples were also measured to control for diagenetic factors. Overall, the study seeks to examine how fluoride exposure and volcanic eruptions impacted the human landscape and how those impacts affected the culture, health and lifestyle of historical Icelanders.

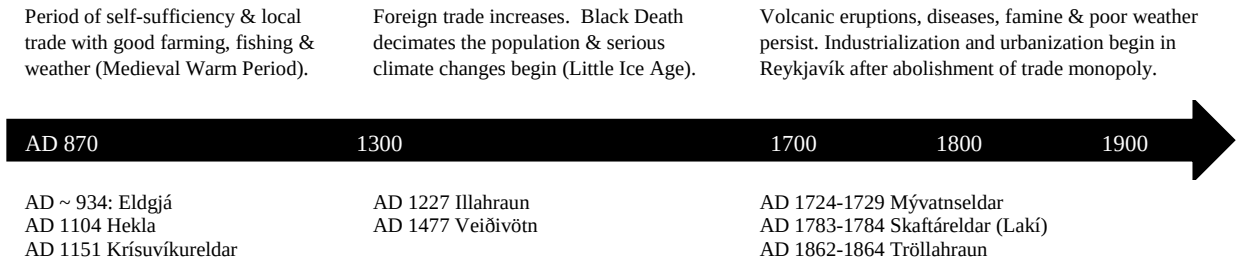

Fig. 1 Changes in the environment, subsistence economy and public health in Iceland over time. Adapted from Mehler (2011). The listed volcanic eruptions were historically recorded as "fires" and in some cases one event represents numerous consecutive eruptions (e.g. 10-11 eruptions occurred during the AD 1783-1784 Skaftáreldar (Lakí) fires) (Thordarsen and Larsen 2007)

Environment and fluoride exposure in Iceland

Eruptions and passive degassing from volcanic systems are the most persistent sources of heavy metals and fluoride worldwide, accounting for up to 90% of all emitted fluorine (Hansell et al. 2006; D'Alessandro 2006; Kagel et al. 2007; Tchounwou et al. 2014). While eruptions are often

short-lived, the ash deposit following an eruption can remain in the environment for several years or even decades and can be remobilised by wind or human activities (Horwell and Baxter 2006).

Volcanic ash is a major component in Icelandic sediments due to frequent eruptions, which have affected the living population since the settlement of the island in the 9th century (Fig. 1). Additionally, folklore, historical records and archaeological data indicate that the effects of Icelandic eruptions were felt across Europe and even caused cold, dark winters that lasted for years (Grattan 2006; Grattan et al. 2003). Eruptions can not only pollute soil but can also contaminate water sources with fluoride, thus endangering wildlife, livestock and the people drinking it (Delmelle et al. 2002; Cronin et al. 2003; D'Alessandro 2006). Furthermore, such pollution has also caused famines through chlorotic or necrotic destruction of flora or crops (Delmelle et al. 2002).

From c. AD 871-1200, at least 205 volcanic eruptions have been documented in Iceland, amounting to approximately ~10 to 12 per century. In the post-Medieval era, improved record keeping documented ~27 eruptions per century (Thordarson and Larsen 2007). Most of these volcanic eruptions lead to complications in farming and feeding livestock for years afterwards, sometimes resulting in long-term site abandonment (Þórðarson 1943: Rafnsson 1990; Dugmore et al. 2007; Dugmore and Vésteinsson 2012). Still, the connection between eruptions and livestock fatality and morbidity was not formally recognized until after the 1693 eruption of Mt. Hekla when a farmer and a clergyman described the deformation of animal teeth, referred to as "ash-teeth" (D'Alessandro 2006) (Fig. 3). Examples of fluorotic sheep bones related to the 1845 eruption of Hekla have been found (Roholm 1939) but are not available for study today (Fig. 3). In present day Iceland, sheep, which roam freely, are the most prone to fluoride toxicity following eruptions due to surface water consumption, grazing on contaminated or ash-laden vegetation (Kristinsson et al. 1991) and due to residential proximity to aluminum smelting factories (Krater and Rose 2009).

For example, the Hekla eruption of 1947-1948 caused fluoride concentrations in nearby stream waters to increase from a maximum of 0.57 ppm to 9.5 ppm in some cases, resulting in the poisoning of farm animals (Stefánsson and Sigurjónsson 1957; Sigurðsson and Pálsson 1957; Stewart et al. 2006; Gunnarsdóttir et al. 2016). Similarly, fluoride in tephra from the 1970 eruption of Mt. Hekla proved to be extremely soluble, thus increasing its toxicity; grazing pastures, foliage and topsoil were permeated with lethal doses of it and up to 8000 animals died (Óskarsson 1980; Cronin et al. 2003; Gudmundsson et al. 2008) (Fig. 2- 3). Death from acute fluorosis occurred at bone concentrations of 30-60 ppm F within the first week. Chronic fluorosis was, for example, diagnosed at bone concentrations of <1329 ppm F (normally ~830 ppm F) in adult sheep but at far lower concentrations in lambs (~698 ppm; normally ~116 ppm (see Georgsson et al. 1981; Stefánsdóttir 2016). While most animal fatalities occurred rapidly due to acute toxicity, some even developed dental enamel changes (e.g. pitting, hypomineralisation or mottling), inappetence (loss of appetite) and ataxia (lack or loss of ability to voluntary control muscles and balance) (Thorarinsson and Sigvaldason 1972; Cronin et al. 2003; Grattan 2006; D'Alessandro 2006).

Fig. 2 Left, a farmer and his son from Húnavatnssýsla stand beside a grave of their own sheep that died of fluoride poisoning following the 1970 eruption of Mt. Hekla. © Magnús Finnsson. Right, sheep that died of fluoride poisoning following the Hekla eruption of 1947-1948. Reprinted from *Hekla on Fire* by Sigurður Þórarinsson (1956)

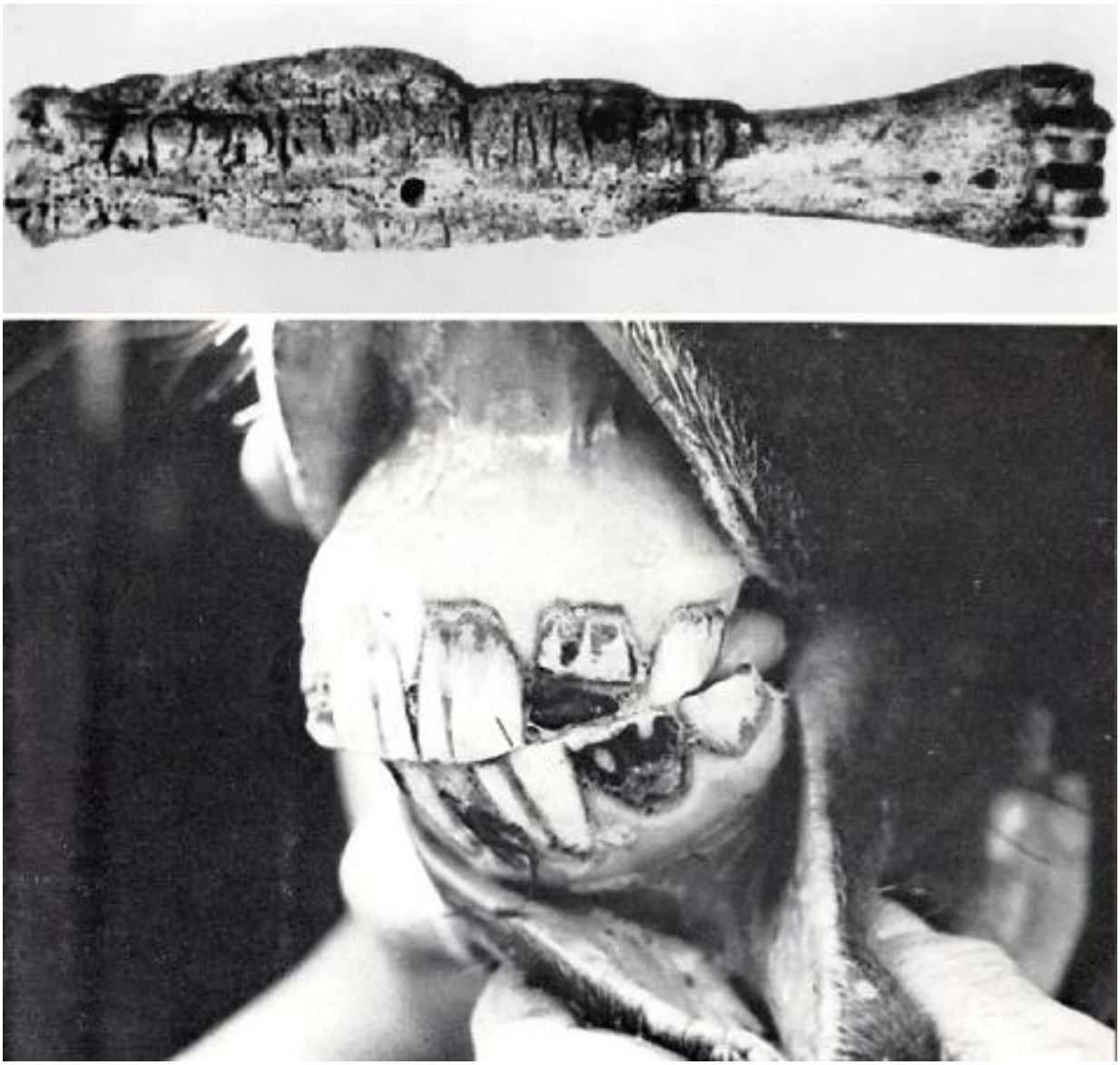

Fig. 3 Top, sheep metatarsal with a bone fluoride concentration of 20,600 ppm and bone changes caused by skeletal fluorosis during the Mt. Hekla eruption of 1845. Reprinted from Roholm (1939). Bottom, dental fluorosis ("ash-teeth") seen in horse dentition following the Hekla eruption of 1970. © Keldur, the Institute for Experimental Pathology at the University of Iceland

Fluoride poisoning

Living conditions, local environments, nutritional status, ethnicity and socio-economic factors make some populations far more susceptible to fluorosis than others (Littleton 1999, Petrone et al. 2013, Nelson et al. 2016). The condition normally occurs due to the combination of drinking water fluoridation and biological (e.g. nutrition, stress, genetics, sex, age, renal function), cultural (e.g. food preparation/additives, calcium intake, water storage, smoking) and environmental (e.g. temperature, climate, water evaporation rate, volcanic emissions) factors (Mabelya et al. 1997; Yoder et al. 1998; Littleton 1999; National Research Council 2006; Barbier et al. 2010; Everett

2011; Nelson et al. 2016). Fluoride is distributed throughout the body via the bloodstream. It accumulates in bone most readily during growth and is permanently retained in bone and teeth. Its concentration thereby naturally increases with increasing age as it alters the homeostasis of bone metabolism (Arnala et al. 1985; Petrone et al. 2013). The skeleton contains about 99% of an individual's total fluoride burden as a result of fluoride (F-) substituting for hydroxide (OH-) in the hydoxylapatite of bone (Whyte et al. 2008). During dental development, excess fluoride ingestion results in porotic, hypomineralised enamel (i.e. dental fluorosis) by disrupting normal crystal growth, which irreversibly alters its mineralising matrix (Den Besten 1999; Brown et al. 2005).

While in extreme cases severe acute exposure may rapidly result in cardiac arrest or coma (Littleton, 1999; EFSA 2010; Everett, 2011), chronic fluorosis is cumulative and gradual and causes numerous skeletal, dental and other pathological conditions and decreases overall life expectancy (Littleton 1999; Baxter 2009; Nelson et al. 2016). According to a 2010 review, no modern cases of lethal fluoride toxicity have been reported (National Research Council, 2010) and it is therefore not possible to accurately provide a fluoride concentration associated with acute mortality. However, though unclear what the data is based upon, previous studies have reported that death may follow severe symptoms that occur with exposure to a dose of 250-450 ppm (Luther et al., 1995; Shomar et al, 2004; Brickley and Ives, 2008). The differing chemical forms of fluoride and mechanisms of exposure, aside from numerous other factors, further complicate the identification of a fluoride concentration likely to cause acute toxicity. Non-lethal acute fluoride poisoning has been reported, with one study showing that 2/3rds of 34 children affected via the consumption of contaminated water completely recovered within 24 hours. In this case lethal exposure did not occur because the salty and bitter taste of the water due to a high fluoride content prevented significant consumption (Hoffman et al., 1980). Rare cases of fatality following workplace exposure to hydrofluoric acid have been documented (Takase et al., 2004).

Although fluoride can provide some protection from dental disease, levels below 0.5 ppm have little or no cariostatic benefits, whilst still altering the stability and appearance of developing teeth (Browne et al. 2005; Susheela 2007; Santa-Rosa et al. 2014; Rajeswari et al. 2016). Ayoob and Gupta (2006) report that adverse effects begin following prolonged ingestion of fluoride at levels over 1 ppm, with serious effects beginning over 4 ppm. Fluorosis has been observed in individuals ingesting as little as 0.5-0.7 ppm, however, these concentrations were determined in regions with high daytime temperatures, which increases the concentration of fluoride in groundwater through evaporation. Due to the natural affinity that fluoride has for water, fluoride concentrations in the environment tend to be highest in areas with substantial groundwater saturation or water flow (King et al., 2011). In historical, as well as in modern Iceland, water was primarily sourced from rivers and streams with a glacial origin, however, some areas of the country relied upon wells of groundwater. The small disparity between safe and unsafe fluoride concentrations and the numerous predisposing factors influencing toxicity makes it impossible to accurately define a toxic threshold (Barbier et al. 2010; Petrone et al. 2013; Nelson et al. 2016).

Identification of fluorosis in the palaeopathological record is uncommon, but it has been recorded in individuals from the Bronze Age onwards from various places, including the Arabian Gulf, Pakistan, Bahrain, United States and Italy (e.g. Walters 1954; Lukacs et al. 1984, 1985; Frohlich et al. 1989; Littleton 1993, 1999; Mastrolorenzo et al. 2001; Yoshimura et al. 2006; Petrone et al.

