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Ocena osiągnięć naukowych

Dr Anny Myszki

adiunkta w Instytutucie Nauk Biolgicznych Uniwersytetu Kardynała Stefana Wyszyńskiego w Warszawie

ubiegającej się o nadanie stopnia doktora habilitowanego w dziedzinie nauk ścisłych i przyrodniczych w dyscyplinie nauki biologiczne

Podstawa prawna i ocena formalna

Ustawa z dnia 20 lipca 2018 r. Prawo o szkolnictwie wyższym i nauce (Dz. U. 2022 r. poz 574, Uchwała Nr 200/2019 Senatu Uniwersytetu Kardynała Stefana Wyszyńskiego w Warszawie z dnia 23 października 2019 r. i Uchwała Rady Dyscypliny Nauk Biologicznych 10/2023 r. w sprawie powołania komisji habilitacyjnej w postępowaniu o nadanie stopnia doktora habilitowanego dr Annie Myszce.

W szczególności odnosiłem się do literalnego brzmienia art. 219 obecnej wersji Prawa o szkolnictwie wyższym (Dz. U. z 2022 r. poz. 574), którego brzmienie kopiuję poniżej:

Art. 219. *1. Stopień doktora habilitowanego nadaje się osobie, która:*

1) posiada stopień doktora;

2) posiada w dorobku osiągnięcia naukowe albo artystyczne, stanowiące znaczny wkład w rozwój określonej dyscypliny, w tym co najmniej:

a) 1 monografię naukową wydaną przez wydawnictwo, które w roku opublikowania monografii w ostatecznej formie było ujęte w wykazie sporządzonym zgodnie z przepisami wydanymi na podstawie art. 267 ust. 2 pkt 2 lit. a, lub

b) 1 cykl powiązanych tematycznie artykułów naukowych opublikowanych w czasopismach naukowych lub w recenzowanych materiałach z konferencji międzynarodowych, które w roku opublikowania artykułu w ostatecznej formie były ujęte w wykazie sporządzonym zgodnie z przepisami wydanymi na podstawie art. 267 ust. 2 pkt 2 lit. b, lub

c) 1 zrealizowane oryginalne osiągnięcie projektowe, konstrukcyjne, technologiczne lub artystyczne;

3) wykazuje się istotną aktywnością naukową albo artystyczną realizowaną w więcej niż jednej uczelni, instytucji naukowej lub instytucji kultury, w szczególności zagranicznej.

2. Osiągnięcie, o którym mowa w ust. 1 pkt 2, może stanowić część pracy zbiorowej, jeżeli opracowanie wydzielonego zagadnienia jest indywidualnym wkładem osoby ubiegającej się o stopień doktora habilitowanego.

Odniosłem się także do art. 3. 2 i do art. 128, 1 Ustawy:

Art. 3.

2. System szkolnictwa wyższego i nauki funkcjonuje z poszanowaniem standardów międzynarodowych, zasad etycznych i dobrych praktyk w zakresie kształcenia i działalności naukowej oraz z uwzględnieniem szczególnego znaczenia społecznej odpowiedzialności nauki.

Art. 128.

1. Nauczyciel akademicki, ... podlega ocenie ... , w szczególności w zakresie wykonywania obowiązków, o których mowa w art. 115, oraz przestrzegania przepisów o prawie autorskim i prawach pokrewnych

Oceny osiągnięcia naukowego pt. „Zmiany zwyrodnieniowe stawów w dawnych populacjach szkieletowych – problemy badawcze i interpretacyjne, perspektywy badawcze" oraz aktywności naukowej dr Anny Myszki dokonałem na podstawie przesłanych mi przez Przewodniczącego Rady Dyscypliny Nauki Biologiczne UKSW Dyrektora Instytutu Nauk Biologicznych UKSW prof. dr hab. Jacka Tomczyka dokumentów.

Uważam, że przekazana mi dokumentacja związana z postępowaniem habilitacyjnym dr Myszki jest pełna i przygotowana zgodnie z obowiązującymi obecnie przepisami i zaleceniami.

<u>Opinia</u>

1. Stwierdzam, że zgodnie z art. 219, 1 Anna Myszka posiada stopień doktora nauk biologicznych w zakresie biologii uzyskany w 2006 r. na Wydziale Biologii Uniwersytetu im. Adama Mickiewicza w Poznaniu. Tytuł: „*Rekonstrukcja budowy somatycznej człowieka na podstawie wybranych cech szkieletu*".

2. Dr Myszka przedstawiła jako swoje osiągnięcie naukowe stanowiące znaczny wkład w rozwój dyscypliny „nauki biologiczne" zestaw czterech współautorskich prac opublikowanych w latach 2019-2022 w czasopismach międzynarodowych o umiarkowanym zasięgu (IF 0,877-2.064, MEiN2020 40-100 punktów). We wszystkich czterech pracach, Dr Myszka jest pierwszym autorem podczas gdy jednym ze współautorów jest jej obecny przełożony, prof. dr hab. Jacek Tomczyk. W odniesieniu do wszystkich czterech prac dr Myszka tak charakteryzuje jej wkład w ich powstanie: „*Mój wkład w powstanie tej pracy polegał na przygotowaniu koncepcji badań, przeprowadzeniu badań laboratoryjnych materiału kostnego, udziale w analizie wyników, interpretacji wyników,przygotowaniu pierwszej wersji manuskryptu, który był następnie recenzowany przez pozostałych*

współautorów". Wynika z tego, że w zasadzie wszystkie prace wchodzące w skład jej osiągnięcia naukowego są wynikiem jej oryginalnej pracy naukowej podczas gdy wkład współautorów był minimalny. To stwierdzenie stawia pod znakiem zapytania etykę zawodową autorów przedstawionych prac gdyż kryteria autorstwa prac naukowych wymagają istotnego wkładu intelektualnego w sformułowanie tematyki pracy, wykonanie badań lub ich twórczą interpretację. Zacytuję tu jedną z wielu definicji autorstwa sformułowaną przez redakcję NATURE:
"Each author is expected to have made substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data; or the creation of new software used in the work; or have drafted the work or substantively revised it".

Od ćwierćwiecza byłem i jestem Editor-in-Chief, lub członkiem rad wydawniczych (Member of the Editorial Board) międzynarodowych czasopism wydawanych przez Elsevier i Springer Nature, jak również głównego polskiego czasopisma w specjalności dr Myszki (Przegląd Antropologiczny/Anthropological Review) gdzie używa się kryteriów autorstwa podobnych do zacytowanych powyżej. W przypadku dr Myszki istnieją dwie interpretacje jej oświadczenia o autorstwie prac wchodzących w skład „osiągnięcia" – albo przesadza ona swój wkład w powstanie przedstawionych czterech prac, albo jej współautorzy postąpili nieetycznie dopisując swoje nazwiska do listy autorów. Tak czy inaczej, postępowanie dr Myszki jest kwestionowalne etycznie bo, co najmniej, zgadzała się na nieuzasadnione „dopisywanie" współautorów do jej prac.

Dr Myszka słusznie zidentyfikowała generalny problem badawczy, któremu jest poświęcone jej „osiągnięcie" – brak zrozumienia przyczyn zmian zwyrodnieniowych stawów pomiędzy ludzkimi kośćmi, a co za tym idzie, brak możliwości interpretacji waruków życia i chorób dawniejszych ludzi na podstawie obserwacji materiałów szkieletowych. Celem przyczynienia się do rozwiązania tego problemu, przeprowadziła ona szereg obserwacji występowania takich zmian w zbiorach szczątków szkieletowych pochodzących z kilku dawnych populacji z terenu Polski datowanych od wczesnego średniowiecza do wczesnych czasów nowożytnych. Liczebność jej materiałów jest wystarczająca do wyciągania wniosków z analiz statystycznych, dane zostały zebrane standardowymi metodami uznanymi międzynarodowo i, w większości przypadków, poprawnie zanalizowane statystycznie. W pracy 01 dr Myszka stwierdziła pewien związek artretyzmu z natężeniem urzeźbienia miejsc przyczepu mięśni do kości wyciągając wniosek o wpływie aktywności ruchowej na pojawianie się zmian zwyrodnieniowych stawów. Badając w pracy 02 związki pomiędzy trzema objawami patologicznymi zmian zwyrodnieniowych stawów – wyroślami kostnymi, porowatością powierzchni stawowych i wyszlifowaniem powierzchni stawowych – autorzy stwierdzili brak wyraźnych związków pomiędzy nimi z wyjątkiem korelacji (rho Spearmana) pomiędzy wyroślami kostnymi i porowatością powierzchni stawów. Korelacja ta, choć formalnie istotna statystycznie, nie przekracza wartości rho 0,370, co oznacza, że zmienność występowania wyrośli wyjaśnia nie więcej niż 14 procent występowania porowatości. Jest to związek słaby. Ten wynik, jakkolwiek uzyskany na podstawie poprawnie przeprowadzonych obserwacji nie wzbogaca wiedzy a tylko potwierdza to co od dawna wiadomo. Przypominam sobie, że gdy w roku 1972 uczyłem się paleopatologii od śp profesora Michała Ćwirko-Godyckiego, praktykującego lekarza habilitowanego do antropologii, mówił on nam o istnieniu tego właśnie związku.
Praca 03 przedstawia wyniki badań występowania zmian artretycznych w materiałach szkieletowych odkopanych w Radomiu i datowanych od XIV do XIX wieku. Dr Myszka i

