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Dinosaur soft tissues still provide compelling evidence of young age

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ABSTRACT

Soft tissues found within dinosaur fossils remain one of the most compelling pieces of evidence in support of young-age creationism. Recently, however, the secular scientific community has proposed various models that challenge the previously held consensus constraining soft tissue preservation to a maximum of one million years. Preservation of 80-million-year-old dinosaur soft tissues is, as a result, now a much easier pill to swallow. I will first outline each of these models and then demonstrate that none of them adequately explains the preservation of soft tissues in *all* dinosaur fossils over geological time.

INTRODUCTION: THE BASICS

Over the last 20 years or so, multiple examples of pliable, stretchy tissues have been found in many dinosaur bones (Schweitzer et al. 2005, 2007; Armitage and Solliday 2020; Bailleul et al. 2020; Armitage 2021). These include intricate blood vessels, many of which still have remnants of haemoglobin attached to vessel walls, bone-forming osteocytes, neural filaments and even dinosaur DNA (Bailleul et al. 2020) (Figure 1).[1] Of course, these finds pose an interesting question: How can organic material preserve for 80 million years and sometimes up to 200 million years?[2]

How can organic material preserve for 80 million years and sometimes up to 200 million years?

Since this paper will primarily focus on the degradation of proteins, it will be helpful to review some basic protein biology. Most tissues are composed of proteins such as collagen, keratin and elastin. Of these three, collagen is the most widespread in our tissues (which are about 25% collagen). The collagen molecule is a three-stranded protein with each strand bound to the other strands by hydrogen bonds (Figure 2C). One of the reasons the collagen molecule preserves well is due to the helical structure that forms when these three strands are woven together during growth (Saitta et al. 2019) (Figure 2C). The durability imposed on this three-stranded molecule is analogous to that of three-ply rope, making collagen a hardy protein suitable for use in our tendons, as well as in other types of connective tissues.

Each of the three strands of the collagen molecule are themselves composed of amino acids which are the building blocks of all proteins (Figure 2C). There is a carboxyl group attached to one side of the amino acid and an amine group attached to the other side (both groups are called 'functional groups').[3] These two functional groups are joined by a special kind of covalent bond called a 'peptide bond'. This bond occurs when two amino acids come together and give off water as part of the reaction. This is called a 'dehydration reaction'[4] because water is lost in the process.

Given these basics, we can now begin to understand how and why collagen proteins denature (unfold), degrade and breakdown. The three most important factors that regulate the breakdown of proteins (proteolysis) are high temperatures, water and microorganism metabolism (Demarchi et al. 2016; Saitta et al. 2019). Increasing temperature leads to increasing motion at the molecular level, which can break the hydrogen bonds and ionic attraction holding proteins in their three-dimensional shape. The higher the temperature the more displacement between the bonds until eventually they break. Miles and Bailey (1999) found that the mammalian collagen molecule will begin to denature (unfold and thus lose its usefulness) at high temperatures of around 60˚C in a dry environment, and about 40˚C in the presence of a fluid. Denatured collagen molecules are susceptible to complete breakdown due to the work of fluids and microorganisms – to which we turn next.

Since peptide bonds (the bond between two amino acids) form by *giving off* water, it only makes sense that they will break with the *addition of water.* This is called a 'hydrolysis reaction'. Simplistically, the water actually becomes part of the chemistry and we say that the products have become 'hydrolysed'. The opposite occurs when amino acids combine, only this time the water molecule is joined back together and removed. This is called 'dehydration' because water is being removed from the growing organic molecules.