2013; Nelson et al. 2016). It is important to note that most palaeopathological studies investigating skeletal fluorosis have been conducted on skeletal samples from arid regions with high daytime temperatures. As previously mentioned, these factors increase the concentration of fluoride in groundwater sources and therefore the risk of fluorosis. Bioarchaeological analysis of skeletal remains from Bahrain, dated 300 BC to AD 600, identified chronic fluorosis but only amongst older individuals (Littleton 1999). At Herculaneum (destroyed by Vesuvius in 79AD), diffuse entheseopathies, osteoscleroses and critically high bone fluoride concentrations (>10000 ppm) were noted in the skeletal assemblage indicating crippling, endemic fluorosis (Mastrolorenzo et al. 2001; Hansell et al. 2006; Petrone et al. 2013). The fluoride concentrations remain toxically high (ca. 4 ppm) in this region of Italy, where people are still affected by skeletal fluorosis, and the groundwater is permanently saturated with a fluoride rich ash deposit (Gombos et al. 1994; Petrone et al. 2013). Despite the very different cultural, geological, dietary, climatic and temperature differences between Italy and Iceland, the Herculaneum study provides useful comparative data for this analysis.

As there are no pathognomonic skeletal changes associated with fluorosis, numerous differential diagnoses must be considered (Crubézy 1999, Littleton 1999, Whyte et al. 2008, Nelson et al. 2016) (Table 1). Individuals with fluorosis will not always exhibit both dental (childhood exposure) and skeletal changes (exposure throughout life), thereby making it impossible to establish a predictive relationship between these two forms of pathological expression (Littleton 1999). Fluorosis may be asymptomatic and go undiagnosed due to the presence of other conditions. For example, chronic toxicity with fluoride results in brittle bones, highly prone to microfractures and osteomalacia (Whyte et al. 2008). Without chemical tests, often only osteomalacia is recognized (Kiely et al. 1999). Furthermore, regarding dental fluorosis, it is often difficult to differentiate enamel hypomineralisation, or mottling, (e.g. white opacities, brown staining of enamel surface) and enamel pitting caused by fluorosis from non-fluorotic causes (Pendrys 1999; Hillson 2008). Fluorosis may nonetheless be indicated when multiple skeletal changes consistent with the condition are observed (Nelson et al. 2016) (Table 1).

Skeletal and dental changes associated with skeletal fluorosis		
Bone formation or changes	*Vertebral changes*	*Dental changes*
dense periosteal deposition	widened vertebral appearance	discoloration
extensive new bone production	disc space narrowing	enamel pitting
joint disease or osteoarthritis	osteophytic vertebral fusion	mottling
Osteosclerosis	ossification of spinal ligaments	hypoplasia
periosteal hyperostosis	thoracic kyphosis	brown staining
hypertrophic bony exostoses	degenerative joint disease	white opacities
Ossification or calcification	*Microstructure*	*Other*
foramen magnum ligaments	thickened cranial diploe	increased fracture rate
tendons	osteopenia	osteomalacia
ligaments	osteoporosis	diaphyseal widening
interosseous membranes	osteophytosis	genu varum
costo-vertebral & -sternal joints	coarse trabecular pattern	genu valgum
intercostal calcification	intermittent growth lines	flexion deformations
Differential diagnoses and other common causes of increased bone mass		
Differential diagnoses	*Common causes of increased bone mass*	

DISH	Craniodyaphyseal dysplasia	Lymphoma
Ankylosing spondylitis	Craniometaphyseal dysplasia	Hypervitaminosis A
Hematogenous osteomyelitis	Endosteal hyperostosis	Hypervitaminosis D
Hyper- or hypoparathyroidism	Melorheostosis	Renal osteodystrophy
Paget's disease	Myelofibrosis	Fibrogenesis imperfecta ossium
Myositis ossificans	Sarcoidosis	Skeletal metastases
Osteopetrosis	Heavy metal toxicity	Engelmann disease

Table 1 Skeletal and dental changes associated with skeletal fluorosis. Compiled from the criteria described by Dean (1936), Shupe et al. (1963), Littleton (1999), Pendrys et al. (1999), Savas et al. (2001), Ayoob and Gupta (2006), Yoshimura et al. (2006), Whyte et al. (2008), EFSA (2010), Petrone et al. (2013), Faccia et al. (2015), Nelson et al. (2016), Den Besten (1999), Brown et al. (2005), Hillson (2008), Alvarez et al. (2009), Blinkhorn and Mekertichian (2013). Differential diagnoses as described by Littleton (1999), Yoshimura et al. (2006), Whyte et al. (2008), Faccia et al. (2015) and Nelson et al. (2016). Common causes of increased bone mass as described by Whyte et al. (2008)

Materials: Archaeological Context and Sites

As previously described, the skeletal remains recorded here derive from seven archaeological sites across Iceland representing different time periods. Samples for the analysis of fluoride concentrations were collected from skeletons from two of these sites.

10^{th}-11^{th} century: Skeljastaðir (ÞSK)

Skeljastaðir (ÞSK) was an inland farm and cemetery located in Þjórsárdalur valley at the base of mount Skeljafell in southern Iceland (Fig. 4). It was occupied by farmers and was in use from around the period of Christianisation (ca. 1000 AD) until the violent eruption of the nearby volcano Mt. Hekla in AD 1104 (Þórðarson 1943, Steffensen 1943, Dugmore et al. 2007). Stable isotope analysis indicates that the site was occupied by individuals born in Iceland (Price and Gestsdóttir 2006). Fifty-six (56) individuals that were excavated from the cemetery are currently available for this study.

13^{th} to 16^{th} century: Haffjarðarey (HFE) and Skriðuklaustur (SKR)

Haffjarðarey (HFE) is an island located just off the southern coast of the Snæfellsnes Peninsula (Fig. 4). There was a farm and church located there, but the area was abandoned due to coastal erosion and religious Reformation in 1563 (Steffensen 1946; Hoffman 2018). From the Haffjarðarey cemetery (AD ca. 1200-1563), a total of 54 skeletons are available from an excavation conducted in 1945. The surrounding area has three volcanic systems, including Snæfellsjökull, Lýsuskarð and Ljósufjöll, the last being very nearby, although none of these erupted while the farm was inhabited (Harðarson 1993). The people residing at Haffjarðarey were farmers and fishermen (Steffensen 1946; Hoffman 2018). Isotope analyses conducted on 11 individuals showed that they were likely local to Iceland (Price and Gestsdóttir 2006).

Skriðuklaustur (SKR) was an inland Augustinian monastery and hospital (AD 1493-1554) located in the Vatnajökull region of eastern Iceland (Fig. 4). It was established 16 years after a catastrophic eruption in the Bárðarbunga-Veiðivötn volcanic system (AD 1477) (Thordarsson and Larsen 2007; Kristjánsdóttir 2012). The eruption caused one of the largest tephra falls of historical times

resulting in the abandonment of farms in the Hrafnkelsdalur valley and devastation to the landscape, crops and human and livestock populations (Larsen 1988; Thordarson and Larsen 2007, Global Volcanism Program 2013, Rafnsson 1990). In 2002-2012, a total of 269 skeletons were excavated from the site and around half of them show notable pathological conditions (Kristjánsdóttir 2012). The population residing at Skriðuklaustur was mostly composed of the brethren, laymen and medical patients, while the cemeteries at Skeljastaðir and Haffjarðarey represent the general Icelandic population of labourers (Gestsdóttir 2004; Kristjánsdóttir 2012; Hoffman 2018). Isotope analyses (n=32) indicate that the assemblage represents individuals born in Iceland seeking treatment or hospice within its monastic district (i.e. the south-eastern quarter of Iceland) (Walser III et al. 2020).

17th to 19th century: Reykholt (RKH), Bessastaðir (BES), Reykjavík (RVK) and Viðey (VEY)
These four sites are located close to each other in south-western Iceland (Fig. 4). Reykholt (RKH) was a high-status inland farm during the medieval period. There was a church there at least since AD 1200. Recently, 18 burials were excavated from its cemetery dating from the mid-16th century to the late 19th century and including several high-status, historically identified individuals (e.g. Reverend Þorleifur Bjarnason) (Sveinbjarnardóttir, 2016).

Bessastaðir (BES), since the 13th century, was the dwelling place of the Danish King's highest-ranking officers based in Iceland. Today it is the current presidential residence. The church and cemetery there have been in use from the time the area was first settled around AD 1000. The 18 high-status individuals excavated from this site date from the 18th-19th centuries (Gestsdóttir 2004; Ólafsson 2013).

Reykjavík (RVK) has been the capital city of Iceland since AD 1786. One of its cemeteries has been excavated several times since the 1940s due to various construction projects. Numerous disarticulated remains and 17 articulated individuals of unknown social status, dating to the 18th-19th centuries, are available for study today (Gestsdóttir 2009, 2012).

Viðey (VEY) is an island located <1 km off the coast of Reykjavík. An Augustinian monastery operated there from AD 1226-1539 followed by a leprosy hospital and farm were run there afterwards. Around the mid-18th century, Árni Magnússon, the representative of the Danish King in Iceland, resided there during. A church and cemetery have been on the island since before the monastic period and up until the present day. In 1987-1988, 36 high-status individuals dated to the 18th-19th centuries were excavated (Hallgrímsdóttir 1989, 1991, 1993; Kristjánsdóttir 1995a, 1995b, 1996; Gestsdóttir 2012; Riddell et al. 2018).

The only volcano near Reykholt, Prestahnúkur (Langjökull system) has not erupted since 3550 BCE (Global Volcanism Program, 2013). Still, there are several volcanic systems (Reykjanes, Krýsuvík, Brennisteinsfjöll, Hengill, Eldvörp-Svartstengi and Fagradalsfjall) on the Reykjanes peninsula, where Bessastaðir, Viðey and Reykjavík are located, but none of them erupted during the lifetimes of the skeletal populations dating between the 17th-19th centuries (Fig. 4) (Global Volcanism Program 2013). However, the catastrophic eruption of Laki in AD 1783-1784 resulted in a volcanic winter causing famine, climactic change and mass mortality to the human population (20-25%) and livestock (75%) (Thordarson et al. 1996; Thordarson and Larsen 2007; Guðmundsson et al. 2008; Jónsson 1994; Halldórsson 2013).

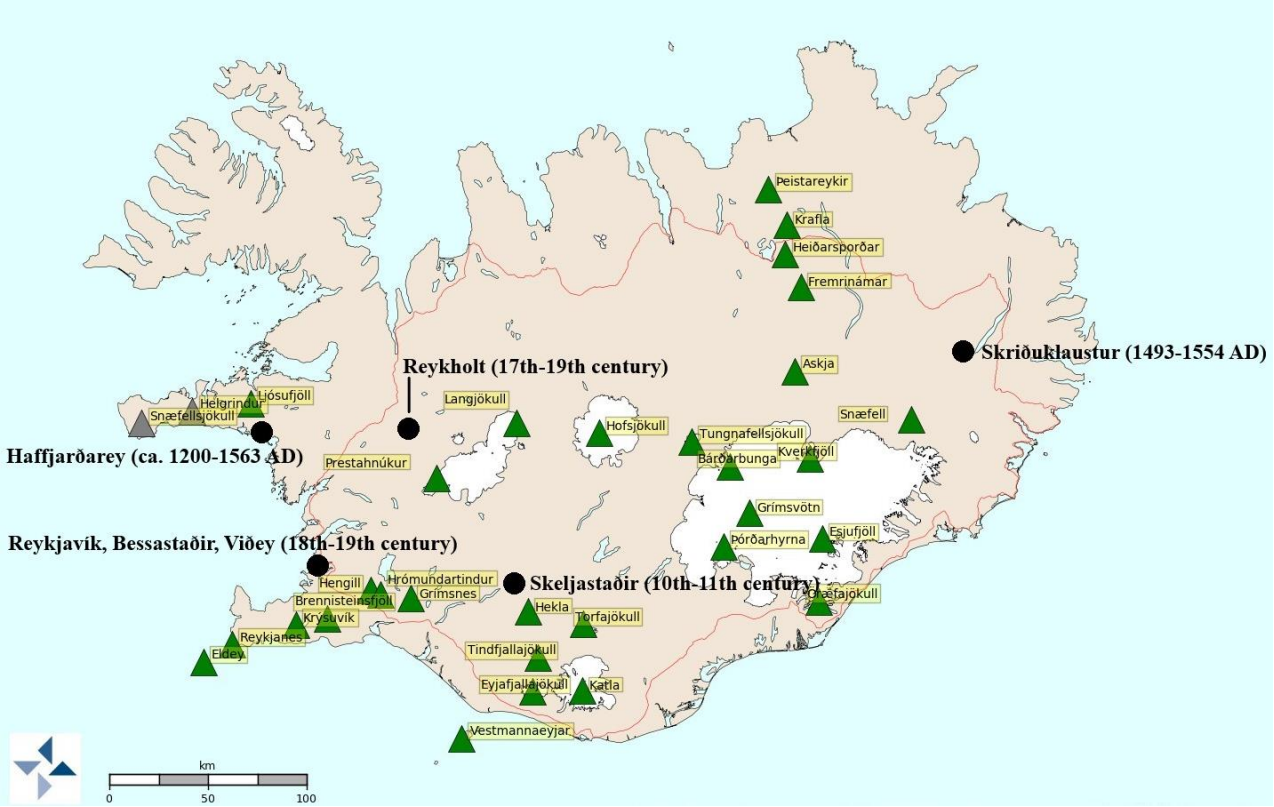

Fig. 4 Map depicting the volcanoes of Iceland and the archaeological sites analysed in this research. The dates correspond to the skeletal assemblages rather than the entirety of the sites use. The map is presented in a modified form from the Volcanic Hazards map issued by the Icelandic Meteorological Office (Veðurstofa Íslands), http://www.vedur.is/skjalftar-og-eldgos/eldgos

Methods

Osteological analysis

Only adult individuals with more than 50% completeness and good preservation (grade 0-1; (see Brickley and McKinley 2004) were analysed from the seven sites used in the present study. Due to the cumulative and progressive nature of skeletal fluorosis, a large range of skeletal changes must be considered in combination with measured fluoride concentrations in bone (Littleton 1999; Petrone et al. 2013; Nelson et al. 2016). Standard anthropological methods as outlined by Buikstra and Ubelaker (1994) and Brickley and McKinley (2004) were used during osteological analysis to estimate age, sex and pathological conditions (see Supplementary materials for further details). Age was limited to two categories, including younger adult (17-36) and older adult (36+) because dental attrition methods tend to overestimate age in Icelandic skeletal assemblages due to high-rate of dental wear (see Lanigan and Bartlett 2013; Richter and Eliasson 2017). Radiography was used to differentiate fluorosis from conditions causing similar bone changes (e.g. Paget's disease). Light microscopy was used to evaluate enamel defects (e.g. pitting, hypomineralisation, chipping) and enhance the observation of skeletal changes when necessary. Indicators of dental and skeletal

fluorosis were recorded according clinical and palaeopathological descriptions (Table 2; e.g. Dean 1936; Hillson 2008; Littleton and Frohlich 1993; Littleton 1999; Petrone et al. 2013, 2016; Nelson et al. 2016). Each bone was assessed for changes potentially connected with skeletal fluorosis and scored according to presence or absence, ossification of soft tissues (e.g. glands, ligamentum flavum, atlanto-occipital membrane) (Steinbock 1989; Binder et al. 2016; Geber and Hammer 2018), calcification of soft tissue attachment sites (Mariotti et al. 2007; Petrone et al. 2013), ankyloses (Rogers et al. 1985; Petrone et al. 2013; Ventades et al. 2018), vitamin D deficiency (Brickley et al. 2005, 2010; Ives and Brickley 2014) and enamel defects (Dean 1936; Brown et al. 2005; Alvarez et al. 2009; Blinkhorn and Mekertichian 2013; Hillson 2008)[1].