jej współautorzy wykazali brak zmian częstości występowania zwyrodnieniowych zmian stawów w obserwowanym przedziale chronologicznym. Autorzy wyciągnęli stąd wniosek, że obserwowane zmiany zwyrodnieniowe nie są dobrym wskaźnikiem statusu społeczno-ekonomicznego czy generalnego stanu zdrowia dawnych populacji. Jest to wniosek słuszny i wartościowy. Często, i niesłusznie, uważa się, że wynik „negatywny" (np. brak zmian w czasie, lub różnic pomiędzy grupami osobników) jest mało wartościowy w literaturze naukowej, podczas gdy takie wyniki falsyfikują niepoprawne interpretacje. Wystarczy wspomnieć stwierdzenie braku różnic we współczynnikach „inteligencji" mężczyzn i kobiet. Wynik uzyskany w badaniu materiałów szkieletowych z Radomia może, na przykład, wskazywać, że pewna część przypadków artretyzmu ma podłoże genetyczne o czym autorzy wspominają też w innych ich pracach.
Praca 04 testuje interesującą hipotezę określaną jako „bone former". Ta hipoteza stanowi, że istnieje zróżnicowanie gnetyczne tendencji do wytwarzania dodatkowej tkanki kostnej u osób dorosłych (patrz wyżej). Autorzy testują tę hipotezę korelując występowanie wyrośli kostnych na kręgosłupie z ich występowaniem na innych częściach szkieletu używając obserwacji zebranych na materiale szkieletowym z Łękna, częściowo zanalizowanych już do pracy przedstawionej jako 01. Chociaż zastosowali nieprawidłową metodę badania korelacji pomiędzy cechami mierzonymi w kategoriach, uzyskali wyniki wskazujące na istotne współzależności potwierdzające hipotezę „bone former". Korelacja rang Spearmana, którą zastosowali autorzy, może być stosowana tylko do monotonicznych relacji pomiędzy zmiennymi mierzonymi w sposób ciągły (skala przedziałowa [intrwałowa]) lub zmiennymi mierzonymi w skalach porządkowych (ordinal scales), a nie w skalach kategorycznych.

Dr Myszka tak podsumowała zawartość czterech publikacji przedstawionych jako „osiągnięcie naukowe":

„Wybrane przeze mnie prace, ..., uważam za istotne, gdyż: (a) uzupełniają wiedzę na temat zmian zwyrodnieniowych stawów w badaniach szkieletowych; (b) zbierają, porządkują i systematyzują dotychczasową wiedzę, poglądy badaczy na temat tych tak powszechnych, a rodzących szereg problemów, zmian patologicznych kośćca;(c) uzupełniają dotychczasowe wyniki badań."

Jakkolwiek zgadzam się z tym podsumowaniem, nie jestem pewien czy uzupełnianie dotychczasowych wyników badań w sposób istotny zmienia stan naszej wiedzy na temat zmian zwyrodnieniowych stawów. Nadal wiemy, że większość ich przyczyn pozostaje nieznana i że nie możemy obserwacji tych zmian w szczególnych zbiorach materiałów szkieletowych stosować jako wskaźników stanu zdrowia czy warunków społeczno-ekonomicznych dawnych grup ludzkich. Dr Myszka wykazała, że przy współpracy szeregu współautorów, potrafi poprawnie zastosować istniejące już metody badawcze do kilku zbiorów materiałów szkieletowych reprezentujących dawne populacje i zinterpretować wyniki tak zebranych obserwacji na tle danych i interpretacji z literatury.

Jako biologa razi mnie używanie w przedstawionych publikacjach zwrotu „populacje szkieletowe". Zwrot ten jest używany w niektórych innych publikacjach międzynarodowych, ale ten fakt nie stanowi uzasadnienia by go bezkrytycznie używać. W moim rozumieniu „populacja" w sensie biologicznym to zespół osobników zdolny do samodzielnej reprodukcji, lub w demografii wszyscy mieszkańcy jakiegoś kraju, regionu, miasta.... W statystyce „populacja" to wszystkie obiekty o szczególnych badanych cechach, a nie ograniczona liczebnie próba poddawana analizie. Na tle tych definicji „populacja szkieletowa" to

antylogia, bo szkielety nie mogą się rozmnażać, a kiedy bada się określoną liczbę szkieletów, trudno odnosić wyniki takiego badania do wszystkich szkieletów istniejących gdziekolwiek. Bezkrytyczne stosowanie zwrotów pojawiających się w literaturze nie świadczy dobrze o samodzielności intelektualnej.

3. Dr Anna Myszka prowadziła i prowadzi istotną aktywność naukową w więcej niż jednej uczelni lub instytucji naukowej. Ta aktywność jest udokumentowana szeregiem publikacji (D1-D23 i DM1-DM7), wystąpień na konferencjach, udziałem w popularyzacji wiedzy i prowadzeniem zajęć dydaktycznych na uczelniach innych niż ta gdzie jest zatrudniona na pełnym etacie (np. w Uniwersytecie Medycznym im Karola Marcinkowskiego w Poznaniu). Dr Myszka, poza analizami materiałów szkieletowych zgromadzonych przez innych badaczy prowadzi również własne badania wykopaliskowe dostarczające nowe materiały szkieletowe. Jej współpraca naukowa obejmuje uczelnie w USA, Czechach i na Litwie oraz szereg instytucji polskich, że wymienię tylko uniwersytety: Jagielloński, Łódzki, Warszawski i Poznański Medyczny, Przyrodniczy w Poznaniu, Szkołę Główną Gospodarki Wiejskiej, instytuty Polskiej Akademii Nauk i muzea: Pierwszych Piastów na Lednicy i Ziemi Dobrzyńskiej w Rypinie.

Tematyka opublikowanych prac dr Myszki jest szersza niż tematyka jej „osiągnięcia" i obejmuje rekonstrukcje budowy ciała na podstawie cech szkieletu, cechy niemetryczne (tzw cechy epigenetyczne) szkieletów, stan uzębienia w przeszłości, urazy kości czaszki, jak również prace z zakresu wirusologii. Szczególnie te ostatnie wskazują na umiejętność dr Myszki znacznego rozszerzania zakresu zainteresowań naukowych. Poniżej omawiam pokrótce kilka jej publikacji, które zwróciły moją szczególną uwagę:

DM1 to monografia zawierająca bardzo dobry przegląd literatury dotyczącej rekonstrukcji budowy somatycznej człowieka na podstawie cech szkieletów a jej wyniki stanowią wartościowy wkład naukowy porównywalny, a może nawet przekraczający, wartość obecnego „osiągnięcia". Jest to publikacja rozprawy doktorskiej.

D21 praca wprowadza oryginalną klasyfikację uformowania miejsc przyczepów mięśni do kości

D15 analizuje otwór nadkłykciowy kości ramiennej jako cechę patologiczną podczas gdy ten otwór jest często uważany za normalny wariant anatomiczny

D10 sugeruje, że słaby mięsień trójgłowy ramienia może powodować nadmierne wyprostowanie (hyperxtension) stawu łokciowego. Wydaje się, że ten mięsień, jako prostownik stawu łokciowego, powinien być szczególnie silny by powodować nadmierne wyprostowanie podczas gdy słaby antagonista prostownika tego stawu – mięsień dwugłowy ramienia – mógłby być przyczyną hyperekstensji.

D3 dobry przegląd literatury

D8 rozległy autoplagiat pracy O1. Załączam kopię O1 gdzie zaznaczyłem identyczny z D8 tekst i tabele. Ponieważ listy autorów obu prac nie są identyczne istnieje możliwość uznania tej sytuacji za plagiat. Szczegóły omawiam osobno poniżej.

D17 – D20 autoplagiat wstępu

D15 – D16 autoplagiat wstępu i części streszczenia.