These reactions usually do not occur on their own but instead are aided by the work of special proteins called enzymes. Our bodies, for example, use enzymes to build up (anabolise) or break down (catabolise) tissues. At a

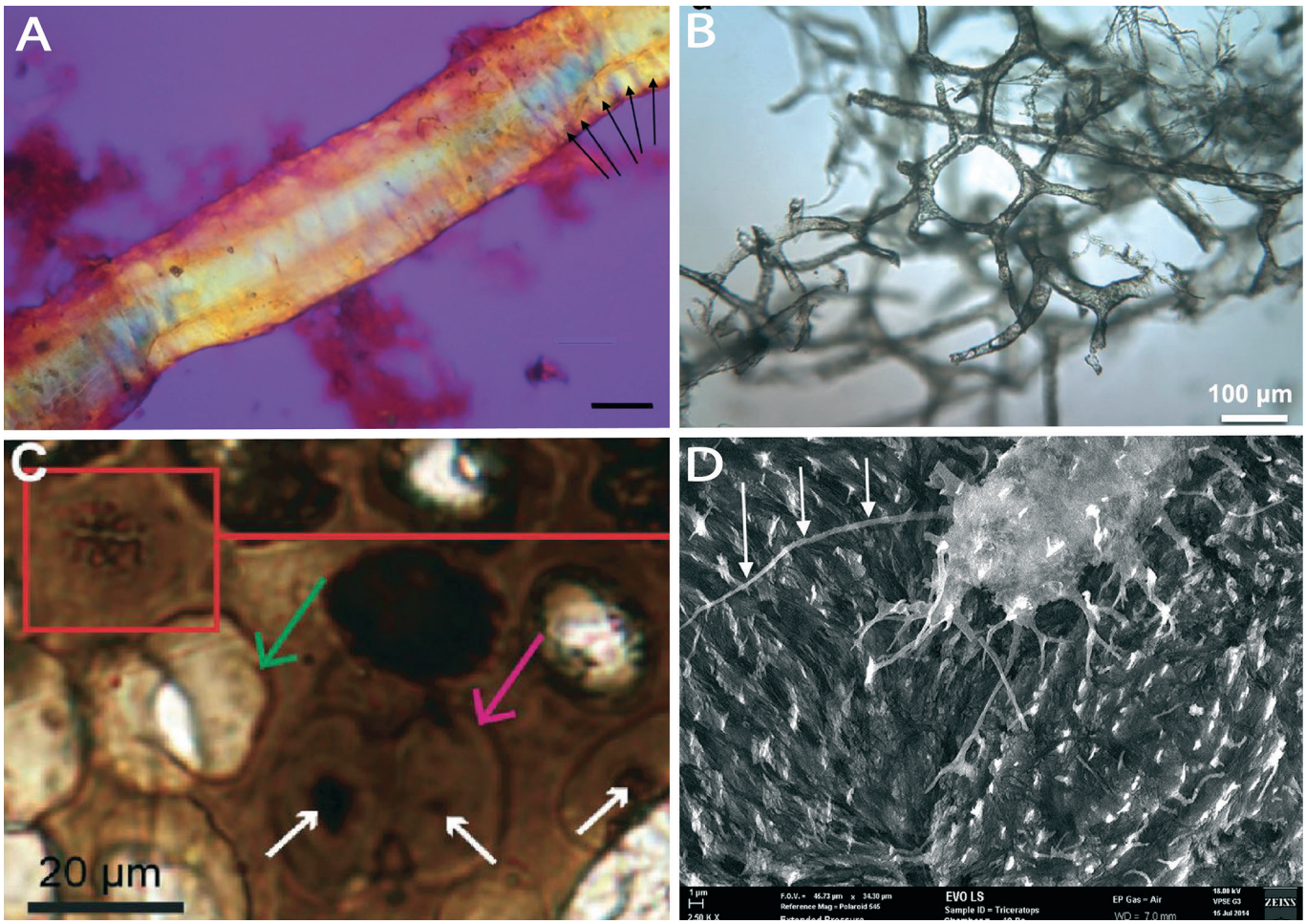

Figure 1. Soft tissues and cells from selected Open Access dinosaur journals. **(A).** Neural filament from a *Triceratops* bone. The clearly noticeable 'Bands of Fontana' (black arrows) cannot be reproduced by microorganisms. Scale bar 20 µm. Both A and D adapted from Armitage and Solliday (2020). See also the incredible images in Armitage (2021). **(B).** *Tyrannosaurus rex* vascular tissue. Notice that the vessels are hollow. A microbial origin is not convincing because, although microbes could replicate the shape of the vessels, there is no reason for them to replicate their hollow interiors. Adapted from Boatman et al. (2019). **(C).** Cartilage from *Hypacrosaurus* showing chondrocyte lacunae. Notice that some are empty, but others have intact chondrocytes with apparent visible nuclei (white arrows). The red box encloses a chondrocyte showing actual chromosomes frozen during metaphase of cell division. The team also recovered remnants of DNA from such chondrocytes. Again, it seems difficult to see how a microbial community can replicate cell nuclei. Adapted from Bailleul et al. (2020). **(D).** A bone-forming osteocyte from a *Triceratops* bone. Notice the long filipodia. It is hard to imagine that microorganismic 'biofilms' could reproduce these cells with such fragile filipodia.

very basic level, the reason the body's hydrated tissues do not break down while we are alive is because special enzymes are always at work promoting tissue stability, keeping amino acid chains (and thus proteins) intact. However, when death occurs, these enzymes eventually stop doing their job, and other enzymes sourced from the organism's own bacterial stockpile[5] go to work breaking the tissues down to the smallest of organic particles (putrefaction). Just to make matters worse, or better depending on your perspective, bacteria, fungi, moulds, protozoa, and other microorganisms that feed on dead tissues, get to work to make sure that the entire carcass is completely consumed. Since these tiny organisms tend to metabolise more efficiently in the presence of heat and water, higher temperatures and moisture will speed the process up and cooler, dryer temperatures will slow it down.

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Since heat, water, and bacteria exist everywhere at Earth's surface, all labile organic material will completely break down. This is even true deep within Earth's sedimentary basins where communities of anaerobic bacteria (those not requiring oxygen to metabolise) can thrive to depths of up to two kilometres (about 1.2 miles) and are found in all kinds of sequestered environments from shallow soils to deeply buried petroleum deposits (Saitta et al. 2019). This is important because it means tissues can still decay and break down when they are buried deep within the Earth, even in the absence of oxygen. You can perhaps begin to see why the idea of tissues