Skeletal change	*Description*
Osteomalacia and residual rickets	Morphological deformities, fractures, others
Ossification of ligamentum flavum	Cranial or caudal ossifications on vertebral arch attachments
Atlanto-occipital membrane	Increased rugosity or mineralisation
Entheseal & interosseous calcification	Increased rugosity or mineralisation
Ankyloses	Joints or bones otherwise fused to one another
Fractures	Healed or unhealed perimortem or antemortem fractures
Enamel defects	Hypomineralisation, mottling, staining; pitting; chipping
Calculus	Presence of mineralised plaque formation on dentition
Caries	Carious lesions on any aspect of tooth roots or crowns
Other ossifications	Ossification or calcification of glands (e.g. thyroid), cartilage or organs

Table 2 Osteological indicators of fluorosis analysed in this research[1]

Bone fluoride concentration analysis

In total, 50 well-preserved bone samples were selected for fluoride analysis: 36 from Skriðuklaustur (n=269 individuals) and 14 from Skeljastaðir (n=56 individuals). They were first cleaned with a synthetic brush and distilled water. The trabecular bone was mechanically removed, and the cortical bone surfaces were abraded with a dental bur to remove surface contamination. Trabecular bone was not included because it is far more susceptible to post-mortem diagenesis than cortical bone (see Rasmussen et al. 2015). All samples were selected from ribs, except for three long bone fragments due to the unavailability of ribs. Sample selection was informed by preservation, age, sex and pathological markers. Soil samples were collected from the Skriðuklaustur (n=2) and Skeljastaðir (n=2) cemeteries. Bone samples could not be selected from the other sites included in this study due to timescale and ethical concerns over the destructive nature of the analysis.

Cumulative bone fluoride concentrations were determined in 50 archaeological human bone samples and four soil samples using ion-selective electrode (ISE). Prior to analysis, NaOh (9 grams) was melted at 500ºC in a nickel-crucible and then cooled down to room temperature. Portions (1 gram) of ground sample material, blanks (same procedure without sample) and control samples (contains 10 mg CaF2 instead of sample) were digested at 500ºC for 60 minutes. After cooling, they were dissolved in 100 mL of water. Aliquots of the samples were neutralized and

[1] For detailed methodological descriptions refer to supplementary information.

TISAB-solution (NaCl, Titriplex and acetic acid in water, pH 5.5) was added. The samples, blanks and controls were then measured with ISE for fluoride. The ISE device is calibrated daily.

Results

Osteological Analysis

In all cases, the recorded pathological markers or skeletal changes appear to increase with age. This is to be expected given that many of the pathologies included in this analysis are also known to be correlated with increasing age. Low rates of ankyloses of the vertebrae (6%) or other joints (8%) were observed, showing no significant differences across age, sex or time periods. Forty-two percent of individuals had evidence of at least one ante-mortem fracture and the men and individuals age 36+ were most frequently affected in all periods (Table 3).

Ossification of the ligamentum flavum was observed in 26% of the overall sample with the highest prevalence observed amongst females from the later period (RKH, BES, RVK and VEY). However, ligamentum flavum ossification was approximately evenly distributed across age and sex when all time periods were pooled. Osseous changes at the atlanto-occipital membrane attachment site, on the posterior margin of the foramen magnum, were included in this research because it corresponds anatomically with the ligamentum flavum (Cramer and Darby 2017; Gonzales and Iwanaga 2018). Ossification of the antlanto-occipital membrane attachment site was observed in 37% of the overall sample and did not show notable differences according to age or sex, except within the 11th-12th century assemblage (ÞSK) where it was less frequent in younger adults (24%) than older adults (44%). Other soft tissue ossifications were more frequent with increasing age, as expected, except for in the 17th-19th century assemblages, which showed similar frequencies between younger (15%) and older adults (11%).

Vitamin D deficiency was observed in 10% of the sample, including osteomalacia in six individuals (3%) and residual rickets in 13 (7%) individuals. About 32% of vitamin D deficiency cases were observed in the individuals from Skeljastaðir and 42% in individuals from Skriðuklaustur. From the 17-19th century assemblages there were 5 individuals with vitamin D deficiency representing 26% of the cases observed in this study.

Period	*Site/Age/Sex*	*N*	*Atlanto-occipital membrane ossification*	*Miscellaneous ossification*	*Ossification of ligamentum flavum*	*Ante-mortem fractures*	*Spinal ankylosis*	*Non-spinal ankylosis*	*Vitamin D deficiency*
17th-19th	RKH, BES, RVK, VEY	54	17/48 (35)	7/53 (13)	19/44 (43)	27/54 (50)	4/46 (9)	3/54 (6)	5/54 (9)
13th-16th	HFE, SKR	82	30/67 (45)	18/80 (23)	14/63 (22)	32/82 (39)	4/58 (7)	8/75 (11)	8/79 (10)
11th-12th	ÞSK	50	12/43 (28)	11/50 (22)	7/43 (16)	19/50 (38)	1/42 (2)	3/50 (6)	6/50 (12)
17th-19th	Females	23	6/20 (30)	4/19 (21)	9/20 (45)	9/23 (39)	3/20 (15)	2/23 (9)	4/23 (17)
17th-19th	Males	31	11/29 (38)	3/31 (10)	10/26 (38)	18/31 (58)	1/26 (4)	1/31 (3)	1/31 (3)
13th-16th	Females	51	16/42 (38)	11/49 (22)	8/38 (21)	19/51 (37)	3/35 (9)	5/49 (10)	6/49 (12)
13th-16th	Males	31	14/25 (56)	7/30 (23)	6/18 (33)	13/31 (42)	1/22 (4)	3/31 (10)	2/29 (7)
11th-12th	Females	26	6/22 (27)	4/26 (15)	2/20 (10)	8/26 (31)	0/19 (0)	0/26 (0)	3/26 (12)
11th-12th	Males	24	6/21 (29)	7/24 (29)	5/23 (22)	11/24 (46)	1/23 (4)	3/24(13)	3/24 (13)
17th-19th	17-36	27	9/26 (35)	4/27 (15)	9/20 (45)	10/27 (37)	2/20 (10)	2/27 (11)	3/27 (11)
17th-19th	36+	27	9/23 (39)	3/27 (11)	10/26 (38)	17/27 (63)	2/26 (8)	1/27 (4)	2/27 (7)
13th-16th	17-36	51	20/42 (48)	8/49 (16)	5/35 (14)	16/51 (31)	1/33 (3)	6/50 (12)	4/49 (8)
13th-16th	36+	31	10/25 (40)	10/30 (33)	9/27 (33)	16/31 (52)	3/24 (13)	2/30 (7)	4/29 (14)
11th-12th	17-36	20	4/17 (24)	2/20 (10)	3/16 (19)	6/20 (30)	0/16 (0)	1/20 (5)	2/20 (10)

11th-12th	36+	30	8/18 (44)	9/30 (30)	4/27 (15)	13/30 (43)	1/26 (4)	2/30 (7)	4/30 (13)
All	Females	99	28/84 (33)	19/98 (19)	19/78 (24)	26/99 (26)	6/74 (8)	7/98 (7)	13/98 (13)
All	Males	87	31/75 (41)	17/86 (20)	21/74 (28)	42/87 (48)	3/72 (4)	7/87 (8)	6/85 (7)
All	17-36	99	33/86 (38)	14/97 (14)	17/72 (24)	32/98 (33)	3/70 (4)	9/98 (9)	9/97 (9)
All	36+	87	26/73 (36)	22/87 (25)	23/80 (29)	46/87 (53)	6/76 (8)	5/87 (6)	10/86 (12)
All	Total	186	59/159 (37)	36/184 (20)	40/152 (26)	78/185 (42)	9/146 (6)	14/185 (8)	19/183 (10)
Key: Presence/Absence (%); RKH = Reykholt, BES = Bessastaðir, RVK = Reykjavík, VEY = Viðey, HFE =Haffjarðarey, SKR = Skriðuklaustur, ÞSK = Skeljastaðir									

Table 3 Breakdown of pathological changes by sites, sex and age categories presented by the number of individuals affected and observable from each assemblage. Percentage in brackets

While no distinct pattern was noted between sexes, the older individuals from the 17th-19th century assemblages showed the highest frequencies (in some cases significantly higher) of calcifications on entheseal and interosseous attachment sites (Table 4). Calcification of the interosseous crest of the radii, ulnae, tibiae and fibulae was present on 12-24% of each bone in younger adults and in 26-46% of each bone in older adults. Entheseal calcification was observed in 6-27% of each long bone or os coxa in younger adults and in 30-57% of older adults (Table 4). In common with previous studies of entheseal changes, the prevalence and severity were higher amongst males than females and increased in the 36+ year age category (e.g. Meyer et al. 2011; Shuler et al. 2012; Henderson and Cardoso 2013; Santana-Cabrera et al. 2015).

4a ***Calcifications of entheseal attachment sites***

Period	*Site/Age/Sex*	*N*	*R deltoid*	*L deltoid*	*R linea aspera*	*L linea aspera*	*R soleal line*	*L soleal line*	*R iliac crest*	*L iliac crest*	*R ischial tuberosity*	*L ischial tuberosity*	*R pectoralis major*	*L pectoralis major*	*R pronator teres*	*L pronator teres*
17th-19th	RKH/BES/RVK/VEY	54	18/46 (39)	16/44 (36)	30/51 (59)	28/47 (60)	19/52 (37)	16/51 (31)	15/41 (37)	15/39 (38)	6/39 (15)	4/36 (11)	22/45 (49)	24/48 (50)	17/36 (37)	16/41 (39)
13th-16th	HFE/SKR	82	24/68 (35)	22/66 (33)	20/68 (29)	20/67 (30)	15/69 (22)	15/68 (22)	16/54 (30)	16/53 (30)	11/54 (20)	10/53 (19)	12/65 (19)	12/60 (20)	10/61 (16)	9/57 (16)
11th-12th	ÞSK	50	13/38 /34)	14/44 (32)	18/48 (38)	20/47 (43)	16/46 (35)	16/46 (35)	16/41 (39)	15/42 (36)	9/39 (23)	9/39 (23)	9/39 (23)	11/44 (25)	14/39 (36)	13/43 (30)
17th-19th	Females	23	7/20 (35)	4/17 (57)	12/22 (55)	12/21 (57)	10/23 (44)	8/22 (36)	1/14 (7)	0/13 (0)	6/18 (33)	6/16 (38)	7/19 (37)	8/20 (40)	6/20 (30)	6/18 (33)
17th-19th	Males	31	12/27 (44)	12/28 (43)	18/29 (62)	16/26 (62)	9/30 (30)	8/29 (28)	4/23 (17)	4/23 (17)	9/24 (38)	8/22 (36)	15/25 (60)	16/27 (59)	11/26 (42)	10/24 (42)
13th-16th	Females	51	14/44 (32)	14/42 (33)	13/43 (30)	13/41 (32)	11/42 (26)	11/41 (27)	11/37 (30)	10/36 (28)	6/36 (17)	5/40 (13)	4/44 (9)	6/51 (12)	5/40 (13)	5/36 (14)
13th-16th	Males	31	12/26 (46)	8/24 (33)	7/25 (28)	7/26 (27)	4/27 (15)	4/27 (15)	5/17 (29)	6/17 (35)	5/18 (28)	5/18 (28)	10/23 (44)	9/23 (39)	5/21 (24)	4/21 (19)
11th-12th	Females	26	2/17 (12)	3/23 (13)	6/25 (24)	7/24 (29)	2/23 (9)	2/23 (9)	2/23 (9)	2/16 (13)	5/20 (29)	2/18 (11)	2/18 (11)	0/18 (0)	1/23 (4)	5/18 (28)	5/21 (24)
11th-12th	Males	24	11/21 (52)	11/22 (50)	12/23 (52)	13/23 (57)	14/23 (61)	14/23 (61)	11/22 (50)	10/22 (46)	7/21 (33)	7/21 (33)	9/21 (43)	10/21 (48)	8/22 (36)	8/22 (36)
17th-19th	17-36	27	7/22 (32)	7/23 (30)	11/25 (44)	9/22 (41)	5/25 (20)	4/25 (16)	4/21 (19)	4/20 (20)	2/20 (10)	0/18 (0)	7/21 (33)	6/22 (27)	2/23 (10)	2/21 (11)
17th-19th	36+	27	11/24 (46)	12/24 (40)	19/26 (73)	19/25 (76)	14/27 (52)	12/26 (46)	11/20 (55)	11/19 (58)	4/19 (21)	4/18 (22)	15/23 (65)	18/24 (75)	15/23 (65)	14/20 (70)
13th-16th	17-36	51	11/42 (26)	11/42 (26)	9/44 (21)	9/43 (21)	10/45 (22)	10/45 (22)	6/30 (20)	6/30 (20)	2/30 (7)	2/30 (7)	7/41 (17)	7/40 (18)	5/38 (13)	5/37 (14)
13th-16th	36+	31	13/28 (46)	11/26 (42)	11/27 (41)	11/27 (41)	5/27 (19)	5/27 (19)	10/24 (42)	10/23 (43)	9/24 (38)	8/23 (35)	7/28 (25)	8/26 (31)	5/26 (19)	4/23 (17)
11th-12th	17-36	20	3/15 (20)	2/16 (13)	4/14 (29)	3/17 (18)	2/18 (11)	2/17 (12)	3/16 (19)	3/16 (19)	2/15 (13)	2/15 (13)	2/17 (12)	2/17 (12)	3/16 (19)	3/16 (19)
11th-12th	36+	30	10/24 (42)	12/29 (41)	15/30 (50)	17/30 (57)	15/29 (52)	15/30 (50)	14/26 (54)	13/27 (48)	8/25 (32)	8/25 (32)	8/23 (35)	10/28 (36)	12/24 (50)	11/28 (39)
All	Females	99	23/83 (28)	21/84 (25)	31/93 (33)	32/89 (36)	23/91 (25)	21/89 (24)	22/73 (30)	21/72 (29)	9/69 (13)	7/66 (11)	11/83 (13)	15/85 (18)	16/81 (20)	16/77 (21)
All	Males	87	32/72 (44)	31/73 (42)	38/78 (49)	36/75 (48)	28/80 (35)	27/80 (34)	26/63 (41)	26/63 (41)	18/64 (28)	17/63 (27)	35/70 (50)	36/72 (50)	26/69 (38)	23/68 (34)
All	17-36	99	21/79 (27)	17/78 (22)	24/88 (27)	21/82 (26)	17/88 (19)	16/86 (19)	13/67 (19)	13/66 (20)	6/65 (9)	4/63 (6)	16/79 (20)	15/79 (19)	10/77 (13)	10/74 (14)
All	36+	87	34/76 (45)	35/79 (44)	45/83 (54)	47/83 (57)	34/83 (41)	32/83 (39)	34/69 (49)	34/69 (49)	21/68 (31)	20/66 (30)	30/74 (41)	36/78 (46)	32/73 (44)	29/71 (41)
	Total	186	55/155 (35)	52/157 (33)	69/171 (40)	68/164 (41)	51/171 (30)	48/169 (28)	48/137 (35)	47/135 (35)	27/133 (20)	24/129 (19)	46/153 (30)	51/157 (32)	42/150 (28)	39/145 (27)
Key: Presence/Absence (%); RKH = Reykholt, BES = Bessastaðir, RVK = Reykjavík, VEY = Viðey, HFE =Haffjarðarey, SKR = Skriðuklaustur, ÞSK = Skeljastaðir; deltoid (insertion of deltoid); linea aspera (insertion of e.g. adductor longus, brevis and magnus); soleal line (origin of soleus); iliac crest (origin of gluteus maximus or medius); ischial tuberosity (origin of hamstrings: semitendinosus, semimembranosus, biceps femoris); pectoralis major (insertion); pronator teres (insertion)															