Naruszenie zasad etyki zawodowej i prawa autorskiego

W kilku pracach dr Myszki pojawiają się fragmenty tekstów skopiowanych *verbatim* z innych jej prac (patrz wyżej). Jakkolwiek nieetyczne, postępowanie takie może być wytłumaczalne wymaganiem używania tekstu w języku angielskim, którego znajomość przez autorów może być niekompletna i używaniem w kolejnych pracach tych samych materiałów, lub metod. Wśród prac, których pierwszym autorem jest dr Myszka, są jednak dwie prace, które poza splagiaryzowaniem obszernych części tekstu i wielu tabel, naruszają prawa autorskie wydawnictw, które je opublikowały i współautorów. W szczególności chodzi tu o :

O1. Myszka A., Krenz Niedbała M., Tomczyk J., Zalewska M. 2020. Osteoarthritis: A problematic disease in past human populations. A dependence between entheseal changes, body size, age, sex, and osteoarthritic changes development. The Anatomical Record 303 (9), 2357-2371. DOI: 10.1002/ar.24316. Epub 2019 Nov 18. © 2019 American Association for Anatomy, Paper received 22 July 2019. Accepted 23 September 2019

D8. Myszka A., Piontek J., Tomczyk J., Zalewska M. 2020. Osteoarthritis–a problematic skeletal trait in past human populations. Osteoarthritic changes vs. entheseal changes in the late medieval and early modern population form Łekno. Anthropological Review 83 (2), 143-161. © 2020 Polish Anthropological Society, Paper received 06 April 2020, Accepted 23 April 2020

Obie prace przedstawiają wyniki otrzymane na postawie tych samych danych i prowadzące do tych samych wniosków, choć uzyskane innymi metodami statystycznymi.
Na załączonej kopii O1 zaznaczyłem tekst, który jest identyczny w obu pracach i takież tabele. Program, którego używałem do zaznaczania PDF niedobrze radzi sobie z zaznaczaniem tabel, podaję więc tutaj numery tabel o identycznej zawartości w kolejności O1 – D8: T1=T1, T2=T2, T4=T3, T5=T4. W mojej ocenie 20 procent tekstu i 50 procent tabel jest w obu pracach identyczne. Oczywiście obie prace zawierają poza zaznaczonymi, fragmenty tekstu podobne do siebie choć nie identyczne.

Praca złożona później do druku (D8) nie cytuje wcześniejszej pracy (O1). Listy autorów obu prac nie są identyczne. Narusza to prawa autorskie pominiętego autora (M. Krenz-Niedbała). Choć polskie prawo nie uznaje autoplagiatu za oczywiste przestępstwo, to naruszenie praw autorskich American Association for Anatomy przez kopiowanie tekstu i tabel, które oni opublikowali, w Anthropological Review jest przestępstwem w świetle zarówno prawa polskiego jak i międzynarodowego. Autoplagiat jest uznawany za naruszenie zasad etyki środowiska naukowego. Złożenie pracy do druku w Anthropological Review zakłada, że składana praca jest oryginalnym tworem autorów i jej zawartość nie jest publikowana gdzie indziej. Jako jeden z redaktorów Anthropological Review zostałem oszukany przez autorów, do których, jako kolegów, miałem zaufanie. Porównanie dat przyjęcia do druku O1 i złożenia do druku D8 nie pozostawia wątpliwości, że autorzy D8 działali z premedytacją zatajając opublikowanie wcześniej w Anatomical Record istotnych części pracy składanej do druku w Anhropological Review.

Prawda jest najwyższą wartością w nauce. Postępowanie celowo fałszujące prawdziwy stan rzeczy dyskwalifikuje pracowników zajmujących etaty naukowe i naukowo-dydaktyczne, W tym przypadku to stwierdzenie odnosi się do wszystkich autorów pracy D8, nie tylko do dr Anny Myszki.

Biorąc pod uwagę opisany szczegółowo powyżej przypadek, jak również, wspomniane wcześniej, mniej drastyczne przypadki naruszania przez nią etyki publikacji naukowych, nie mam zaufania do prawdziwości przedstawianych przez dr Myszkę w jej pracach wyników i wniosków, a co za tym idzie do wartości jej dorobku naukowego. Mój brak zaufania odnosi się także do wartości jej przyszłej działalności akademickiej.

9 stycznia 2023 roku

Prof. dr hab. dr h.c. Maciej Henneberg, FRSB

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ARTICLE

WILEY

Osteoarthritis: A problematic disease in past human populations. A dependence between entheseal changes, body size, age, sex, and osteoarthritic changes development

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Abstract

Osteoarthritis is a problematic trait in terms of etiology and interpretation in past human populations. The relationships between osteoarthritic changes (osteophytes, porosity, and eburnation) and entheseal changes, body mass, stature, bone massiveness, sex, and age on the basis of skeletal material from Łekno (Poland) are analyzed here. Entheses were the strongest contributor to the prediction of osteophyte expression and when all types of changes and all joints were taken together. Stature demonstrates a negative dependence on porosity. When each joint was analyzed separately, entheses were the strongest contributor to the prediction of arthritis expression in the wrist and hip. Age was the strongest contributor to the prediction of arthritis expression in the elbow. Body mass, stature, bone massiveness, and sex had no effect on osteoarthritic changes in any of the examined joints. The results of the present study suggest an important dependence between entheses and osteoarthritic changes. Other factors had little to no effect on differences in OA severity. These results do not dispel all doubts but enrich knowledge about the effect of etiological factors on osteoarthritic change formation. This knowledge is essential for proper, reliable interpretation of osteoarthritic changes in the context of past human biology, ecology, and behavior.

KEYWORDS
eburnation, entheses, multivariate statistics, osteophytes, porosity

1 | INTRODUCTION

Osteoarthritis (OA) is a ubiquitous pathological condition in skeletal populations (Calce, Kurki, Weston, & Gould, 2018; Lovejoy, Weber, Bazaliitskiy, Gorianova, & Savelyev, 2007; Weiss & Jurmain, 2007). It is also a common joint disorder observed today (Arden & Nevitt, 2006; Rothschild & Woods, 2012).

The etiology of OA is multifactorial (Felson, 2006; Gabay, Hall, Berenbaum, Henrotin, & Sanchez, 2008; Manek-Pelletier, 2004; Roach & Tilley, 2007; Teichtahl, Wluka, Proietto, & Cicuttini, 2005). Age, sex, obesity, genes, metabolic factors, articular cartilage nutrition, endocrine factors, bone density, overloading of the musculoskeletal system, joint injuries, joint infections, congenital defects, joint instability, congenital and/or developmental joint deformities, physical activity and occupation, or even muscle weakness are given as etiological factors (Anderson & Loeser, 2010; Arden & Nevitt, 2006; Felson, 2006; Gabay et al., 2008; Tachdjian et al., 2002).

Despite its high incidence, OA is still a problematic trait in the context of analyses of past human populations. Although biological anthropologists have tried to use OA changes to describe the biology of past human groups, especially with regard to health status and habitual behavior

(Rothschild & Woods, 2012; Weiss & Jurmain, 2007; Zhang et al., 2017), its interpretation raises many doubts. The relationships between OA changes and some etiological factors (e.g., age, sex, body size, and physical activity) in skeletal populations are not unanimous, and in many cases, these data do not coincide with clinical views (Weiss & Jurmain, 2007).

OA is thought to be a classic age-related disorder (Anderson & Loeser, 2010). Taking contemporary data, OA is a progressive disease that affects 60% of males and 70% of females over the age of 65 (Sarzi-Puttini et al., 2005), as well as more than 30% of adults between 45 and 64 years of age (WHO, 2003). A strong dependence between age and OA is thought to be connected with biochemical changes in the cartilage that make it weaker and less resistant to biomechanical stress (Alexander, 1990). There is no such homogeneity in anthropological literature results regarding age differences in OA. An increase of OA changes with age was observed by Waldron (1991) in the Georgian and early Victorian London skeletal population (18th–19th), by Weiss (2006) in Californian Amerinds (500–1500 AD), by Molnar, Ahlstrom, and Leden (2011) in the Neolithic hunter–gatherer populations from Gotland (Sweden), and by Eng (2016) in Bronze and Iron Age skeletal collections from China and Mongolia. However, nonsignificant age differences in OA have also been observed by Palmer, Hoogland, and Waters-Rist (2016) in the post-medieval Dutch population. In studies by Woo and Pak (2013) for Joseon Dynasty population from Korea (15th–20th century), and Schrader (2012) for Tombos population (Nubia) of New Kingdom Period (1550–1069 BC) older individuals had greater OA changes, but the correlation was significant only for a few joints.