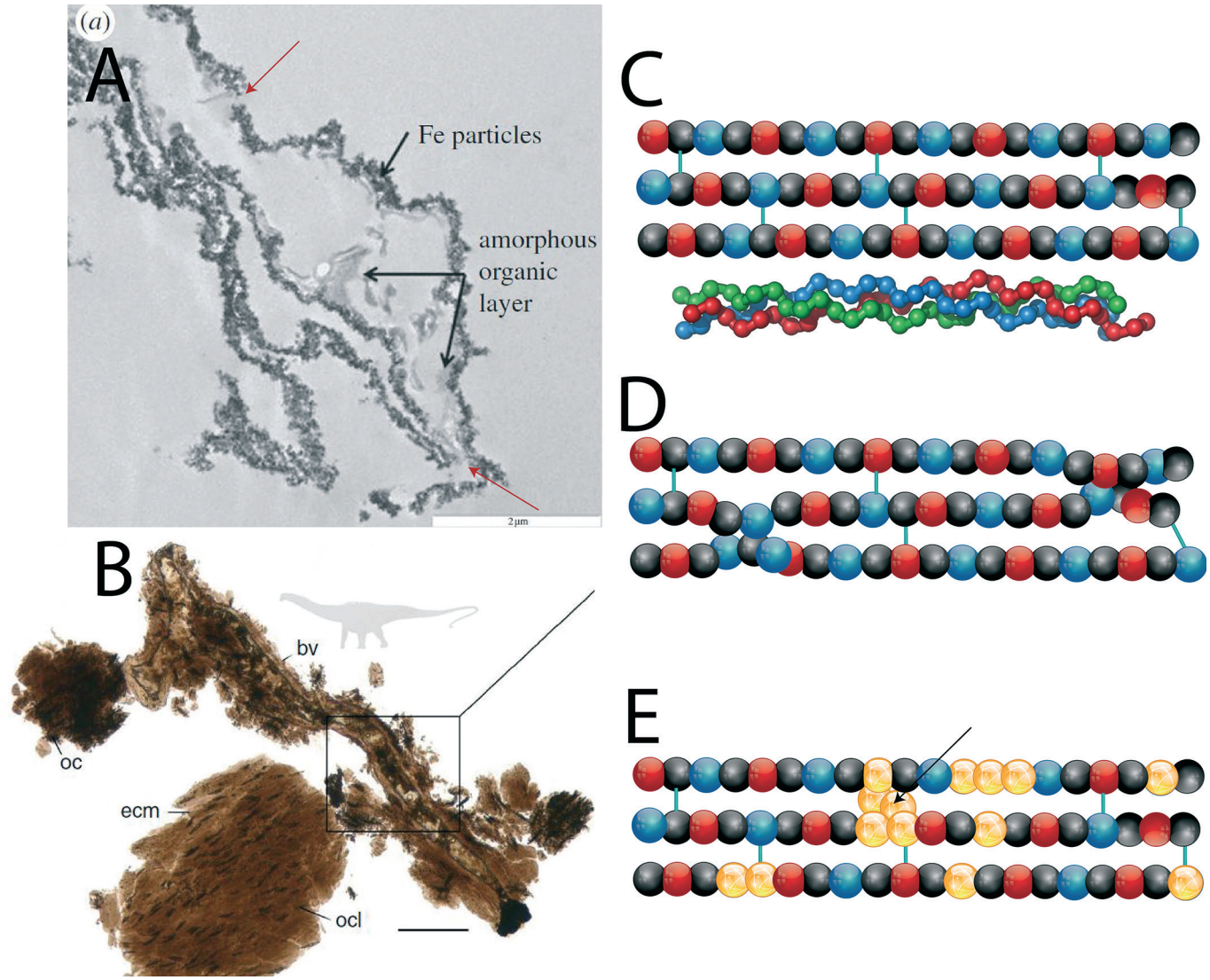

Figure 2. (A). Dinosaur soft tissue encapsulated in 'iron jacket'. Red arrows point to 'holes' in the jacket. This is a thin-section, so presumably 'holes' like this will be found throughout the three-dimensional structure. Adapted from Schweitzer et al. (2013). **(B).** Dinosaur soft tissue heavily altered by AGEs and ALEs. Note the dark brown colour. Scale bar = 100 µm. Adapted from, Wiemann et al. (2018). **(C).** Simplified uncoiled collagen model. Coloured spheres represent various amino acids. Light blue lines represent hydrogen bonds. Helix structure after coiling is depicted below main figure. Image: Wikimedia Commons / CC BY-SA 3.0. Nothing is to scale and the 'amino acid' colours and bonds do not conform to any known collagen blueprint. **(D).** Amino acid chains in molecule are cross-linked together. **(E).** Amino acids are altered by AGEs and ALEs (yellow spheres). AGEs and ALEs can also link chains of amino acids together (arrow).

surviving for 200 million years was once thought scientifically impossible.

PRESERVATION OF SOFT TISSUES

So how long does it take for proteins, for example, to *completely* disintegrate? Most organic material will break down completely in just a few months, but hardier organic tissues such as collagen, elastin and even some bone-forming cells, given a cool (about 10˚C), dry environment can last for thousands of years. But of course, finding collagen in a bone that is a thousand years old is not the same as finding it in bones that are 80 million to 200 million years old.