4b ***Calcifications of interosseous crests***

Period	*Site/Age/Sex*	*N*	*R tibia*	*L tibia*	*R fibula*	*L fibula*	*R radius*	*L radius*	*R ulna*	*L ulna*
17th-19th	RKH, BES, RVK, VEY	54	18/52 (35)	19/52 (37)	13/43 (30)	14/41 (34)	25/46 (54)	24/44 (55)	30/48 (63)	27/44 (61)
13th-16th	HFE, SKR	82	12/71 (17)	12/71 (17)	11/70 (16)	13/70 (19)	13/68 (19)	12/68 (18)	13/69 (19)	15/67 (22)
11th-12th	ÞSK	50	5/45 (11)	7/46 (15)	6/44 (14)	7/43 (16)	8/43 (19)	10/44 (23)	7/44 (16)	10/44 (23)
17th-19th	Females	23	11/24 (46)	10/22 (45)	7/18 (39)	7/15 (47)	8/19 (42)	8/19 (42)	11/20 (55)	11/20 (55)
17th-19th	Males	31	7/29(24)	9/30 (30)	6/25 (24)	7/26 (27)	17/27 (63)	16/25 (64)	19/28 (68)	18/25 (72)
13th-16th	Females	51	6/46 (13)	5/45 (11)	5/45 (11)	5/45 (11)	6/44 (14)	4/44 (9)	6/45 (13)	5/43 (12)
13th-16th	Males	31	6/24 (25)	6/24 (25)	6/24 (25)	8/24 (33)	7/23 (30)	8/23 (35)	7/23 (30)	10/23 (43)
11th-12th	Females	26	1/22 (5)	2/23 (9)	2/21 (10)	2/21 (10)	5/20 (25)	5/21 (24)	5/22 (23)	5/21 (24)
11th-12th	Males	24	4/23 (17)	5/23 (22)	4/23 (17)	5/22 (23)	3/23 (13)	5/23 (22)	2/22 (9)	5/23 (22)
17th-19th	17-36	27	7/26 (27)	9/25 (36)	3/19 (16)	5/17 (29)	9/23 (39)	9/22 (41)	12/24 (50)	12/23 (55)
17th-19th	36+	27	11/26 (42)	10/27 (37)	10/24 (42)	9/24 (38)	16/23 (70)	15/22 (68)	18/24 (75)	17/22 (77)
13th-16th	17-36	51	7/42 (17)	6/42 (14)	6/41 (15)	7/41 (17)	6/40 (15)	5/42 (12)	6/40 (15)	6/41 (15)
13th-16th	36+	31	5/28 (18)	6/28 (21)	5/28 (18)	6/28 (21)	7/27 (26)	7/25 (28)	7/28 (25)	9/25 (36)
11th-12th	17-36	20	0/18 (0)	1/18 (6)	0/17 (0)	0/17 (0)	2/17 (12)	2/16 (13)	2/19 (11)	2/17 (12)
11th-12th	36+	30	5/27 (19)	6/28 (21)	6/27 (22)	7/26 (27)	6/26 (23)	8/28 (29)	5/25 (20)	8/27 (30)
All	Females	99	18/91 (20)	17/91 (19)	14/84 (17)	14/81 (17)	19/83 (23)	17/81 (21)	22/87 (25)	21/84 (25)
All	Males	87	17/77 (22)	21/78 (27)	16/73 (22)	20/73 (27)	27/74 (36)	27/70 (39)	28/74 (38)	33/72 (46)
All	17-36	99	14/87 (16)	16/86 (19)	9/78 (12)	12/76 (16)	17/81 (21)	16/81 (20)	20/84 (24)	20/82 (24)
All	36+	87	21/81 (26)	22/83 (27)	21/79 (27)	22/78 (28)	29/76 (38)	30/75 (40)	30/77 (39)	34/74 (46)
All	Total	186	35/168 (21)	38/169 (22)	30/157 (19)	34/154 (22)	46/157 (29)	46/156 (29)	50/161 (31)	54/156 (35)
Key: Presence/Absence (%); RKH = Reykholt, BES = Bessastaðir, RVK = Reykjavík, VEY = Viðey, HFE =Haffjarðarey, SKR = Skriðuklaustur, ÞSK = Skeljastaðir;										

Table 4 Breakdown of entheseal (4a) and interosseous calcifications (4b) by sites, sex and age categories presented by the number of individuals affected and observable from each assemblage. Percentage in brackets

True prevalence rates for dental disease are presented in Table 5. Calculus had a high prevalence rate with 60% of maxillary and 68% of mandibular teeth affected. Linear enamel hypoplasia, most commonly observed on the canines and central incisors, had a low prevalence (maxilla 15%; mandible 16%). Enamel hypomineralisation or opacities (enamel mottling or staining) (maxilla 7%; mandible 8%), pitting (maxilla 3%; mandible 3%) and hypercementosis (maxilla 4%; mandible <1%) were even lower. Dental chipping was noted in 8% of maxillary and 7% of mandibular teeth. Enamel mottling was most frequently observed on the central incisors, followed by the other anterior teeth. Carious lesions had a very low prevalence across all teeth (maxilla 1%; mandible <1%).

Maxilla										
Tooth	***Present***	***Unobservable***	***AM loss***	***Pitting***	***Hypercementosis***	***Calculus***	***Caries***	***Hypomineralisation***	***LEH***	***Chipping***
RM3	84/186 (45)	78/186 (42)	24/186 (13)	3/84 (4)	2/84 (2)	65/84 (77)	0/84 (0)	4/84 (5)	5/84 (6)	1/84 (1)
RM2	129/186 (69)	24/186 (13)	23/186 (12)	2/129 (2)	0/129 (0)	96/129 (74)	1/129 (<1)	5/129 (4)	9/129 (7)	4/129 (3)
RM1	135/186 (73)	35/186 (19)	16/186 (9)	3/135 (2)	2/135 (1)	115/135 (85)	2/135 (1)	7/135 (5)	5/135 (4)	14/135 (10)
RPM2	134/186 (72)	36/186 (19)	16/186 (9)	3/134 (2)	3/134 (2)	93/134 (69)	1/134 (<1)	8/134 (6)	5/134 (4)	12/134 (9)
RPM1	139/186 (75)	31/186 (17)	16/186 (9)	5/139 (4)	5/139 (4)	82/139 (59)	0/139 (0)	8/139 (6)	18/139 (13)	10/139 (7)
RC	144/186 (77)	32/186 (17)	10/186 (5)	2/144 (1)	3/144 (2)	84/144 (58)	2/144 (1)	11/144 (8)	42/144 (29)	21/144 (15)
RLI	130/186 (70)	38/186 (20)	18/186 (10)	4/130 (3)	2/130 (2)	42/130 (32)	2/130 (2)	10/130 (8)	26/130 (20)	11/130 (8)
RCI	127/186 (68)	37/186 (20)	22/186 (12)	6/127 (5)	2/127 (2)	39/127 (31)	2/127 (2)	16/127 (13)	34/127 (27)	8/127 (6)
LCI	129/186 (69)	34/186 (18)	33/186 (18)	8/129 (6)	1/129 (<1)	39/129 (30)	0/129 (0)	18/129 (14)	39/129 (30)	10/129 (8)
LLI	127/186 (68)	32/186 (17)	27/186 (15)	2/127 (2)	1/127 (<1)	47/127 (37)	0/127 (0)	12/127 (9)	30/127 (24)	8/127 (6)
LC	141/186 (76)	36/186 (19)	9/186 (5)	4/141 (3)	1/141 (<1)	78/141 (55)	2/141 (1)	13/141 (9)	52/141 (37)	16/141 (11)
LPM1	138/186 (74)	34/186 (18)	14/186 (8)	5/138 (4)	2/138 (1)	93/138 (67)	3/138 (2)	12/138 (9)	24/138 (17)	12/138 (9)
LPM2	139/186 (75)	36/186 (19)	11/186 (6)	4/139 (3)	1/139 (<1)	96/139 (69)	1/139 (<1)	7/139 (5)	8/139 (6)	11/139 (8)
LM1	137/186 (74)	36/186 (19)	13/186 (7)	3/137 (2)	3/137 (2)	95/137 (69)	3/137 (2)	7/137 (5)	4/137 (3)	9/137 (7)
LM2	120/186 (65)	39/186 (21)	27/186 (15)	4/120 (3)	1/120 (<1)	95/120 (79)	3/120 (3)	8/120 (7)	5/120 (4)	5/120 (4)
LM3	88/186 (47)	80/186 (43)	19/186 (10)	5/88 (6)	2/88 (2)	61/88 (69)	2/88 (2)	6/88 (7)	5/88 (6)	3/88 (3)
All	2041/2976 (69)	648/2976 (68)	298/2976 (8)	63/2041 (3)	31/2041 (4)	1220/2041 (60)	24/2041 (1)	152/2041 (7)	311/2041 (15)	155/2041 (8)
Mandible										
Tooth	***Present***	***Unobservable***	***AM loss***	***Pitting***	***Hypercementosis***	***Calculus***	***Caries***	***Hypomineralisation***	***LEH***	***Chipping***
RM3	109/186 (59)	63/186 (34)	14/186 (8)	7/109 (6)	0/109 (0)	76/109 (70)	2/109 (2)	4/109 (4)	3/109 (3)	2/109 (2)
RM2	147/186 (79)	24/186 (13)	15/186 (8)	3/147 (2)	1/147 (<1)	113/147 (77)	2/147 (1)	6/147 (4)	5/147 (3)	10/147 (7)
RM1	142/186 (76)	25/186 (13)	19/186 (10)	3/142 (2)	1/142 (<1)	114/142 (80)	2/142 (1)	7/142 (5)	9/142 (6)	14/142 (10)
RPM2	141/186 (76)	26/186 (14)	19/186 (10)	5/141 (4)	0/141 (0)	106/141 (75)	0/141 (0)	10/141 (7)	19/141 (13)	13/141 (9)
RPM1	145/186 (78)	27/186 (15)	14/186 (8)	6/145 (4)	1/145 (<1)	104/145 (72)	1/145 (<1)	13/145 (9)	34/145 (23)	11/145 (8)
RC	145/186 (78)	33/186 (18)	8/186 (4)	6/145 (4)	0/145 (0)	97/145 (67)	0/145 (0)	15/145 (10)	63/145 (43)	12/145 (8)
RLI	142/186 (76)	29/186 (16)	15/186 (8)	1/142 (<1)	0/142 (0)	84/142 (59)	1/142 (<1)	12/142 (8)	27/142 (19)	10/142 (7)
RCI	136/186 (73)	28/186 (15)	22/186 (12)	2/136 (1)	1/136 (<1)	65/136 (48)	0/136 (0)	10/136 (7)	17/136 (13)	8/136 (6)
LCI	133/186 (72)	32/186 (17)	21/186 (11)	1/133 (<1)	0/133 (0)	65/133 (49)	1/133 (<1)	11/133 (8)	17/133 (13)	6/133 (5)
LLI	142/186 (76)	32/186 (17)	12/186 (6)	1/142 (<1)	0/142 (0)	72/142 (51)	0/142 (0)	13/142 (9)	26/142 (18)	7/142 (5)
LC	148/186 (80)	29/186 (16)	9/186 (5)	5/148 (3)	1/148 (<1)	95/148 (64)	1/148 (<1)	15/148 (10)	65/148 (44)	15/148 (10)
LPM1	145/186 (78)	30/186 (16)	11/186 (6)	5/145 (3)	0/145 (0)	99/145 (53)	0/145 (0)	15/145 (10)	34/145 (23)	16/145 (11)
LPM2	144/186 (77)	30/186 (16)	12/186 (6)	3/144 (2)	2/144 (1)	100/144 (69)	1/144 (<1)	10/144 (7)	17/144 (12)	10/144 (7)
LM1	135/186 (73)	32/186 (17)	19/186 (10)	3/135 (2)	1/135 (<1)	111/135 (82)	1/135 (<1)	10/135 (7)	8/135 (6)	12/135 (9)
LM2	140/186 (75)	27/186 (15)	19/186 (10)	3/140 (2)	1/140 (<1)	109/140 (78)	3/140 (2)	10/140 (7)	7/140 (5)	11/140 (8)
LM3	108/186 (58)	65/186 (35)	13/186 (7)	7/108 (6)	4/108 (4)	85/108 (79)	1/108 (<1)	10/108 (9)	2/108 (2)	3/108 (3)
All	2202/2976 (74)	532/2976 (18)	242/2976 (8)	61/2202 (3)	13/2202 (<1)	1495/2202 (68)	16/2202 (<1)	171/2202 (8)	353/2202 (16)	160/2202 (7)
Key: Presence/Absence (%) pooled across all sites/periods; Present (presence of tooth); Unobservable (post-mortem loss, unerupted, other); AM loss (ante-mortem loss); Pitting (enamel pitting); Hypercementosis (hypercementosis on tooth root); Calculus (calculus deposition); Caries (carious lesions); Hypomineralisation (enamel mottling or staining); LEH (linear enamel hypoplasia); Chipping (ante-mortem enamel chipping)										