Sex is thought to be a joint specific risk factor (Hanna et al., 2009; McKean et al., 2007; Prieto-Alhambra et al., 2013; Srikanth et al., 2005). According to contemporary epidemiological data, OA has a higher prevalence in women than men, especially after the age of 50 (Felson, 2003). However, before this age, a higher prevalence has been observed in men, and after this age, a higher prevalence has been reported in women than men (Van Saase, Van Romunde, Cats, Vandenbroucke, & Valkenburg, 1989). These sex-related differences are linked to hormones (especially estrogen deficiency in the postmenopausal period) (Felson et al., 2000; Gokhale, Frenkel, & Dicesare, 2004; Mandl, 2007; Nevitt et al., 1995; Oliveria, Felson, Klein, Reed, & Walker, 1996; Zhang et al., 1998). In the majority of bioarchaeological studies, there is no homogeneity in sex differences with regard to the frequency and prevalence of OA. There are populations where males (or some joints in males) have higher OA scores than females. The results were obtained in hunter–gatherers and agriculturalists from the southeastern Alabama collection (Bridges, 1991), in medieval (10th–12th century) population from Stenjevec (Croatia) (Šlaus, 2002), in Californian Amerinds (500–1500 AD) (Weiss, 2006), in pre-Hispanic and post-contact collections from Peru (Klaus, Spencer Larsen, & Tam, 2009), or in Bronze and Iron Age populations from China and Mongolia (Eng, 2016). Although in Neolithic hunter–gatherers from Sweden (Molnar et al., 2011), Bronze and Iron Age skeletons from China and Mongolia (Eng, 2016) females (or some joints in females) had higher frequencies of OA than males. Some researchers did not find any significant or very small sex differences in OA. Such results were obtained for Holocene foragers of Siberia's Cis-Baikal region (Lieverse et al., 2007), Natufian hunter–gatherers and Neolithic farmers in the Levant (Eshed, Gopher, Galili, & Hershkovitz, 2004), for Tombos population (Nubia) (1550–1069 BC) (Schrader, 2012), for Joseon Dynasty collection from Korea (15th–20th century) (Woo & Pak, 2013), Archaic period (1000–500 BC) Ohio population (Woo & Sciulli, 2013), and for post-medieval Dutch (Palmer et al., 2016). As seen above, it can be assumed that age correlations and sex differences in OA changes in skeletal populations do not always exist, are not clear enough, are not homogeneous and are different for different skeletal populations and joints. This brings about a need for taking a look at these differences in other populations.

According to clinical data, obesity is considered one of the most important risk factors for OA (Felson et al., 2000). This relationship is more pronounced in women than in men and in weight-bearing joints, with a visibly higher prevalence in the knee (Anderson & Loeser, 2010; Arden & Nevitt, 2006). There are two theories explaining the effect of obesity on OA. The mechanical theory presupposes that increased mechanical loading on joints is connected with increasing body weight and is responsible for the development of OA (Grotle, Hagen, Natvig, Dahl, & Kvien, 2008; Teichtahl et al., 2005). The metabolic theory, in turn, postulates an effect of local hormones and biological mediators connected with obesity (e.g., leptins) on the development of OA (Dumond et al., 2003; Teichtahl et al., 2005). Past human population studies are scarce, and their results differ from the clinical data. Jurmain (1991) found a nonsignificant correlation between OA and reconstructed body mass. Interestingly, the relationship, even though nonsignificant, was negative (Jurmain, 1991). Significant and also negative (smaller and lighter individuals have greater OA scores) correlations between OA changes and reconstructed body mass were found in Amerind (3500–1500 BP) and Euroamerican (19th century) (Weiss, 2004) and in Californian Amerinds (500–1500 AD), (Weiss, 2006). These results that contradict current medical data and are not homogeneous for each joint indicate the need for further analyses of the effects of body mass on OA in past human groups. Further studies are necessary to check whether and how etiological factors influenced the formation of OA changes in past humans.

According to current medical knowledge, physical activity and occupation are given as etiological factors in OA

(Anderson & Loeser, 2010; Arden & Nevitt, 2006; Felson, 2003; Gabay et al., 2008; Teichtahl et al., 2005). Some data confirm that stronger muscle contraction forces increase joint loads and therefore increase the risk of the development of OA (Chaisson, Zhang, Sharma, Kannel, & Felson, 1999). However, there are studies showing opposite results, which is the protective role of strong muscles against OA, for example, strong quadriceps are associated with decreased OA progression (Sharma, Dunlop, Song, & Hayes, 2003; Slemenda et al., 1997, 1998).

A number of studies have examined the relationship between OA and entheseal changes (Rojas-Sepúlveda & Dutour, 2014; Schrader, 2012; Woo & Pak, 2013). Entheses define the area where a capsule, a tendon or a ligament attaches to bone and covers non-pathological changes in the attachment site (Villotte & Knüsel, 2013). Taking the assumption that bone tissue changes in response to environmental stress (biomechanical stimuli connected with physical activity) to protect itself against breakage (Ruff, Holt, & Trincaus, 2006; Schoenau & Frost, 2002) or to prevent a ligament/tendon rupture (Hawkey, 1998), entheseal changes are treated by some authors as physical activity markers (Eshed et al., 2004; Havelková, Villotte, Velemínský, Poláček, & Dobisíková, 2011; Hawkey & Merbs, 1995; Henderson & Cardoso, 2013; Henderson, Craps, Caffell, Millard, & Gowland, 2013).

Taking the above it was hypothesized that if positive relationships between OA and entheseal changes exist, it was possible that OA changes had a similar etiology as entheses. Therefore, they could be activity indicators, and they could be used in the reconstruction of activity patterns (Palmer et al., 2016; Rojas-Sepúlveda & Dutour, 2014; Schrader, 2012; Woo & Pak, 2013). There are studies where no significant relationship between OA changes and entheseal changes were found, such as in the Joseon Dynasty population from Korea (15th–20th century) (Woo & Pak, 2013) and in the Pre-Columbian skeletal collections from South America (Rojas-Sepúlveda & Dutour, 2014). These results suggested a different etiology for these two groups of skeletal traits (Rojas-Sepúlveda & Dutour, 2014; Woo & Pak, 2013). Not always existing, low or even negative correlations between OA and attachment sites were found by Schrader (2012) for the Tombos population of New Kingdom Period Nubia (1550–1069 BC) and Myszka (2015) for medieval/early modern collections from Poland. Henderson et al. (2013) who examined the value of the historical data according to occupational mobility in the 19th century rural England collection did not always found a correspondence between entheses and degenerative joint changes, indicating that other underlying factors affect the frequency of these skeletal features. Calce et al. (2018) examined the relationship between OA changes and femoral torsional strength (another proxy for measures of activity) in modern European skeletal samples and found no significant impact of activity on the development of OA, although the authors refer the negative dependence between pelvic OA and femoral torsional strength to the protective role of physical work capacity in childhood. As seen above, the previous study results are not homogenous; they do not speak clearly for or against the existence of a relationship between OA and entheses, indicating a need to continue investigating this relationship.

From clinical data from modern humans, individuals with OA generally have stronger body build, wider geometrical bone measurements, increased bone mineral density, and higher peak bone mass (Hunter & Spector, 2003; Roach & Tilley, 2007). High bone density and wider geometrical bone measurements seem to increase the risk of OA, especially with regard to osteophyte formation (Felson et al., 2000). It is hypothesized that tough, dense, stiffer subchondral bone is not capable of dissipating the biomechanical loading, which leads finally to cartilage failure and the appearance of OA changes (Dequeker, Boonen, Aerssens, & Westhovens, 1996; Hart et al., 2002; Hunter & Spector, 2003; Nevit, 2006; Sowers, 2001). Anthropological studies that examine the associations between OA and bone robusticity are rare and, in some cases, the results differ from those obtained for modern populations, where more massive bones are usually predisposed to OA (Brickley & Waldron, 1998; Weiss, 2013). A positive association between OA changes of metacarpal joints and metacarpal bone robusticity (more robust bones have higher OA) was assessed by Cope, Berryman, Martin, and Potts (2005) for a Bronze Age population from Tell Abraq (United Arab Emirates). Similar, but not always significant, results are obtained by Weiss (2013) for OA and metacarpal bone robusticity in pre-European contact California Amerinds (2180–250 BC) and by Weiss (2004) for humeral bone robusticity in British Columbians (3500–1500 BP) and Euroamericans (19th century). Calce et al. (2018), who examined modern European collections, observed that increased femoral robusticity was significantly correlated with lower pelvic OA scores. Analyses of the relationship between OA and bone robusticity in past populations are rare, so it is reasonable to continue them on other skeletal collections.

The effect of stature on OA changes is not well understood. The clinical literature focuses on the relationship between body mass index and OA, paying special attention to body mass increases, with less attention to the influence of body height (Marks & Allegrante, 2002; Reijman et al., 2007). However new research using biological markers to identify genetic variants associated with height has found a connection between short stature and susceptibility to OA (Sanna et al., 2008). Bioarchaeological data about the stature-OA connection are very rare. Calce et al. (2018) did not find an important role for stature in determining arthritic

patterning in modern collections from Portugal, Italy. For palaeopathologists, the relationship between overall body size and the mechanical stress threshold of joints may be specifically important factors to control for in evaluating joint failure and arthritic patterning in once living populations.