Recent, quantitative scientific research validates this qualitative assessment for protein preservation. Multiple scientific experiments have been conducted on various tissues to discover their preservability over time. Nielsen-Marsh (2002), for example, proposes that at 10˚C, collagen will completely break down in 180,000 years. Buckley and Collins (2011) are more hopeful. They sped up the rate of collagen degradation in abundant water at high temperatures. This of course only took a few months. They then calibrated this rate to a surface temperature of 10˚C, the proposed temperature of Britain over the last million years. Based on their modelling, they predicted that after 200,000 to 700,000 years, 1% of the total mass of collagen (I) would be left. That's astounding; 180,000 years to perhaps as long as 700,000 years is a long time, but we are still far short of multiple millions of years, let alone tens of millions of years.

What about DNA? Doing research on DNA, Tomas Lindahl (1993) determined that at low temperatures and with exceptional burial conditions, long sequences of DNA might last as long as several tens of thousands of years. Smith et al. (2001) agreed. This time frame was augmented by the work of Allentoft et al. (2012) who calibrated the rate of DNA decay to fossils that ranged a conventional time span of about 8,000 years. Their research indicated that at a surface temperature of about 15˚C, DNA would be completely broken down into single base pairs in about 130,000 years. Again, quite astonishing, but far, far, from the multi-million-year mark.

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Essentially, these analyses indicate that given exceptional burial settings, dry conditions and very low temperatures, proteins can last up to about 700,000 years and DNA can last about 130,000 years. (Only as base pairs, however. Longer sequences can only last for hundreds of thousands of years at sub-zero temperatures. In other words, they must be frozen.) Given these data, many scientists have sought to explain soft tissue preservation over geological time by appealing to several very complex geochemical models.

FOUR MECHANISMS FOR SOFT TISSUE PRESERVATION OVER GEOLOGICAL TIME

In the last six years or so, several papers have been published that claim to have pushed the preservation potential of organic material into the realm of geological time. Four basic models have been proposed: (1) Sequestration and protection within bone matrices; (2) Cross-linking processes that can link together proteins as well as amino acid chains within proteins; (3) Organometallic complexing and physical or chemical binding to mineral surfaces; and (4) Altering of proteins and other organic molecules with N-heterocyclic polymers.

1. Bone sequestration model

Proponents of this model suggest that the compact environment of the bone is itself sufficient to protect organic material given exceptional conditions of preservation (Collins et al. 2000). Yet other scientists contradict this hypothesis by pointing out the ecologically attractive setting that bone affords microorganisms:

> Furthermore, given that microbes can inhabit the crust kilometres below the surface … it might be predicted that bone remains a biologically active habitat even when buried hundreds of meters deep for millions of years. (Saitta et al. 2019)

In other words, since microorganisms are ubiquitous, even deep within the Earth's sediments, one cannot appeal to the 'safety' of deeply sequestered bone to preserve soft tissues over deep time.

2. Cross-linking model

The second model, cross-linking, occurs when two or more chains of organic elements are 'linked' together by naturally synthesised polymers after the death of an organism (Figure 2D). It is thought that unstable iron ions, sourced from endogenous[6] haemoglobin and myoglobin, contribute to the formation of free radicals. These free radicals, in the form of superoxides, hydroxyl radicals and/or hydrogen peroxides, react with free iron to form iron-oxygen complexes that contribute to the formation of cross-links across chains of organic material (Schweitzer et al. 2007; Boatman et al. 2019). Chains of amino acids (proteins), for example, can be covalently or ionically joined to each other by these iron-oxygen complexes. Given the three-ply structure of the collagen molecule, for example, such a mechanism would inhibit the work of water and enzymes which tend to cleave away amino acids in unfolded (denatured) collagen molecules. These 'linked' organic chains, therefore, tend to be more resistant to enzymatic attack and thus degradation.