Table 5 Breakdown of dental changes by tooth and pathological condition pooled across all sites/periods

Bone fluoride analysis

Cumulative bone fluoride concentrations were determined in all of the human bone samples and four soil samples. The fluoride concentrations of all samples (n=50) ranged from 223-4370 ppm with a mean of 2056 ppm. At Skriðuklaustur (n=36), the bone fluoride concentrations ranged between 223-4370 ppm and the overall mean was 2324 ± 1067 ppm. At Skeljastaðir (n=14), the bone fluoride concentrations ranged between 223-3030 ppm and the overall mean was 1366 ± 937

ppm. Fluoride concentrations, skeletal indicators and pathological changers for all of the individuals analysed from both sites are presented in Supplementary Table 1. Inter-site differences in fluoride concentrations were statistically significant, but intra-site differences across age and sex categories were not (Table 7). The bone fluoride concentrations were significantly higher at Skriðuklaustur than at Skeljastaðir, even across age and sex categories. From Skriðuklaustur, the majority (24/36; 67%) of individuals exhibited fluoride concentrations within the normal range (<3000 ppm), while the remainder (12/36; 33%) had elevated concentrations. Though none of the individuals analysed showed concentrations consistent with clinical skeletal fluorosis, 5/36 (14%) of individuals had elevated concentrations consistent with the pre-clinical phase (>3500 ppm) (Table 6). The mean fluoride bone concentration for non-adults from Skriðuklaustur was 1638 ± 1018 ppm, however, it was not significantly lower (p = 0.084) than the adults (2461 ± 1039 ppm). Only 2/14 (14%) of the individuals analysed from Skeljastaðir had elevated concentrations (Table 6).

The general background range of fluoride in soil worldwide is 30-500 ppm (Edmunds and Smedley 2005; Ozsvath 2008) but can temporarily increase following volcanic eruptions (Pyle and Mather 2009). However, measurements performed on archaeological soil may not accurately represent historic fluoride concentrations if extensive topsoil leaching occurred; soil concentrations eventually return to normal as the fluoride is washed out of the ash, soil and vegetation over time (Thorarinsson and Sigvaldason 1972; EFSA 2010). The soil concentrations (n=4) of fluoride were very low at both sites (<68 ppm), indicating that diagenetic enrichment from the burial environment was unlikely.

Individual	***Sex***	***Age***	***F ppm***	***Severity***	***Pathological changes***
SKR 100	M	YA	4340	Pre-clinical	Linear enamel hypoplasia; enamel hypomineralisation/mottling; ante-mortem chipping; cribra orbitalia; osteitis and anterior bowing of ulnae, fibulae and tibiae; calculus; atlanto-occipital membrane ossification, new bone formation on fibulae and tibiae; ulnae with lytic lesions; glabella with cranial depressions; distal humeri flared with lamellar bone
SKR 126	F	OA	3690	Pre-clinical	Gummatous lesion on right parietal; slight degenerative change throughout spinal articular facets; healed fracture with bone callous on right 1st and 2nd hand phalanges; right 1st and 2nd hand phalanges ankylosed (due to fracture?); linear enamel hypoplasia; enamel hypomineralisation/mottling; enamel pitting; entheseal changes to deltoid tuberosity, soleal line, teres major and others; maxillary sinusitis; hydatid cysts
SKR 181	F	YA	3620	Pre-clinical	Left femur with large area of lamellar bone formation; necrotic lesion on frontal (reaction to trauma?); calculus; periapical lesions; pronounced alveolar resorption; ante-mortem chipping; mandibular molars with lingual wear
SKR 195	F	OA	3640	Pre-clinical	Left tibia and femur with remodelling lamellar bone formation; frontal bone with possible gummatous lesion; atlanto-occipital membrane ossification; linear enamel hypoplasia; enamel pitting; ante-mortem chipping; calculus; entheseal changes to deltoid tuberosity and pronator teres; left tibia shaft with osteitis; maxillary sinusitis; nodular bone formation throughout ribs
SKR 135	M	YA	4370	Pre-clinical	Left tibia with striated and porous bone formation on medial aspect of shaft; femora with anterior-posterior bowing; fibulae with medial-lateral bowing; atlanto-ocipital membrane ossification; linear enamel hypoplasia; enamel hypomineralisation/mottling; ante-mortem chipping; calculus; entheseal changes to teres major, pronator teres and others; severe entheseal changes to deltoid tuberosities (marked by cavitations); interosseous mineralisation of tibiae and fibulae
SKR 128	F	OA	3190	Elevated	Kyphosis of spine; frontal bones with button osteomata; numerous vertebral compressions fractures; healed cranial trauma? (circular, concave lesion with reactive new bone); linear enamel hypoplasia; entheseal calcification of deltoid tuberosity, linea aspera and others
SKR 189	F	YA	3450	Elevated	Femora, tibiae and fibulae with striated, porous, lamellar bone formation covering all of shafts; calculus; Linear enamel hypoplasia; enamel pitting; ante-mortem chipping; entheseal changes to fibulae and soleal lines; hydatid cyst
SKR 115	M	OA	3080	Elevated	Linear enamel hypoplasia
SKR 150	M	YA	3440	Elevated	Tibiae with lamellar new bone formation; degenerative joint disease in C1 and C2; atlanto-occipital membrane ossification; ossification of the stylohyoid chain (Eagle's syndrome?);

					left tibia ankylosed with fibula; calculus; alveolar resorption; linear enamel hypoplasia; enamel hypomineralisation/mottling; ante-mortem chipping; left linea aspera with reactive activity: porous, striated bone deposition on entire length of attachment site; right tibia with osteitis
SKR 152	M	YA	3050	Elevated	Clavicles with lytic activity in the rhomboid fossae that penetrates from inferior to superior aspect; linear enamel hypoplasia; ante-mortem chipping; calculus; two rib fragments with nodular bone formation
SKR 201	F	YA	3290	Elevated	Tibiae, fibulae and ulnae with erosive lesions and lamellar new bone formation on shafts; possible gummatous palatine lesion; calculus; periapical lesions; alveolar resorption; linear enamel hypoplasia; enamel hypomineralisation/mottling; ante-mortem chipping; entheseal changes to deltoid tuberosity and soleal line; maxillary sinusitis; prominent spaces between mandibular teeth; retained deciduous teeth; differential dental wear
SKR 221	?	NA	3180	Elevated	non-adult/adolescent. linear enamel hypoplasia.
ÞSK 4	F	YA	3030	Elevated	degenerative joint disease throughout spine and rib joints; deviated septum; healed rib fracture; calculus; linear enamel hypoplasia; cribra orbitalia; nodular bone formations on 3 ribs; incomplete medial cleft premaxilla; bone spicule extending from intraconal aspect of right orbit
ÞSK 44	M	OA	3010	Elevated	degenerative joint disease on L5 and S1 bodies and facets and proximal and distal humeri, ulnar guiding ridges, fovea capitis, scapula glenoids and temporo-mandibular joints; left posterior parietal with healed blunt force trauma; osteochondritis dissecans on distal right tibia; calculus; alveolar resorption; severe enamel hypomineralisation/mottling; entheseal changes to deltoid tuberosity, linea aspera, soleal line, teres major, posterior iliac crests and others; interosseous mineralisation of tibiae and fibulae; maxillary sinusitis; partial cleft neural arch in S4 (left)
Key: SKR = Skriðuklaustur, ÞSK = Skeljastaðir; NA = Non-adult, M = Male, F = Female; Non-adult (<17), Younger adult (17-36), Older adult (36+); Elevated (3000-3500 ppm F), Pre-clinical (>3500 ppm F)					

Table 6 Individuals with elevated bone fluoride concentrations and dental and skeletal changes potentially associated with fluorosis

SKR					
Sex	**n**	**Mean**	**Range**	**>3000 ppm**	**>3500 ppm**
M	12	2571 ± 1109	655-4370	42% (5/12)	17% (2/12)
F	18	2388 ± 1016	223-3640	33% (6/18)	17% (3/18)
NA	6	1638 ± 1018	468-2520	17% (1/6)	0% (0/6)
Age	**n**	**Mean**	**Range**	**>3000 ppm**	**>3500 ppm**
YA	14	2681 ± 1252	223-4370	50% (7/14)	21% (3/14)
OA	16	2269 ± 803	525-3690	25% (4/16)	13% (2/16)
NA	6	1638 ± 1018	468-2520	17% (1/6)	0% (0/6)
All	36	2324 ± 1067	223-4370	31% (11/36)	14% (5/36)
ÞSK					
Sex	**n**	**Mean**	**Range**	**>3000 ppm**	**>3500 ppm**
M	9	1501 ± 859	233-3010	11% (1/9)	0% (0/9)
F	5	1123 ± 1124	223-3030	20% (1/5)	0% (0/5)
Age	**n**	**Mean**	**Range**	**>3000 ppm**	**>3500 ppm**
YA	4	1774 ± 1110	396-3030	25% (1/4)	0% (0/4)
OA	10	1203 ± 869	223-3010	10% (1/10)	0% (0/10)
All	14	1366 ± 937	223-3030	14% (2/14)	0% (0/14)

Table 7 Bone fluoride concentrations means according to age, sex and total amongst individuals sampled from Skriðuklaustur and Skeljastaðir. Elevated fluoride concentrations are indicated by

presenting with >3000 ppm. Concentrations consistent with the pre-clinical stage of skeletal fluorosis are indicated by those presenting with >3500 ppm

Site	*Category*	*f*	*p*
SKR vs ÞSK	All individuals	0.633	**0.005**
SKR vs ÞSK	All Adults	0.715	**0.002**
SKR vs ÞSK	Females vs Females	0.675	**0.025**
SKR vs ÞSK	Males vs Males	0.480	**0.027**
SKR vs ÞSK	Younger vs Younger	0.954	0.210
SKR vs ÞSK	Older vs Older	0.756	**0.004**
SKR	Adults vs Nonadults	0.917	0.084
SKR	Males vs Females	0.723	0.645
SKR	Older vs Younger	0.102	0.286
ÞSK	Males vs Females	0.480	0.492
ÞSK	Older vs Younger	0.500	0.322

Table 8 Variance and p-values for statistical differences in fluorine bone concentrations from the individuals sampled from Skriðuklaustur and Skeljastaðir

Discussion

Skeletal Changes

Across all time periods, fractures, interosseous and entheseal calcifications were higher (in some cases significantly so) among men and individuals belonging to the 36+ category, likely because they are greatly affected by age and sexually dimorphic differences in biology and behaviour – as has been noted in other studies (e.g. Meyer et al. 2011; Shuler et al. 2012; Henderson and Cardoso 2013; Santana-Cabrera et al. 2015). However, fluoride accumulates with age, thus long-term, low-level fluoride exposure could also be implicated (Arnala et al. 1985; Barbier et al. 2010; Petrone et al. 2011).

A high proportion of individuals (42%) exhibited at least one healed ante-mortem post-cranial fracture. This prevalence was higher than observed at Herculaneum (32%), where skeletal fluorosis was severe and endemic (Petrone et al., 2013). The higher fracture rate observed in Icelandic skeletal remains may, however, also be associated with the local geography or landscape, environmental conditions and occupational factors in addition to, or other than, fluorosis. Soft tissue ossifications (e.g. cartilage, glands) were recorded in 20% (36/184) of individuals and, as expected, were observed more frequently in older individuals (25%; 22/87) than younger individuals (14%; 14/97), also likely as a function of age. The low rates of ankylosis observed in the spine (6%) and non-spinal joints (8%) were not indicative of fluorosis. In comparison, Petrone et al. (2013) observed ankylosis in at least one anatomical site in 39% of the Herculaneum individuals. In clinical studies, changes to the atlanto-occipital membrane or attachment site are normally reported following cases of traumatic injury (e.g. whiplash) but underlying congenital or developmental conditions can act as predisposing factors (Vangilder and Menezes 1983; Lustrin et al. 2003; Riascos et al. 2015). Perhaps the higher frequency noted in older adults from the 10th-11th century assemblage relates to occupational behaviours, such as heavy lifting associated with the farming lifestyle.