The purpose of this study is to examine the dependences between entheseal changes, body mass, stature, bone massiveness, sex, age, and OA changes (osteophytes [OP], porosity [POR], and eburnation [EB]) on the basis of the late medieval, early modern (14th–16th century) skeletal material from Łekno (Poland). Giving the fact that the etiology of OA is not completely understood, the results of bioarchaeological studies on the etiological factors influence the development of OA are not unanimous and, in many cases, are opposite to the clinical data on contemporary humans. Continued studies are important to better understand the occurrence and etiology of OA in past human populations and to integrate this knowledge into further understanding the human biology, ecology, and behaviors of these past human populations

2 | MATERIALS AND METHODS

Skeletal material used in this study came from the late medieval, early modern (14th–16th century) (Wyrwa, 1989) population from Łekno, Poland, in a collection of the Department of Human Evolutionary Anthropology, Adam Mickiewicz University in Poznań. The study sample consisted of 110 males and 56 females.

Only adult remains were included in this study. Standard anthropological methods were applied to determine the sex and age of the individuals (Buikstra & Ubelaker, 1994). Features of the cranium and pelvis were assessed for sex estimation. Age was estimated through the analysis of pubic symphysis changes. Age categories considered in the study were applied according to the following standards set by Buikstra and Ubelaker (1994): Young Adult (20–34 years), Middle Adult (35–49 years), and Old Adult (50+ years). The exact number of male and female individuals in each age category examined in this study is presented in Table 1. The group of skeletons examined here included individuals without any observable skeletal changes (illnesses, trauma, fractures, or bone deformities), except osteoarthritic changes.

Osteoarthritic changes were examined in accordance with the standard methods proposed by Buikstra and Ubelaker (1994). Three types of OA changes were examined: (a) OP; marginal proliferation of new bone in either the horizontal or vertical direction that produces a change in the shape of the joint contour; (b) POR (pitting and/or erosion of the joint surface); (c) EB (polished subchondral bone with or without ridges) (Buikstra & Ubelaker, 1994). OA changes were scored in: (a) shoulder (articular surface of scapula and humeral head); (b) elbow (articular surfaces of distal end of humerus and articular surfaces of proximal end of ulna and proximal end of radius); (c) wrist (articular surfaces of distal end of ulna and articular surfaces of distal end of radius); (d) hip (acetabulum [pelvic bone] and articular surface of femoral head); (e) knee (articular surface of distal end of femur and articular surfaces of proximal end of tibia); and (f) ankle (distal end of tibia). Data were recorded using the following four-point rating scale developed by Buikstra and Ubelaker (1994): OP: (0) no observable change; (1) barely discernible; (2) sharp ridge, sometimes curled with spicules; and (3) extensive spicule formation. POR: (0) no observable change; (1) pinpoint; (2) coalesced; and (3) both pinpoint and coalesced. EB: (0) no observable change; (1) barely discernible; (2) polished only; and (3) polished with groove(s).

When selecting entheses to examine, the following factors were considered: (a) usage of muscle in "daily activity" (Bochenek & Reicher, 2010); (b) more frequent occurrence of specific entheses in studies by various authors, (c) entheses variability, and (d) degree of bone material preservation. Considering the above, seven entheses were examined here. The details of the entheses under analysis are presented in Table 2.

The scoring system developed by Hawkey (1998) and Hawkey and Merbs (1995) was used to analyze the entheseal changes. This scoring system was chosen in order to be comparable to other studies (e.g., Eshed et al., 2004; Molnar, 2006; Lieverse, Bazaliiskii, Goriunova, & Weber, 2009;

TABLE 1 Age and sex distribution of the Łekno samples

Age category	Males	Females
Young adult (20–34 years)	28	23
Middle adult (35–49 years)	57	22
Old adult (50+ years)	25	11
Total	110	56

TABLE 2 Entheses analyzed in the present study

Insertion site (insertion site)	Muscle
Bicipital groove	Pectoralis major
Deltoid tuberosity	Deltoid
Bicipital tuberosity	Biceps
Midshaft of radius	Pronator teres
Gluteal tuberosity	Gluteus maximus
Linea aspera	Adductor brevis, Adductor longus, Adductor magnus, Biceps femoris (short head), Vastus lateralis, vastus lateralis
Soleal crest	Soleus

TABLE 3 Equations used for body mass reconstruction according to McHenry (1992)

Formulae
logBM = 0.7927 × logFEMSHFT − 0.5233
logBM = 0.8069 × logFEMSHFT − 0.5628
logBM = 0.8107 × logFEMSHFT − 0.5733

FEMSHFT-femoral shaft product (M9 × M10), abbreviation of bone measurements (M9 – lateral-medial upper cross-section of the bone shaft in the place 3–4 cm below the greater trochanter; M10 – anteroposterior upper cross-section of the bone shaft at the same height as M9) according to Martin and Saller (1957). Body mass (kg) was calculated as a mean value of the results obtained from the above equations.

Niinimäki, 2011, Niinimäki, 2012; Weiss, Corona, & Schultz, 2012). Hawkey (1998) and Hawkey and Merbs (1995) distinguish three types of entheseal changes: robusticity, stress lesion, and ossification exostosis. The robusticity marker is observed as a sharp ridge or crest. The stress lesion is defined as a pitting or furrow on the cortical bone. Ossification exostosis is observed as marginal lipping within entheses (Hawkey & Merbs, 1995). The robusticity marker is treated as a normal, non-pathological reaction to habitual muscle usage and it reflects the daily activity. Stress lesion and ossification are treated as pathological changes (Benjamin et al., 2002; Galera & Garralda, 1993; Villotte et al., 2010) and are thought to be response to micro- or

TABLE 4 The results for sample size (n), mean (x), and SD for osteoarthritic changes (OP-osteophytes, POR-porosity, and EB-eburnation) and the Mann–Whitney U test (Z) results for osteoarthritic changes between males and females in the Lekno material

Compare table 3 from D8 Please note that marking specific rows or columns is difficult there may be minor errors in highlighting

		Males			Females				
		n	x	SD	n	x	SD	Z	p
Shoulder	OP	105	0.408	0.635	41	0.365	0.667	−0.903	.366
	POR	109	0.403	0.896	40	0.449	0.999	0.181	.856
	EB	104	0.009	0.047	46	0.024	0.109	0.106	.915
	All	318	0.277	0.449	127	0.263	0.401	−0.418	.676
Elbow	OP	107	0.535	0.641	33	0.577	0.781	0.197	.843
	POR	117	0.046	0.291	41	0.162	0.506	0.766	.444
	EB	125	0.000	0.000	41	0.000	0.000	−0.004	.997
	All	347	0.172	0.248	115	0.205	0.326	−0.159	.874
Wrist	OP	104	0.243	0.509	32	0.249	0.503	0.036	.971
	POR	104	0.141	0.597	32	0.094	0.376	−0.293	.770
	EB	109	0.000	0.000	32	0.000	0.000	−0.005	.996
	All	317	0.134	0.307	96	0.117	0.200	−0.035	.972
Hip	OP	105	0.434	0.629	44	0.303	0.503	−0.958	.338
	POR	109	0.402	0.832	48	0.818	1.189	0.985	.325
	EB	108	0.000	0.000	48	0.049	0.209	0.246	.806
	All	322	0.275	0.420	140	0.415	0.592	0.504	.614
Knee	OP	82	0.408	0.670	28	0.631	1.046	0.007	.994
	POR	86	0.283	0.753	35	0.314	0.759	−0.179	.858
	EB	97	0.000	0.000	35	0.000	0.000	−0.005	.996
	All	265	0.256	0.432	98	0.276	0.474	−0.610	.542
Ankle	OP	80	0.072	0.261	40	0.024	0.109	−0.403	.687
	POR	83	0.199	0.719	38	0.074	0.334	−0.488	.626
	EB	82	0.000	0.000	37	0.000	0.000	−0.005	.996
	All	245	0.090	0.265	113	0.036	0.138	−0.742	.458
Total	OP	583	0.377	0.461	216	0.303	0.504	−1.910	.056
	POR	606	0.289	0.617	234	0.339	0.695	−0.025	.980
	EB	625	0.002	0.014	239	0.009	0.055	0.220	.826
	All	1818	0.219	0.283	689	0.222	0.360	−1.343	.179

*Statistically significant at $p \leq .05$; n, number of articular surfaces where each type of osteoarthritic change could be examined; x, mean calculated from the available osteoarthritic changes.

TABLE 5 Entheseal changes at specific skeletal sites in males and females in the Łekno material

	Males			Females				
Entheses	*n*	*x*	*SD*	*n*	*x*	*SD*	*Z*	*p*
Bicipital groove	104	1.71	0.934	46	1.46	0.650	−1.790	.073
Deltoid tuberosity	109	1.07	0.743	46	1.20	0.859	0.840	.401
Bicipital tuberosity	79	1.71	0.667	31	1.77	0.706	−0.217	.828
Midshaft of radius	84	1.30	0.760	41	0.87	0.653	−2.463	.014*
Gluteal tuberosity	94	2.02	0.617	37	2.11	0.727	0.384	.701
Linea aspera	99	2.07	0.807	51	1.84	0.593	−1.650	.099
Soleal crest	88	1.62	0.803	38	1.13	0.760	−1.807	.071
Total	657	1.62	0.608	290	1.37	0.612	−2.470	.013

*Statistically significant at $p \leq .05$ using the Mann–Whitney *U* test (*Z*); *n*, number of available entheses; *x*, mean calculated from the all available entheses; *SD*, standard deviation.

TABLE 6 The stature, body mass, and humeral massiveness index (HMI) males and females in the Łekno material

	Males		Females	
	N	*x*	*N*	*x*
Stature (cm)	103	168.6 ± 4.3	48	157.5 ± 5.6
Body mass (kg)	75	63.3 ± 7.8	40	51.7 ± 7.2
HMI	61	20.4 ± 1.4	30	19.9 ± 1.3

N, sample size; *x*, mean.

macro-traumas accompanying a strenuous physical activity (Hawkey, 1998; Mariotti, Facchini, & Belvastro, 2004; Takigawa, 2014) as well as a number of diseases of the locomotor system (Capasso, Kennedy, & Wilczak, 1999; Cardoso & Henderson, 2010).

In order to eliminate those entheseal changes that may be related to diseases or injuries, which could bias the research results, only the robusticity type of changes are included in this study. The following four grades of robusticity were analyzed according to Hawkey (1998) and Hawkey and Merbs (1995): (0) no observable changes in tendon attachment site, (1) weakly expressed robusticity, (2) moderate grade of robusticity, and (3) strong robusticity at the attachment site.

The reconstruction of body build traits includes reconstructed body mass, reconstructed stature and bone massiveness index (HMI). For this, the following seven bone measurements were made: humerus (M1-maximum length; M7-the smallest circumference of the shaft), radius (M1-maximum length), femur (M1-maximum length; M9-upper transverse cross-section of the shaft; M10-upper sagittal cross-section of the shaft), and tibia (M1-total length). The measurements were taken using the techniques described by Martin and Saller (1957).

In the present study, reconstruction of body mass was based on lower ("weight-bearing") limb bone diameters (femur), which are more correlated with body mass than upper limbs (Porter, 1999; Ruff, 2000). A mechanical method of body mass estimation was used in this work. The mechanical methods can be divided into those that use articular surface dimensions (Ruff, Scott, & Liu, 1991; Squyers & Ruff, 2015) and those that use cross-sectional dimensions and diaphyseal breadths (Auerbach & Ruff, 2004). Due to the fact that the osteoarthritic changes analyzed in this study can affect the shape and dimensions of articular surfaces, and therefore can influence the obtained results, methods other than those based on articular surface dimensions had to be chosen. McHenry (1992) proposed methods that used the anteroposterior and transverse diameters of the femoral shaft (just inferior to the lesser trochanter). McHenry (1992) found a high correlation of the femoral shaft with reconstructed body weight. Therefore, in this study, the method of McHenry (1992) based on upper transverse and sagittal cross-section of the femoral shaft was chosen for body mass reconstruction. The formulas used for body mass estimation are presented in Table 3. Body mass is given in kilograms.

Stature was reconstructed on the basis of the method according to Pearson (1899). This method is considered to be one of the most useful methods of stature reconstruction for medieval skeletal populations from Poland (Kozak, 1996). Stature was estimated using the maximum length of the humerus (M1), the maximum length of the radius (M1), the maximum length of the femur (M1), and the total length of the tibia (M1). Stature is given in centimeters.

To examine the relationships between osteoarthritic changes and bone massiveness, the humeral massiveness index (HMI) was calculated. Humeral bone was chosen for massiveness index calculation because: (a) since the humerus is a non-weight bearing bone, it was possible to eliminate an influence of body mass, as one of the other etiological factors in osteoarthritic changes, on bone massiveness; and (b) in the upper limbs, the proximal and distal bones are equally variable (Holliday, 1999), and the number

of humeral bones was greater than the number of radial or ulnar bones. The size of the humerus was estimated using the HMI: [(M7/M1) × 100; M1-maximum length (in mm); M7-the smallest circumference of the shaft (in mm)] (Piontek, 1996). HMI is given without any units.

Statistical analyses were made using a mean value of OA calculated as a mean value of observable OA changes. The analyses were made for each type of OA change in each joint, for each type of OA change from all joints taken together, and for each type of OA change and each joint taken together. The mean values of entheseal changes as a mean value calculated from observable entheseal changes were used. Differences between males and females in OA changes and entheseal changes were examined using Mann–Whitney *U* statistics. Multiple Logistic Regression was used to analyze the mutual interactions between OA changes, entheseal changes, body mass, stature, HMI, sex, and age. To make the analysis, OA changes were used as dichotomic

TABLE 7 Osteoarthritic changes predictors in Multiple Logistic Regression in the Łękno material

	B	(*SE*)	*p* value	Odds ratio	95% CI
OA					
Intercept	54.989	36.597	.133		
Entheses	**0.436**	**0.213**	**.041***	**1.546**	**1.018, 2.348**
Body mass	0.509	0.261	.051	1.663	0.997, 2.775
Stature	−0.331	0.239	.166	0.718	0.450, 1.146
HMI	**−1.818**	**0.860**	**.034***	**0.162**	**0.030, 0.875**
Sex	3.249	2.558	.204	25.756	0.171, 3870.963
Age	1.401	1.268	.270	4.058	0.338, 48.742
OP					
Intercept	16.586	27.142	.541		
Entheses	**0.277**	**0.127**	**.029***	**1.319**	**1.029, 1.691**
Body mass	0.248	0.135	.065	1.282	0.985, 1.668
Stature	−0.185	0.180	.303	0.831	0.584, 1.182
HMI	−0.352	0.399	.377	0.703	0.322, 1.536
Sex	2.627	2.177	.228	13.833	0.194, 985.89
Age	1.557	0.967	.107	4.742	0.713, 31.531
POR					
Intercept	36.405	18.142	.045		
Entheses	0.036	0.075	.631	1.036	0.896, 1.199
Body mass	0.050	0.057	.387	1.051	0.939, 1.176
Stature	**−0.195**	**0.099**	**.049***	**0.823**	**0.678, 0.999**
HMI	−0.523	0.304	.085	0.593	0.327, 1.076
Sex	1.228	1.090	.260	3.415	0.403, 28.946
Age	1.010	0.602	.093	2.744	0.844, 8.923
EB					
Intercept	−8.137	50.391	.872		
Entheses	0.422	0.298	.156	1.525	0.851, 2.732
Body mass	−0.035	0.123	.775	0.965	0.758, 1.229
Stature	−0.049	0.249	.843	0.952	0.585, 1.550
HMI	0.550	0.980	.575	1.733	0.254, 11.824
Sex	−0.185	2.906	.949	0.831	0.003, 247.187
Age	−1.780	1.881	.339	0.165	0.004, 6.602

*Statistically significant at $p \leq .05$. B, unstandardized regression coefficient; *SE*, standard error; CI, confidence interval; OA, all types of osteoarthritic changes, and all of joints taken together; OP, osteophytes from all of joints, POR, porosity from all of joints; EB, eburnation from all of joints; Entheses, mean of all observable entheses; HMI, humeral massiveness index.

TABLE 8 Osteoarthritic changes predictors in Multiple Logistic Regression according to joint in the Łękno material

	B	(*SE*)	*p* value	Odds ratio	95% CI
Shoulder					
Intercept	1.514	18.500	.934		
Entheses	0.101	0.077	.191	1.106	0.951, 1.287
Body mass	0.014	0.055	.801	1.014	0.910, 1.130
Stature	−0.035	0.099	.722	0.965	0.795, 1.172
HMI	0.158	0.303	.601	1.172	0.647, 2.121
Sex	1.869	1.216	.124	6.479	0.598, 70.198
Age	0.039	0.601	.949	1.040	0.320, 3.377
Elbow					
Intercept	19.389	21.463	.366		
Entheses	0.122	0.106	.246	1.130	0.904, 1.434
Body mass	0.100	0.074	.174	1.105	0.957, 1.278
Stature	−0.151	0.121	.211	0.860	0.679, 1.093
HMI	−0.407	0.382	.286	0.665	0.315, 1.404
Sex	2.337	1.599	.144	10.348	0.425, 239.14
Age	**2.302**	**0.934**	**.014***	**9.989**	**0.472, 4.131**
Wrist					
Intercept	22.968	25.536	0.368		
Entheses	**0.350**	**0.130**	**.007***	**1.419**	**1.099, 1.831**
Body mass	−0.010	0.072	.891	0.990	0.861, 1.139
Stature	−0.146	0.136	.284	0.864	0.662, 1.129
HMI	−0.179	0.428	.676	0.836	0.362, 1.934
Sex	1.983	1.829	.278	7.266	0.202, 261.80
Age	−1.091	0.837	.192	0.336	0.065,1.732
Hip					
Intercept	15.243	19.571	.436		
Entheses	**0.302**	**0.106**	**.004***	**1.353**	**1.099, 1.667**
Body mass	−0.009	0.075	.905	0.991	0.855, 1.148
Stature	−0.091	0.114	.426	0.913	0.730, 1.142
HMI	−0.086	0.324	.791	0.918	0.486, 1.731
Sex	0.139	1.172	.906	1.149	0.116, 11.411
Age	−1.017	0.750	.175	0.362	0.083, 1.573
Knee					
Intercept	5.606	22.528	.804		
Entheses	0.033	0.089	.706	1.034	0.246, 7.937
BM	−0.033	0.069	.627	0.967	0.845, 1.107
Body mass	−0.035	0.122	.775	0.966	0.760, 1.227
Stature	−0.109	0.379	.774	0.897	0.427, 1.884
Sex	0.754	1.380	.585	2.126	0.142, 31.795
Age	1.131	0.739	.126	3.100	0.729, 13.185
Ankle					
Intercept	34.008	38.212	.374		
Entheses	0.075	0.127	.556	1.078	0.175, 25.635