The pathogenesis for the ossification of the ligamentum flavum has been observed to be correlated with diet and genetics (e.g. Mobbs and Dvorak 2007; Shepard et al. 2015; Geber and Hammer 2018), although numerous other factors may lead to its pathological change. The higher prevalence observed in the 17th-19th century assemblages (43%) than in the 13th-16th century (22%) and 11th-12th century (16%) assemblages may reflect a combination of factors including the richer, more varied diet that the upper class consumed (see Jónsson 1998), genetic drift (see Ebenesersdóttir et al. 2018), the diversification of occupations occurring during the urbanization of Reykjavík (see Harrison and Snæsdóttir 2013; Hayeur-Smith et al. 2018) and environmental fluoride contamination from the Skaftáreldar fires (Lakí) of 1783-1784 AD (see Steingrímsson 1998; Halldórsson 2013). Ossification of the ligamentum flavum was observed in 23.6% of male and 15.6% of female thoracic vertebrae in a medieval sample (900-1000 AD) from Poland (Swedborg 1974). In a study of English skeletons from the *Mary Rose*, 46.3% of individuals presented with ligamentum flavum ossification in the thoracic spine, which was attributed to strenuous physical activities (Stirland and Waldron 1997).

Overall, less than 41% of individuals of all ages from all sites displayed evidence of calcification on interosseous or entheseal attachment sites on at least one bone. By comparison, at Herculaneum, 92% of individuals (all ages) exhibited notable calcification on at least one bone (see Petrone et al. 2013). Numerous studies have reported rates of entheseal changes similar to those recorded here, all demonstrating a strong correlation with increasing age rather than activity patterns or specific conditions such as fluorosis (e.g. Campanacho and Santos 2012; Henderson and Cardoso 2013; Henderson et al. 2013). Bones displaying calcification on the interosseous crests and entheseal attachment sites were slightly more prevalent in the 17th-19th century assemblages than the older assemblages. Since fluoride also increases with age, the higher prevalence of entheseal and interosseous calcifications observed in these assemblages could be associated with increased fluoride concentrations in the environment following Iceland's most catastrophic volcanic eruption – the Skaftáreldar fires (Lakí) of 1783-1784 AD. Aside from fluoride contamination, the population faced serious malnourishment, deficiency diseases became endemic (e.g. scurvy) and plagues, such as smallpox, raged across the country (Pétursson et al. 1984; Halldórsson 2013).

According to folklore, historical records and supposition due to latitude, it is thought that rickets and osteomalacia (vitamin D deficiency) were common in historical Iceland (Jónsson 1998; Sigurðardóttir 2017), although it is not frequently diagnosed in the Icelandic skeletal record (Steffensen 1939; Gestsdóttir 1991; Sundman 2011; Zoëga and Murphy 2016). In this study, the frequency of vitamin D deficiency cases is comparable with studies conducted on British adult skeletons dating from the 18th-19th centuries AD, which found a prevalence of 14% (20/135) for residual rickets (Brickley et al. 2010) and 1.43% (19/1323) for osteomalacia (Ives and Brickley 2014). Osteomalacia is an important predisposing factor for skeletal fluorosis (Gupta et al. 1996; Khandare et al. 2005) but diagnosis can be problematic (Ives and Brickley 2014). While vitamin D deficiency was equal between men (n=3) and women (n=3) at Skeljastaðir (11th-12th century), it was observed more frequently in women from the 13th-16th century (n=6) and 17th-19th century (n=4) assemblages than in men (n=2 and n=1, respectively). Although these differences are not statistically significant and may be an artefact of the small sample sizes, gendered social roles may also be reflected: it is believed that women and children likely spent more time indoors than men, particularly in the later periods (see Norrman 2008; Hayeur-Smith 2014; Veselka et al. 2018). Although about 3% of individuals showed evidence of osteomalacia in the present study, about

8% of the individuals affected by skeletal fluorosis at Herculaneum presented with osteomalacia (see Petrone et al. 2013).

Dental Changes

Previous research has noted a very high rate of dental wear, likely from parafunctional activities (e.g. during the weaving of wool fabric called *vaðmál*) and the consumption of the typical historic Icelandic diet of tough and gritty foods, dairy products and acidic beverages (Scott and Jolie 2008; Lanigan and Bartlett 2013; Richter and Elíasson 2017; Hoffman 2018). Dietary staples included dried fish, shellfish, stone-ground grain, dried or cured meat, milk, cheese and fermented milk products (i.e. *mysa, sýra*) (Gísladóttir 1999; Mehler 2011; Svanberg and Ægisson 2012).

Dental chipping, which could indicate poorly mineralized enamel (e.g. as caused by dental fluorosis) (Thylstrup and Fejerskov 1978), was observed in 7% of teeth, but this is likely related predominately to diet rather than solely poor enamel mineralization. Linear enamel hypoplasia may manifest as a result of toxic exposure to fluoride during childhood (Thylstrup and Fejerskov 1978; Marklein et al. 2016; Petrone et al. 2011, 2013) and it was more common (16%) in this study than any other enamel defects (pitting, 3%; hypomineralisation/mottling, 8%) (Table 8). Enamel hypomineralisation (mottling) were only slightly more frequent than are normally observed in healthy teeth from modern populations (ca. 5%) (Hillson 1996). The prevalence of hypercementosis was very low (maxilla, 4% of maxillary; mandible, <1%). For comparison, Corruccini et al. (1987) reported a very high prevalence (85% of 104) of hypercementosis in 17th-19th century slaves from Barbados. Hypercementosis is often idiopathic but can result from numerous local and systemic factors (Pinheiro 2008; García-González et al. 2018) as well as dental fluorosis (Littleton 1999).

The very low prevalence of carious lesions (<1%) is likely to be related to the high calculus rates (>60%) (see Green et al., 2005) and the Icelandic diet, which was high in protein and low in grains, flour and sugar prior to the 20th century (see Jónsson 1998; Gísladóttir 1999; Sigurðsson 2010; Mehler 2011; Bjarnadóttir 2016). Considering the high calculus rate, fluoride bound within it may have also contributed to cariostasis (see Tatevossian 1990; Aspiras et al. 2010) in addition to low natural fluoride concentrations in Icelandic drinking water or aquifers (see Gunnarsdóttir et al. 2016). Overall, the results of the dental analyses indicate that the childhood disease burden of dental fluorosis was likely low, although some individuals may have been affected. For example, at Herculaneum, 47% of teeth exhibited linear enamel hypoplasia, 55% had enamel pitting, 18% presented with enamel hypomineralisation (mottling), 20% of teeth exhibited dental caries and hypercementosis was noted in a few cases (Petrone et al. 2013).

Fluoride Concentrations

In both palaeopathological (e.g. Jolly et al. 1969; Frohlich et al. 1989; Grimaldo et al. 1995; Littleton 1999; Yoshimura et al. 2006; Petrone et al. 2011, 2013) and modern studies (e.g. Ayoob and Gupta 2006), fluoride bone concentrations have been categorized into indefinite ranges associated with progressive changes. The condition particularly affects older adults, due to the lifelong retention of fluoride in the skeleton (Arnala et al. 1985; Barbier et al. 2010; Petrone et al. 2011) thus elevated concentrations in elderly individuals do not necessarily indicate fluorosis

(Richards et al. 1994). Essentially, background concentrations for normal fluoride concentrations in unaffected bone range between <500-3000 ppm dependent upon age and geographic residence (Eble et al. 1992; Sastri et al. 2001; Petrone et al. 2013). While osteosclerosis and renal impairment may occur with elevations just beyond the normal threshold, extensive skeletal changes are normally seen with concentrations of 6000-9000 ppm (clinical phase 1-2). Concentrations of >9000 ppm usually indicate crippling skeletal fluorosis (clinical phase 3) (USDHHS 1991; Ayoob and Gupta 2006). Although skeletal fluorosis has occurred at lower bone concentrations, this study considers >3500 ppm (pre-clinical phase) in cortical bone the threshold for elevated fluoride concentrations potentially suggestive of bone fluorosis (see Franke et al. 1975; Smith and Hodge 1979; USDHHS 1991). Concentrations between 3000-3500 ppm are considered elevated, though such concentrations are not necessarily abnormal or indicative of toxic fluoride exposure.

The fluoride concentrations of all samples ranged between 223-4370 ppm with a mean of 2056 ppm. By comparison, at Herculaneum the concentrations were far more indicative of fluorosis, ranging between 2042-11342 ppm with a mean of 6672 ppm. In contrast to the results from Iceland, the fluoride concentrations from Herculaneum also clearly increased as a function of age (Petrone et al. 2013). Although the overall mean fluoride values were significantly higher (p = 0.005) at Skriðuklaustur (2324 ± 1067) than at Skeljastaðir (1366 ± 937), this difference may be biased by the small sample size. No statistical differences were observed across age or sex at either site (Table 6). Eleven individuals from Skriðuklaustur and two individuals from Skeljastaðir had elevated bone fluoride concentrations, but only five of these individuals had concentrations indicative of the pre-clinical phase of skeletal fluorosis (Table 8). All but two individuals with elevated or pre-clinical concentrations were categorized as younger adults, possibly indicating increased exposure (e.g. from volcanic emissions) beyond normal age-related fluoride retention from uncontaminated drinking water. One of these older adults was a female (SKR 128) who exhibited multiple vertebral fractures and reduced bone density under microscopy and radiography, indicative of osteoporosis. In a clinical study on a Japanese population, Ishiguro et al. (1993) found that bone fluoride concentrations steadily increased with age in men, while elevations were more noticeable in women over 55 years old likely due to the concurrence of menopause and senile osteoporosis. The elevated concentration (3190 ppm) in SKR 128 may therefore correspond with age related accumulation and osteoporosis rather than toxic exposure. The individuals with elevated fluoride concentrations showed above average rates of linear enamel hypoplasia (83%, 10/12), enamel hypomineralisation (mottling/staining) (50%, 6/12) and pitting and ante-mortem chipping (23%, 3/12). However, the rates of entheseal and/or interosseous calcifications (50%, 6/12), fractures (33%, 4/12), ankyloses (16%, 2/12) and joint disease (25%, 3/12) were not notably different between individuals with elevated fluoride concentrations and the rest of the population sample when controlled for age and sex. None of them showed evidence of ossification of the ligamentum flavum, but one (SKR 150) individual had an elongated, ossified stylohyoid chain measuring 56.3 mm. The styloid process normally measures 20-30 mm on average and symptoms (e.g. cervicofacial pain, cerebral ischemia, dysphagia) of Eagle's syndrome are normally clinically observed in individuals with styloid processes exceeding a mean length of 40 mm (see Balcioglu et al. 2009; Salega and Farba 2018).

At Skriðuklaustur, the non-adults showed the lowest mean fluoride value (1638 ± 1018), likely because bone fluoride concentrations increase with age and should be low if toxic exposure has not occurred. All the non-adults had normal fluoride concentrations, except one teenager (SKR 221) with a concentration of 3180 ppm. Due to the hand to mouth activities and other behavioural tendencies, such as outdoor play, children tend to be more easily exposed to toxic earth elements if they are present in the local environment (Wittners et al. 2002; Jacobs and Nevin 2006). As fluoride leaches out of soil at a very slow rate (Ayoob and Gupta 2006), it is possible that this individual was exposed to volcanogenic fluoride in soil and groundwater from the eruption of Veiðivötn (1477 AD) that occurred just 16 years before the establishment of the monastery. Two young, male adults (SKR 100 and 135) showed the highest fluoride concentrations. SKR 100 presented with a fluoride concentration of 4340 ppm and ante-mortem chipping and enamel pitting in both mandibular and maxillary teeth, but entheseal changes could not be evaluated due to severe periosteal changes associated with treponemal disease. SKR 135 had a fluoride concentration of 4370 ppm, severe entheseal changes to the deltoid tuberosities resulting in cavitations and hypomineralisation and mottling (brown stains and white opacities) in the dental enamel of four mandibular teeth. Additionally, he presented with mineralization of the right tibia to fibula interosseous crest and bowing (i.e. residual rickets) of the femora. An older, male adult (SKR 174) that exhibits skeletal changes suggestive of Paget's disease presented with a fluoride concentration of 2120 ppm, indicating that advanced skeletal fluorosis is an unlikely differential diagnosis (see Fig. 5).

The absorption of calcium, lead and other trace elements into the bone hydroxyapatite can alter or decrease the absorption of fluoride into the bone hydroxyapatite. Clinical studies indicate that simultaneous exposure to fluoride and lead causes an increase in lead concentrations in calcified tissues, without increasing bone fluoride concentration (Whyte et al. 2008; Sawan et al. 2010; Leite et al. 2011). The lowest fluoride concentrations (<525 ppm F) presented in individuals with treponemal disease that were also exposed to lead during childhood (>0.7 ppm in enamel): SKR 22 (2.7 ppm Pb) and SKR 23 (4.1 ppm Pb) (see Walser III et al., in press). The low fluoride concentrations in these individuals could partly correspond with simultaneous exposure to lead – if exposure persisted into adulthood – or other toxic heavy metals that interact with the bone hydroxyapatite. Calcium in Icelandic aquifers (range 1.3-52.8 ppm; median 5.5 ppm) is 3-5 times higher than in other Nordic countries (Gunnarsdóttir et al. 2016) and certain components of the historical diet (e.g. dairy, bone marrow) are likewise high in calcium. As calcium intake can reduce fluoride uptake (Whyte et al. 2008), it is thereby also worth considering whether these individuals were provided special, nourishing diets as long-term medical patients at the monastery. Recent isotopic analyses of carbon and nitrogen conducted on skeletal remains from Skriðuklaustur indicate a mixed terrestrial and marine diet, while at Skeljastaðir the results indicate a greater emphasis on terrestrial protein (Walser III et al. in press). Fish were an important part of the diet since the Settlement period but never as much as during the Catholic period (c. 1000-1550 AD) due to religious fasting (Kristjánsdóttir 2017). Since both fresh and dried fish are considerable sources of modern fluoride exposure (Shomar et al. 2004; Marva 2011; Ganta et al. 2015) diet could be implicated as a potential origin of the higher fluoride levels noted at Skriðuklaustur than at Skeljastaðir.