(Continues)

TABLE 8 (Continued)

	B	(*SE*)	*p* value	Odds ratio	95% CI
Body mass	0.046	0.106	.661	1.047	0.852, 1.288
Stature	−0.189	0.209	.367	0.828	0.550, 1.247
HMI	−0.373	0.546	.495	0.689	0.236, 2.009
Sex	0.901	2.232	.687	2.461	0.031, 195.47
Age	−1.013	0.993	.308	0.363	0.052, 2.542

*Statistically significant at $p \leq .05$. *B*, unstandardized regression coefficient; *SE*, standard error; CI, confidence interval; entheses, mean of all observable entheses; HMI, humeral massiveness index.

traits (0-no observed change and 1-observed change). The following traits were correlated: (a) dependent variables: OA changes within the shoulder, elbow, wrist, hip, knee, and ankle; (b) independent variables: entheses (mean of all observable entheses), body mass, stature, HMI, sex, and age. Statistical significance was determined at the probability level of .05. Statistical analyses were carried out using the Statistica 10.0 PL software and R-project (R Core Team, 2016).

3 | RESULTS

Table 4 contains results for the mean (x), standard deviation (*SD*), and sample size (n) for OA changes (OP, POR, and EB) according to the joints noted in the Łekno material. In males, the mean of OP was 0.38, POR was 0.29, and EB was 0.002. When all joints and OA changes were taken together, the mean was 0.219. In the female group, the mean of OP was 0.303, POR was 0.34, and EB was 0.009. When all joints and OA changes were taken together the mean was 0.222. The differences in OA changes between males and females were not statistically significant.

Table 5 contains the results for mean (x), standard deviation (*SD*), and sample size (n) for entheseal changes in the Łekno material. In males, when all entheses were taken together, the mean was 1.62, and in females, it was 1.37. In males, the means were highest for the gluteal tuberosity ($x = 2.02$) and linea aspera ($x = 2.07$) and the lowest for the deltoid tuberosity ($x = 1.07$). In females, the means were highest for the gluteal tuberosity ($x = 2.11$) and linea aspera ($x = 1.84$) and the lowest for the pronator teres origin ($x = 0.87$). Statistically significant differences between males and females were obtained for the pronator teres origin and when all entheses were analyzed together.

Table 6 presents the basic statistical characteristics (results of mean [x], sample size [N], and *SD*) in the skeletal material from Łekno.

Multiple Logistic Regression results for the interaction between osteoarthritic changes (OA—mean of all observable osteoarthritic changes; OP—mean of all observable OP; POR—mean of all observable POR; and EB—mean of all observable EB) and the body mass, stature, HMI, sex, and age in the Łekno material are given in Table 7. Entheses were the strongest contributor for the prediction of osteophyte expression (all changes taken together) ($p = .029$) and arthritis expression when all types of changes and all joints were taken together (OA) ($p = .041$). The more severe the OP and general OA (all changes taken together) were, the higher the values of entheses will be. Stature demonstrated a negative dependence with POR, which means that as stature increases, POR decreases ($B = -0.195$). Individuals with more massive humeri were more likely to have less severe OA (all OA changes and all joints taken together) ($B = -1.82$). Neither body mass, humeral bone massiveness, and sex, nor age variables demonstrated a statistical relationship with OA, OP, POR, or EB. It suggests that these traits are not relatively important in determining OA changes in the study sample and have little to no effect on differences in OA severity (Table 7).

The Multiple Logistic Regression results for the interaction between OA changes in the shoulder, elbow, wrist, hip, knee and ankle and the body mass, stature, bone massiveness index, sex and age in the Łekno sample are given in Table 8. Entheses were the strongest contributor to the prediction of arthritis expression only in the wrist ($B = 0.35$) and hip ($B = 0.302$). The more severe the OA changes in the wrist and hip were, the higher the values of entheses will be. Age was the strongest contributor to the prediction of arthritis expression only in the elbow ($B = 2.302$). The oldest individuals were predicted to more likely have OA changes in the elbow. Body mass, stature, bone massiveness, and sex had no effect on OA changes in any of the examined joints (Table 8).

4 | DISCUSSION

In the skeletal material from Łekno, entheses were the strongest contributor to the prediction of arthritis expression when all

types of changes and all joints were taken together (OA) (p = .041) and when OP from all joints were taken together (p = .029). More muscular individuals were predicted to more likely have developed OA and OP (Table 7). Individuals with more pronounced muscle attachment sites (entheses) were more likely to have more developed OA changes in the wrist (B = 0.35) and hip (B = 0.302) (Table 8). Entheses had no significant influence on the expression of POR, EB, and OA changes in the shoulder, elbow, knee, or ankle. Age was the strongest contributor to the prediction of arthritis expression in the elbow only (B = 2.302). The oldest individuals were predicted to more likely have OA changes in the elbow. In the analyzed skeletal group, smaller individuals were predicted to more likely have developed POR (B = –0.20, p = .049) (Table 7). Body mass, stature, bone massiveness, and sex had no significant effect on OA changes in any of the examined joints (Table 8).

Taking the present study results (an existence of the dependences between OA changes and entheses; Table. 8) and the assumption that entheses are treated (with a causation; see below) as physical activity markers one could assume that there is an impact of physical activity on the OA changes appearance—more active individuals tend to have more expressed OA. But we must be cautious about such a simple interpretation of the relationship between OA and entheses. First, because the effect of physical activity on the formation of entheseal changes is questioned in the anthropological literature (Daly, Saxon, Turner, Robling, & Bass, 2004; Havelková, Hladík, & Velemínský, 2013; Henderson et al., 2013; Henderson & Cardoso, 2013; Lieverse et al., 2009; Lopreno, Cardoso, Assis, Milella, & Speith, 2013; Lovejoy, Mccollum, Reno, & Rosenman, 2003; Niinimäki, 2012; Villotte & Knüsel, 2013; Weiss, 2007; Weiss et al., 2012). Most researcher stress the multifactorial etiology of entheses and emphasize the role of factors other than physical activity including, for example, genes, age, sex, hormones, and body mass (Milella, 2014; Milella, Giovanna Belcastro, Zollikofer, & Mariotti, 2012; Niinimäki, 2011; Schlecht, 2012; Villotte & Knüsel, 2013). Second, although in this study individuals with more developed entheses were predicted to have more severe OA (all types of changes and all joints taken together), when each joint was analyzed separately, not all dependences were significant (Table 8). Also, results from other bioarchaeological studies do not support a simple explanation of the activity-related relationship between OA and entheses (Myszka, 2015; Palmer et al., 2016; Schrader, 2012; Woo & Pak, 2013). These studies show a lack of a significant dependence between OA and entheses, thus illustrating the variability of these two groups of skeletal features, their complex etiologies and suggesting that they are a result of different activity-related phenomena (Palmer et al., 2016; Schrader, 2012). OA and entheses react in different ways to variable etiological factors, and they have different levels of vulnerability to various causes (Rojas-Sepúlveda & Dutour, 2014; Woo & Pak, 2013) Definite confirmation or refuting of the theory about the impact of physical activity on the development of OA is not possible yet, and effects of other factors on the development of OA must be considered. It also seems that further research should be placed on occupations and their roles in the pathogenesis of OA.

The positive significant dependence between OA changes and entheses obtained here might indicate the validity of the "bone formers theory," according to which some individuals have greater tendency to form new bone (especially OP, entheses, and enthesophytes) in response to mechanical stress (Crubézy et al., 2002; Rogers, Shepstone, & Dieppe, 1997). As Rogers et al. (1997) suggest, bone formation is one of the components of the response of the musculoskeletal system to stress, and the variation in bone formation could be due to differences in individual ability to form bone in response to stress rather than due to differences in stress. It can suggest a genetic control of the pathogenesis of musculoskeletal disorders (Rogers et al., 1997). In our study, when a joint or OA changes were considered individually, a dependence between entheses and OA did not always exist, but when all joints were taken together, a dependence between OA and entheses was significant. Taking this into account, it can be hypothesized that individuals are either predicted or not predicted to have OA changes and they are predicted to be bone formers (on the basis of genes). But whether they will have OA changes or not will depend on their lifespan. However, this is just a hypothesis and further studies are needed to confirm it. While in light of the "bone formers theory," the existence of a dependence between entheses and OP is easy to explain, the relationship between entheses and EB or POR may be unexpected. Similar positive dependences between POR and/or EB and entheses were obtained by Rogers, Shepstone, and Dieppe (2004) in an England collection (900–1850 BC) and Molnar et al. (2011) in Neolithic hunter–gatherer populations from Gotland. Rogers et al. (2004) explain this by the assumption that such a relationship is a consequence of a generalized predisposition to skeletal remodeling in response to mechanical stress and suggest that OA is part of a systemic bone disorder.

The significant relationship between entheses and OA changes obtained in this work and in other studies (Molnar et al., 2011; Palmer et al., 2016; Rogers et al., 2004; Schrader, 2012) are meaningful. Although these results cannot undeniably indicate a similar etiology of these features in the two skeletal groups, they also cannot be ignored. These results may indicate that increased physical activity or the lack of it can be significant for the formation of OA changes. It should be borne in mind that either OA or entheses have a multifactorial etiology (Arden & Nevitt,

2006; Gabay et al., 2008; Roach & Tilley, 2007; Weiss & Jurmain, 2007), and physical activity is not the only etiological factor. Furthermore, the existence of a relationship between OA and entheses might not indicate a link between these two skeletal traits and physical activity.

Łekno was a part of settlement complex where in historical times settlements and architectural structures of considerable political, administrative, socio-economic, and religious significance were located (Wyrwa, 1989). But the examined population is not well documented in terms of lifestyle and occupation, which additionally hinders the interpretation of the dependency between physical activity and analyzed skeletal traits.

In the Łekno sample, age was the strongest contributor to the prediction of arthritis expression in the elbow only ($B = 2.302$) (Table 8). In past skeletal populations, the influence of age on OA is not so obvious. It is both positive and negative, and in many cases, it is not significant and depends mostly on an individual joint (for the discussion see Weiss & Jurmain, 2007). Specificity of the skeletal material does not always allow for detailed analysis and/or reliable interpretation of the differences in the separate age groups (e.g., small sample sizes and often not well preserved). Moreover, some researchers question the concept of a simple relationship between these two features, underlining the multifactorial etiology of OA and suggest that aging contributes to but does not directly cause the OA changes (Anderson & Loeser, 2010; Loeser, 2011). They argue that the occurrence of these dependences is not only an effect of aging of joint tissues but also results from the influence of other factors, such as joint loading from obesity over time (Newman et al., 2003), increased joint instability due to ligamentous laxity and others (Sharma, 1999).

In the present study, stature demonstrated a negative dependence with POR ($B = -0.195$). Neither OP and EB (Table 7) nor OA in any single joint was significantly affected by stature (Table 8). It is difficult to justify the reasons for this result. For example if OA is connected with mechanical loading of the joint (Arden & Nevitt, 2006; Dieppe, 1995), one would expect that first, the relationship should be positive, and second, the OA changes in the lower limb joints should be more severe (greater body height puts more mechanical pressure on lower joints). The lack of effect of stature on OA severity in any joint region (Calce et al., 2018) could explain that stature is a complex polygenic trait influenced by both genes and environmental factors, wherein more than 80% of the variation in height may be genetically determined (Kannu, Bateman, Belluoccio, Fosang, & Savarirayan, 2009; Sanna et al., 2008). It can be suggested that not stature but body size (with an important role of body mass) plays a role in the development and progression of OA (Marks & Allegrante, 2002; Reijman et al., 2007).

Body mass had no significant effect on OA changes both when each type of OA change was examined (Table 7) and when each joint was analyzed separately (Table 8). The impact of body mass on OA when all joints and all types of OA changes were taken together was marginally significant ($B = 0.51$; $p = .051$) and, in some cases, that effect was negative (although nonsignificant). These results are similar to earlier anthropological data, where nonsignificant/marginally significant (positive or negative) relationships between body mass and OA were found (Jurmain, 1991; Weiss, 2004, 2006). Weiss (2006), who examined OA in Californian Amerinds (500–1500 AD) and Weiss and Jurmain (2007) who revisited OA in past human populations claimed that obesity could be a modern phenomenon and therefore an influence of both mechanical and biological factors on past human joints could be less important. Weiss (2006) also underlined the effect of reconstructed body mass deficiency. Body mass reconstruction methods are not entirely reliable, and therefore the effect of body mass on the development of OA in skeletal collections cannot be precisely defined.

In the Łekno sample, individuals with more massive humeri had less severe OA (all OA changes and all joints taken together) ($B = -1.82$, $p = .034$) (Table 7). Cope et al. (2005), referred to the obtained correlations between OA and bone massiveness as Wolff's law, according to which mechanical stress affects cortical bone development resulting in bone remodeling (Cope et al., 2005; Ruff, Trinkaus, Walker, & Larsen, 1993). Therefore, people who engage in physically stressful activities tend to have enlarged bones compared to people living sedentary lives. More active individuals with resulting more massive bones tend to have more severe OA (Cope et al., 2005). However, it must be stressed that the model is far too simplified and should not be applied indiscriminately. The general model of bone modeling and remodeling during the human lifespan proposed by Ruff, Walker, and Trinkaus (1994) assumes that while juveniles deposit subperiosteal bone and slow the endosteal resorption in response to strenuous mechanical loading, adults tend to slow the endosteal resorption and are not able to add substantial amounts of subperiosteal bone, even if they are very active during adulthood. Nevertheless, taking these indirect relationships between physical activity, OA, and bone robusticity, it could be hypothesized that more active individuals (especially in childhood and adolescence) tend to have more massive bones, by increasing the magnitude of bone cross-sections. Strenuous activity is a very effective way to strengthen bones (Turner & Robling, 2003). According to Sowers (2001) and Hunter and Spector (2003) stronger, stiffer bones are not capable of dissipating the biomechanical loading that finally leads to cartilage failure and

the appearance of OA changes. But the detailed mechanisms and factors underlying changes in bone robusticity and geometry are not completely understood (Pearson & Lieberman, 2004). The relationship between OA and bone robusticity is not unequivocal in the anthropological literature. The dependence between entheses and bone robusticity does not always exist (for the discussion see Myszka & Piontek, 2013). Therefore, no final conclusions regarding the relationship between bone robusticity and OA in skeletal populations can be drawn here, and further studies are needed.

In our sample, males were usually more affected than females, but sex differences were not significant (Tables 4, 7, and 8). While in clinical studies, the relationship between OA and sex is more clear (Hanna et al., 2009; Prieto-Alhambra et al., 2013), there is no consistency in sex differences in the frequency and prevalence of OA in anthropological studies (Eng, 2016; Klaus et al., 2009). Although considering the genetic and environmental background of sex differences, the assessment of sex differences in the analysis of OA as a necessary condition for reliable interpretation of the disorder in past populations is needed (Weiss & Jurmain, 2007). As a result, palaeopathologists are limited in explaining of the inhomogeneity of these results. There are a number of other factors that complicate the analyses of archived bone samples that include small sample sizes, inadequate preservation, difficulty in assigning sex, and other unknowns about the populations and the individuals within the populations.

In Summary, in the Łekno sample, entheseal changes appear to play an important role in OA changes. More "muscular" individuals tended to have more severe OA (all OA changes and all joints taken together), OP (all joints taken together), and OA in the wrist and hip. Other etiological factors had little to no effect on the development of OA. Bone massiveness appears to have a role in the appearance of OA (more massive individuals have less severe OA). Age had a significant (and positive) effect on OA changes in the elbow only. Stature affected POR only (negative dependence). Body mass did not significantly influence OA changes. These results may indicate that increased physical activity or the lack of it can be significant for the formation of OA changes.

The use of entheseal changes as markers of occupational stress is still in question; therefore, the existence of a relationship between OA and entheses might not indicate a link between these two skeletal traits and physical activity. The results support the view that the formation of OA changes is a complex process with a multifactorial etiology and suggests the need to further define the contribution of these etiological factors to the onset and development of OA in past humans. Further investigations on OA in past populations that include studies on the environment, occupations, ecology, and genetics will help elucidate the etiological factors in the occurrence and progression of OA in both modern and prior human populations.

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