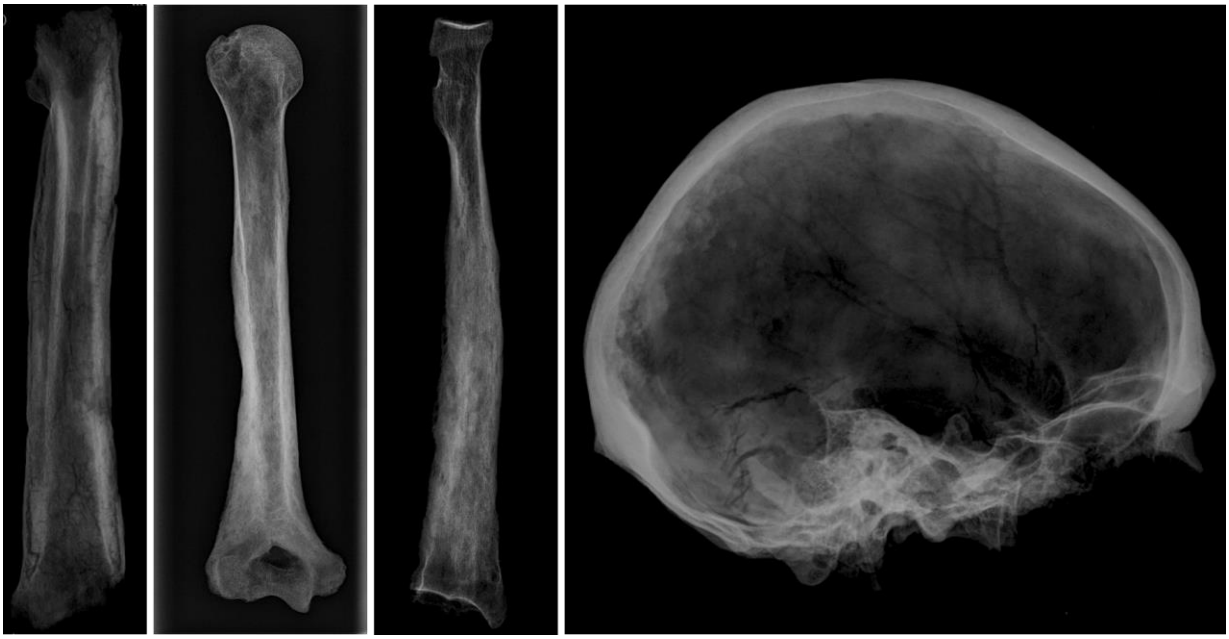

Fig. 5 Radiographs showing skeletal changes observed in the older adult male (SKR 174) with Paget's disease, a differential diagnosis of skeletal fluorosis, from Skriðuklaustur. From left: left femur (anterior), right humerus (anterior), right radius (anterior) and cranium (right lateral aspect). © Joe W. Walser III, National Museum of Iceland

The overall average bone fluoride concentration at Skeljastaðir was lower than expected, considering the sites proximity to the volcano Hekla, which is known to be a heavy fluoride (and mercury) emitter (Coderre and Steinthorsson 1970; Thorarinsson and Sigvaldason 1970; Thordarson and Larsen 2007). The highest fluoride concentrations were primarily noted amongst the individuals (ÞSK 4, 44) that also presented with the highest mercury concentrations in another study (see Walser III et al., 2018). As some of the individuals buried in the Skeljastaðir cemetery happen to post-date the 1104 AD eruption (Sveinbjörnsdóttir et al. 2010), it is possible that the individuals with high elemental concentrations were substantially exposed to volcanic emissions during the eruption, while others may have only been exposed to passive emissions. For example, an older, adult male (ÞSK 44) had a fluoride concentration of 3010 ppm as well as entheseal and interosseous calcification on the long bones and osteochondritis dissecans on the proximal end of the right ulna. Furthermore, at least 14 individuals from Skeljastaðir presented with notable entheseal changes that could theoretically relate to volcanogenic fluoride exposure (Gestsdóttir 1998, 2009; this study).

Conclusions

A social group's ability to cope with environmental stresses plays a major role in disaster mitigation (Black 1981). Risk assessment likely played an ancillary role in the selection of places for farms and villages in historical Iceland. In New Zealand, for example, Maori legends and ethnographic data describe taboos about entering areas with high volcanic risk (Lowe et al. 2002). Entire regions or settlements can be destroyed as a consequence of volcanic eruptions, causing population displacement and potential cultural discontinuity through separation from known

resources, allies and trade routes. However, place abandonment should likely be perceived as a short-term response because many abandoned settlements are later reoccupied or relocated nearby (Black 1981). Even after the abandonment of Skeljastaðir and other farms in Þjórsárdalur following the eruption of Hekla in 1104 AD, human activity and animal grazing persisted at least until a subsequent eruption in 1300 AD (Dugmore et al. 2007).

Overall, the results indicate that the skeletal and dental changes noted in this study are more likely to relate to the environment, population dynamics, culturally mediated behaviours (e.g. marine food sourcing, gendered social roles) and increasing urbanisation than serious fluoride contamination. These results are in line with a previous study by Gestsdóttir et al. (2006) who analysed bone fluoride concentrations using ICP-MS in combination with radiography and palaeopathological analyses on skeletal remains from two cemeteries, located near the Laki fissure, which were in use during the 1783-1784 AD eruption. The results showed no bone changes indicative of fluorosis and the bone fluoride concentrations were within the normal background limits (Gestsdóttir et al. 2006). The human population likely fled during eruptions and avoided contaminated drinking water, unlike the livestock.

However, low-level chronic exposure or fluctuations in fluoride concentrations following eruptions or increased geothermal activity may be indicated. Although the fluoride concentrations determined from individuals buried at Skriðuklaustur (mean 2324 ± 1067 ppm) and Skeljastaðir (mean 1366 ± 937 ppm) are predominately within the normal range, at least 12 of the individuals analysed were likely exposed to fluoride contamination. At Skriðuklaustur, five individuals had elevated concentrations (>3000 ppm) and another five individuals had fluoride concentrations consistent with the pre-clinical phase (>3500 ppm) of skeletal fluorosis. At Skeljastaðir only two individuals had elevated concentrations.

Though a fluorotic origin cannot be ruled out without bone fluoride analyses, the overall data suggests that industrialisation, urbanisation, rich diets, increased genetic diversity and marine food sourcing likely relate to the increased prevalence of changes to entheses and interosseous membranes observed in the 17th-19th century assemblages (RKH, BES, REY and VEY). Still, some historical accounts from the 16th-18th centuries claim that European travelers in Iceland perceived the country to be primitive. This stereotype, amongst others, persisted into at least the 19th century (Ísleifsson 1996; Loftsdóttir 2008). Recent evidence indicates that while Iceland remained rural well into modern times, the overall health, economic standing and general living conditions were not notably different from any of the neighboring countries (Júlíusson 2018; Kristinsson 2018). Even considering some potentially predisposing factors, the disease burden of fluorosis appears to be lower than originally hypothesised in historical Iceland. Unlike the arid Arabian Gulf where fluorosis was common in the past and the present, Iceland has a subarctic climate that may have been an important factor in the low representation of fluorosis despite being a highly active volcanic region. Thus, the danger of volcanic eruptions may have not been so hidden at all when it was possible to temporarily move away – even if only a short distance – from the visible fires and the ashfall that spread across the country. Perhaps these dangers were only concealed to those living within the closest vicinity of eruptions and passive volcanic emissions. In the future it may be important to consider the stability and preparedness of social, political, environmental and economic conditions that regions subject to extreme volcanic events maintain rather than the severity of the immediate and temporary physical effects they cause.

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Supplementary Information: Osteological Methods

Standard anthropological methods as outlined by Buikstra and Ubelaker (1994) and Brickley and McKinley (2004) were used during osteological analysis to estimate age, sex and pathological conditions. Age was estimated according to dental attrition (Brothwell 1981), morphological of the auricular surface (Lovejoy et al., 1985), cranial suture closure (Meindl and Lovejoy 1985; İşcan et al. 1984, 1985), age-related changes to the pubic symphysis (Brooks and Suchey 1990), epiphyseal fusion (Buikstra and Ubelaker 1994; Scheuer and Black, 2000), dental eruption and development (Ubelaker 1989; AlQahtani 2009) and acetabular degeneration (Calce 2012). Sex determination was performed using metrical estimations, cranial morphology and os coxae morphology (Buikstra and Ubelaker 1994) and distal humerus morphology (Rogers et al. 1999; Falys et al. 2005; Vance et al. 2011). Differential diagnoses for pathological conditions were considered according to the descriptions in reference material by Ortner (2003), Roberts and Manchester (2010) and Aufderheide and Rodriguez-Martin (2011).

Though not presented in the current study, periosteal new bone formation was described by type (e.g. lamellar, woven) and location (e.g. Weston 2012). Pathological ossifications (e.g. thyroid gland, organs) and the ossification of cartilage were recorded according to examples from Steinbock (1989) and Binder et al. (2016). Ossification of the ligamentum flavum was scored as present (grade 2+) or absent (grade 0-1) at the caudal or cranial attachment sites of the thoracic vertebrae, according to the criteria described by Geber and Hammer (2018). Ossification of the ligamentum flavum was only considered observable if at least 6 of the thoracic vertebrae were present. Ankyloses were recorded when two vertebrae or other bones (e.g. long bones or hip joints) were observed. Ankyloses clearly occurring due to congenital or development causes (e.g. block vertebrae, congenital radioulnar synostosis) were not considered to be related to skeletal fluorosis. In some cases, radiography was used for assessing aetiology. See Rogers et al. (1985), Petrone et al. (2013) and Ventades et al. (2018) for examples. Ankyloses were only considered observable if at least 50% of the spine and 50% of the post-cranial bones were present.

Calcification of soft tissues (e.g. tendons, ligaments) at entheseal attachment sites analysed include pronator teres (insertion), pectoralis major (insertion), posterior iliac crest (origin of gluteus medius or maximus), ischial tuberosity (origin of hamstrings: semitendinosus, semimembranosus, biceps femoris), deltoid (insertion), linea aspera (insertion of e.g. adductor longus, brevis and magnus), soleal line (origin of soleus muscle) and the interosseous crests (i.e. ligaments) of the radius, ulna, tibia and fibula. Entheseal and interosseous calcifications (e.g. ossification exostoses) were recorded as present or absent following the method outlined by Mariotti et al. (2007). Alterations to the posterior margin of the foramen magnum at the attachment site of the atlanto-occipital membrane were recorded using the same criteria. This research did not aim to reconstruct activity patterns using musculo-skeletal markers and recognises that entheseal changes are age related, so the changes were not scored on a scale, but rather as present (score 2-3) or absent (score 0-1) (see Mariotti et al. 2007). Healed or unhealed ante-mortem fractures were recorded as present or absent (see Buikstra and Ubelaker 1994; Lovell 1997; Judd 2002; Brickley and McKinley 2004; Mitchell and Brickley 2017). No cases of sharp force or projectile trauma were observed, therefore fractures only refer to blunt force trauma in this study. Vitamin D deficiency (osteomalacia and residual rickets) was assessed according to the pathological indicators described by Brickley et al. (2005, 2010) and Ives and Brickley (2014). Individuals presenting with at least one long bone with marked bowing were considered to exhibit residual rickets. Individuals with multiple fractures and morphological changes to the sternum, ribs, os coxae or scapulae in addition to residual rickets were considered to exhibit possible osteomalacia.

Enamel hypomineralisation (mottling) was recorded as present or absent according to the criteria described by Hillson (2008) and methods therein. The descriptions by Dean (1936) Alvarez et al. (2009) and Blinkhorn and Mekertichian (2013) were also considered when evaluating enamel defects, such as mottling and pitting associated with dental fluorosis . All dental analyses were recorded by individual tooth and the results are presented by presence of lesion against total teeth observable. Linear enamel hypoplasia was recorded according to presence and absence of at least one hypoplastic line. Enamel defects were considered unobservable if the tooth crown exhibited severe attrition or carious lesions on the buccal/labial aspects of the teeth. Caries were recorded according to the criteria described by Lukacs (1989) and calculus was recorded according to the method provided by Brothwell (1981).

Supplementary Figures

Individual	*Sex*	*Age*	*Bone*	*F ppm*	*Vitamin D deficiency*	*Atlanto-occipital membrane ossification*	*Miscellaneous ossification*	*Ante-mortem fractures*	*Joint or other ankylosis*	*Ossification of ligamentum flavum*	*Dental Changes*	*Entheseal calcification*	*Interosseous calcification*	*Pathological changes*
SKR 4	M	OA	Rib	1750	A	A	P	A	A	P	P	P	P	Maxillary sinusitis and nodular bone formation throughout ribs; ossified thyroid; entheseal calcification to deltoid tuberosity, teres major, pronator teres, and more; linear enamel hypoplasia, ante-mortem chipping; calcification of tibiae and fibulae interosseous crests
SKR 10	F	OA	Rib	2510	A	A	A	P	A	A	A	A	A	Nodular bone formation on ribs; healed fractures to right 9th and 10th ribs, lateral end of right clavicle, right calcaneus

SKR 14	NA	NA	Rib	2520	A	A	P	A	A	A	P	A	P	Non-adult (adolescent). hydatid cysts; nodular bone formation on ribs; left distal tibia with exostosis; linear enamel hypoplasia; calcification of tibiae and fibulae interosseous crests
SKR 22	NA	NA	Rib	468	A	A	A	P	A	A	P	A	A	Non-adult (adolescent). cleft palate and premaxilla; Incomplete fracture of tibial diaðhysis and tuberosity; linear enamel hypoplasia, enamel pitting
SKR 23	F	YA	Rib	223	A	P	A	A	A	A	A	A	A	Osteitis (long bones); cribra orbitalia
SKR 29	F	YA	Rib	2140	A	P	A	P	A	A	P	A	U	Cribra orbitalia; maxillary sinusitis; unidentified right rib body with healed fracture callous; enamel pitting
SKR 30	F	OA	Rib	525	P	A	A	P	A	A	U	A	A	Cribra orbitalia; maxillary sinusitis; nodular bone formation on ribs; ribs with pronounced curvature and loss of trabecular density; vertebral compression fractures with evidence of decreased trabecular density (osteoporosis?)
SKR 33	F	OA	Rib	1830	A	A	A	P	A	A	P	P	A	1st right rib with pseudojoint at vertebral aspect of shaft; mixture of vascular impressions and woven and lamellar new bone formation throughout ribs; unidentified left rib with well healed fracture at the midshaft; entheseal calcification to deltoid tuberosity, posterior iliac crests, ischia; Linear enamel hypoplasia, ante-mortem chipping
SKR 46	NA	NA	Rib	1110	A	A	A	A	A	A	P	A	A	Non-adult (adolescent). cribra orbitalia; woven bone on right 4th rib and nodular bone formation throughout ribs; atlas bilateral hypoplasia of neural arch; linear enamel hypoplasia, enamel hypomineralisation/mottling, ante-mortem chipping
SKR 65	F	YA	Rib	1690	A	P	A	A	A	P	P	P	P	Maxillary sinusitis; nodular bone formation on ribs; large arachnoid granulations; calcification of radius and ulna interosseous crests; entheseal calcification of deltoid tuberosity, linea aspera, soleal line, posterior iliac crest; Linear enamel hypoplasia, enamel hypomineralisation/mottling, hypercementosis and other dental changes
SKR 81	F	YA	Rib	2990	A	A	A	A	P	U	P	A	P	Right mandibular ramus and ulnae with osteitis; maxillary sinusitis; right tibia is partly ankylosed with the right fibula; ante-mortem dentalchipping; calcification of right tibia and fibula interosseous crests
SKR 91	M	YA	Rib	655	A	P	P	A	A	P	P	P	P	Maxillary sinusitis; posterior aspects of thoracic and lumbar supeiror vertebral facets; linear enamel hypoplasia, enamel hypomineralisation/mottling, ante-mortem chipping; entheseal calcification of deltoid tuberosity, linea aspera, soleal line, pronator teres and others; calcification of tibiae, fibulae, radii, ulnae interosseous crests
SKR 100	M	YA	Rib	4340	A	P	A	A	A	U	P	U	A	Cribra orbitalia; osteitis and anterior bowing of ulnae, fibulae and tibiae; linear enamel hypoplasia enamel hypomineralisation/mottling, ante-mortem chipping
SKR 115	M	OA	Rib	3080	A	U	A	A	A	U	P	U	U	Linear enamel hypoplasia
SKR 122	F	YA	Parietal	2290	A	A	A	A	A	U	P	U	U	Cribra orbitalia; linear enamel hypoplasia
SKR 126	F	OA	Rib	3690	A	A	A	P	P	A	P	P	A	Maxillary sinusitis; hydatid cysts; healed fracture with bone callous on right 1st and 2nd hand phalanges; right 1st and 2nd hand phalanges ankylosed (due to fracture?); linear enamel hypoplasia, enamel hypomineralisation/mottling, enamel pitting; entheseal calcification to delthoid tuberosity, soleal line, teres major and others
SKR 128	F	OA	Rib	3190	A	A	A	P	A	A	P	P	A	Kyphosis of spine; frontal bones with button osteomata; numerous vertebral compressions fractures; healed cranial trauma? (circular, concave lesion with reactive new bone); linear enamel hypoplasia; entheseal calcification of deltoid tuberosity, linea aspera and others
SKR 130	M	OA	Rib	2240	A	A	A	P	A	A	P	P	P	Tibiae with osteitis; pronounced lingual wear on the maxillary incisors and canines; mandibular teeth worn to roots; nodular bone formation throughout ribs; distal right femur fractured; calcification of radii and ulnae interosseous crests; ante-mortem chipping; entheseal calcification to linea aspera, teres major and others
SKR 135	M	YA	Clavicle	4370	P	P	A	A	A	U	P	P	P	Femora with anterior-posterior bowing; fibulae with medial-lateral bowing; calcification of tibiae and fibulae interosseous crests; linear enamel hypoplasia, enamel hypomineralisation/mottling, ante-mortem chipping; entheseal calcification and changes to teres major, pronator teres, deltoid tuberosity (severe, causing cavitations) and others
SKR 144	F	OA	Rib	1890	A	U	A	A	A	U	U	P	A	None; entheseal calcification of linea aspera
SKR 146	NA	NA	Rib	1040	U	U	U	U	U	U	U	U	U	Non-adult (neonate). Skeletal changes were not assessed due to age.
SKR 150	M	YA	Rib	3440	A	P	P	A	P	U	P	P	A	Right tibia with osteitis; Stylohyoid chain (Eagle's syndrome) ossified; left tibia ankylosed with fibula; linear enamel hypoplasia, enamel hypomineralisation/mottling, ante-mortem chipping; entheseal calcification to linea aspera (left with reactive activity: porous, striated bone deposition on entire length of attachment site)
SKR 152	M	YA	Rib	3050	A	A	A	A	A	A	P	A	A	Two rib fragments with nodular bone formation; linear enamel hypoplasia and ante-mortem chipping
SKR 155	M	OA	Rib	1770	A	P	A	P	P	A	P	P	P	Hydatid cysts; sacralisation; nodular bone formation throughout ribs; left hyoid cornua with healed fracture; left radius and ulna partially ankylosed through remodelling woven bone formation; calcification of right radius and ulna interosseous crests; hypercementosis, ante-mortem chipping, remodelling fracture of L 1st premolar root; entheseal calcification to the posterior iliac crests and other (pronounced change on retroauricular surface of ilia)
SKR 163	NA	NA	Rib	1510	U	U	U	U	U	U	U	U	U	Non-adult (neonate). Skeletal changes were not assessed due to age.
SKR 167	M	YA	Rib	1970	A	P	A	A	A	A	A	A	A	White, chalky biological concretions adhered to three right ribs (hydatic cysts?)
SKR 169	F	OA	Rib	1810	A	A	P	A	A	P	P	P	P	Left fibula shaft with osteitis; maxillary sinusitis; posterior aspects of all vertebral facets with osteosclerosis; calcification of left tibia and fibula interosseous crests; ante-mortem chipping; entheseal calcification to deltoid tuberosity, linea aspera, soleal line and numerous other pronounced entheseal changes
SKR 172	M	OA	Rib	2070	A	P	A	A	A	A	P	P	P	White, chalky biological concretions adhered to three right ribs (hydatic cysts?); diffuse active and remodelling porosity on palatine surface and ectocranium; calcification of radii and ulnae interosseous crests; linear enamel hypoplasia, hypercementosis, enamel hypomineralisation/mottling, retained dental roots; entheseal calcification to teres major
SKR 174	M	OA	Femur	2120	A	A	A	A	A	U	A	P	A	Cribra orbitalia; entheseal calcification to deltoid tuberosity, teres major, pronator teres and others

SKR 181	F	OA	Temporal	3620	U	U	U	P	A	U	P	U	U	Mandibular molars with lingual wear; necrotic lesion on frontal (reaction to trauma?); ante-mortem chipping
SKR 189	F	YA	Rib	3450	A	A	A	A	A	U	P	P	P	Hydatid cyst; calcification of radii interossesous crests; linear enamel hypoplasia, enamel pitting, ante-mortem chipping; entheseal calcifcation to soleal line
SKR 195	F	YA	Rib	3640	A	P	A	A	A	A	P	P	A	Left tibia shaft with osteitis; maxillary sinusitis; nodular bone formation throughout ribs; linear enamel hypoplasia, enamel pitting, ante-mortem chipping; entheseal calcification to deltoid tuberosity and pronator teres
SKR 201	F	YA	Rib	3290	A	U	A	A	A	U	P	P	A	Maxillary sinusitis; prominent spaces between mandibular teeth; retained deciduous teeth; differential dental wear; linear enamel hypoplasia, enamel hypomineralisation/mottling, ante-mortem chipping; entheseal calcification to deltoid tuberosity and soleal line
SKR 221	NA	NA	Rib	3180	A	A	A	A	U	U	P	A	A	Non-adult (adolescent); linear enamel hypoplasia
SKR 236	F	OA	Rib	1980	A	A	A	P	A	A	A	P	P	Nodular bone formation on ribs; spondylolisis L4 and L5 with non-united pseudoarthroses; calcification of tibiae and fibulae interosseous crests; entheseal calcification to deltoid tuberosity, linea aspera, posterior iliac crests, pronator teres and others
SKR 241	F	OA	Rib	2230	A	A	P	A	P	P	P	P	A	Hydatid cyst; cleft neural arch in C1; nodular bone formations on some rib fragments; 1st ribs with ossified cartilage (>15mm); manubrium fused to sternum; C2 to C3 ankylosed; linear enamel hypoplasia and ante-mortem chipping; entheseal changes to deltoid tuberosity, linea apsera, ischia, teres major and others
ÞSK 3	F	OA	Rib	660	P	A	A	P	A	A	P	P	P	Cribra orbitalia; nodular bone formation throughout ribs; atrophy of right arm bones (paralysis?); right ulna and radius with curvature and shortening; distal end of ulna malformed; healed coccyx fracture; calcification of radii and ulnae interosseous crests; linear enamel hypoplasia; entheseal calcification to 5th metacarpal (other)
ÞSK 4	F	YA	Rib	3030	A	A	A	P	A	A	P	A	A	Cribra orbitalia; nodular bone formations on 3 ribs; incomplete medial cleft premaxilla; bone spicule extending from intraconal aspect of right orbit; deviated septum; healed rib fracture; linear enamel hypoplasia
ÞSK 16	F	OA	Rib	1200	A	P	A	P	A	A	P	P	A	Cribra orbitalia; maxillary sinusitis; nodular bone formations throughout ribs; right maxillary lateral incisor with lingual wear; extensive porosity on palatine surfaces and across entire anterior maxilla; pronounced oblique fracture of distal 1/3 of tibial shaft (evidence of both active infection and remodelling); linear enamel hypoplasia; entheseal calcification to posterior iliac crests, other (palmar surfaces of all proximal and intermediate hand phalanges, right patella with entheseophyte (>7mm), superior and costal margins of the ribs)
ÞSK 17	F	OA	Rib	223	A	A	P	A	A	A	P	P	P	Cribra orbitalia; maxillary sinusitis; nodular bone formations throughout ribs; posterior aspects of lumbar facets and bodies; calcification of tibiae, fibulae, radii and ulnae interosseous crests; Ante-mortem chipping; entheseal calcification to deltoid tuberosity, linea aspera, posterior iliac crests and pronator teres
ÞSK 29	M	OA	Rib	1320	A	A	P	P	P	A	A	P	P	Nodular bone formation throughout ribs; xiphoid process, rib heads, sternal rib ends, manubrium and sternum with pronounced ossification; DISH (T12-L5); healed compression fracture of the left 7th thoracic vertebra; tibiae ankylosed to fibulae; T12-L1 fracture of left scapula; calcification of tibiae and fibulae interosseous crests; entheseal calcification to linea aspera, soleal line, posterior iliac crests, ischia and others
ÞSK 32	M	YA	Rib	396	A	A	A	P	A	A	P	P	P	Vascular impressions and nodular and woven bone formations throughout ribs; compression fracture of L4 resulting in wedge shaped vertebra (and spinal kyphosis); healed fracture at distal 1/2 of right 5th metacarpal; calcification of radii and ulnae interosseous crests; linear enamel hypoplasia; entheseal calcification to soleal line, posterior iliac crests, pronator teres and others
ÞSK 34	M	YA	Rib	1510	P	A	A	A	P	A	P	P	A	Healed osteomyelitis of left tibia; cleft neural arch ins S4-S5; antero-medial bowing of both femoral shafts (right is more pronounced); sacrum fused to auricular surface; right acetabulum fused to femoral head; left joints with extensive bone formation but not ankylosis; linear enamel hypoplasia, enamel hypomineralisation/mottling; entheseal calcification to deltoid tuberosity and others (clavicle with entheseal change at deltoid attachment, rhomboid fossae, conoid tubercle)
ÞSK 37	M	OA	Rib	1840	A	P	A	A	A	A	P	P	P	Nodular bone formation throughout ribs; bilateral hypoplasia vertebral arch of C1; calcification of tibiae and fibulae interosseous crests; Ante-mortem chipping; entheseal calcification to deltoid tuberosity, linea aspera, soleal line, teres major, pronator teres and others (numerous)
ÞSK 41b	M	OA	Rib	233	A	A	A	A	A	A	P	P	P	Cribra orbitalia; maxillary sinusitis; porotic hyperostosis; nodular bone formation throughout ribs; calcification of fibulae interosseous crests; linear enamel hypoplasia, enamel hypomineralisation/mottling; entheseal calcification to linea aspera, posterior iliac crests, ischia, teres major, pronator teres and others
ÞSK 42	M	OA	Rib	1190	A	A	A	A	A	P	P	P	A	Hyperostosis frontalis interna; porotic hyperostosis; cribra orbitalia; maxillary sinusitis; nodular bone formations throughout ribs; cleft neural arch S1 and S5; developmental pit on left distal radius joint surface; linear enamel hypoplasia, enamel hypomineralisation/mottling; entheseal calcification to deltoid tuberosity, soleal line, posterior iliac crests, teres major and others
ÞSK 44	M	OA	Os coxa	3010	A	P	A	P	A	A	P	P	P	Maxillary sinusitis; partial cleft neural arch in S4 (left); left posterior parietal with healed blunt force trauma; osteochondritis dissecans on distal right tibia; calcification of tibiae and fibulae interosseous crests; Severe enamel hypomineralisation/mottling; entheseal calcification to deltoid tuberosity, linea aspera, soleal line, teres major, posterior iliac crests and others
ÞSK 48	M	OA	Parietal	1850	A	A	A	A	A	A	P	P	P	Cribra orbitalia; developmental pit on left distal humerus; lumbarisation of S1 and pseudoarthrosis with L5; calcification of right ulna interosseous crest; linear enamel hypoplasia, enamel pitting, enamel hypomineralisation/mottling; entheseal calcification to linea aspera, soleal line, teres major, pronator teres and others
ÞSK 51	F	OA	Rib	503	P	A	A	P	A	A	P	A	A	Cribra orbitalia; maxillary sinusitis; nodular bone formation throughout ribs; Right fibula prominently bowed with central shaft taking a convex shpe laterally (left unobservable due to post-mortem fracture); fracture of two unsided intermediate hand phalanges, left femur and one unsided proximal hand phalanx; linear enamel hypoplasia, enamel hypomineralisation/mottling
ÞSK 54	M	YA	Rib	2160	A	A	A	A	A	U	P	P	A	Enlarged nutrient foramina on right ulna; lingual dental wear in central incisors (almost all lingual enamel obliterated); linear enamel hypoplasia, ante-mortem chipping; entheseal calcification to linea aspera, posterior iliac crests, ischia, pronator teres and others

Key: SKR = Skriðuklaustur, ÞSK = Skeljastaðir; NA = Non-adult, M = Male, F = Female; Non-adult (<17), Younger adult (17-36), Older adult (36+); P = Present, A = Absent, U = Unobservable

Supplementary Table 1 Fluoride bone concentrations for each sampled individual from Skriðuklaustur and Skeljastaðir and corresponding palaeopathological descriptions