Cross-linking of organic material is not a new discovery. Formaldehyde, which has been used as an embalming agent by humans for thousands of years, works in much the same way by 'linking' proteins together and extending preservation of tissues by sometimes thousands of years.

That dinosaur soft tissues have been altered by metal-catalysed intermolecular cross-linking seems fairly robust (Boatman et al. 2019). Yet how *effective* is cross-linking in preserving tissues over tens of millions of years, and has cross-linking altered *all* extant dinosaur soft tissues? The answer to the first question is quite vague. In an effort to answer that question, Schweitzer et al. (2013) performed actualistic experiments by soaking ostrich tissues in haemoglobin to induce iron-catalysed non-enzymatic cross-linking. The ostrich tissues were then left in storage for two years at room temperature in an oxygenated environment. Throughout this incubation period, the tissues retained their shape and did not appear to degrade much at all. So, what conclusion can we gather from this experiment? Although the tissues suffered very little degradation, can we use this single example to explain the preservation of soft tissues over *geological* time? I do not

think we can. Interestingly, Armitage and Solliday (2020, p.34) question the usefulness of this experiment given the lack of 'real-world conditions of buried animal remains'. Apparently, Schweitzer et al. (2013), took 'extraordinary means' to inhibit thrombosis. This included the infusion of anticoagulants, and the centrifugation of heme to separate out any serum, cells, platelets and other debris that might initiate a thrombotic reaction. According to Armitage and Solliday (2020), this was unrealistic because in the 'real-world' blood rapidly coagulates in minutes, causing a cascade of biochemical reactions that inhibit Fenton reactions, the production of cross-links, and thus the preservation of soft tissues.

Regarding the effectiveness of cross-linking to preserve dinosaur soft tissues, Boatman et al. (2019, p.9) say:

> Molecular crosslinks (essentially, hyper-crosslinking) would have afforded exceptional resistance to mechanical, biological, and thermal degradation …

They then furnish nine peer-reviewed journal article citations in support. Yet eight of these articles discuss the effects of cross-linking on human physiology and health, and one discusses the application of cross-linking to the development of polymers. These sources could appropriately be referenced if the researchers were discussing models of cadaver preservation, or perhaps explaining the preservation of organs in ancient Egyptian mummies, but in order to persuade the reader that cross-linking processes can really work to preserve tissues over geological time the authors must appeal to more relevant research. This becomes even more pertinent given a host of other variables that affect fossil diagenesis over deep time (see below).

a fascinating paper on the preservation of soft tissues in *cartilage* from an 80-million-year-old duck-billed dinosaur.

Now to the second question: Do examples of soft, pliable, stretchy tissues, unaltered by Fenton chemistry and cross-linking, exist? And the answer is yes. In 2020, Schweitzer and her team published a fascinating paper on the preservation of soft tissues in *cartilage* from an 80-million-year-old duck-billed dinosaur (Bailleul et al. 2020) (Figure 1C). Cartilage lacks all of the iron-producing and/or -containing proteins found in bone that act as catalysts for Fenton chemistry and thus cross-linking. Armitage and Solliday (2020) also discuss the existence of exceptionally preserved osteocytes, nerves and thin cuspids of incredibly fragile venule valves from *Triceratops* bones. According to the authors, these tissues show no sign of Fenton chemistry alteration.

Finally, other experts in this field seem to suggest that this mechanism cannot inhibit organic degradation over millions of years. Schweitzer et al. (2007, p.193), for example, say:

> Iron-triggered cross-linking of organic components, however, *is not sufficient to explain the persistence of soft tissues across geological time. (emphasis mine)*

Wiemann et al. (2018, p.2) likewise explain:

> Such preservation has been attributed to … anhydrous sugar-protein crosslinking[7] processes …, *but none of these models provides an explanation for patterns of originally proteinaceous soft tissue preservation in vertebrate hard tissues in deep time. (emphasis mine)*

3. Organo-metallic complexing or 'iron jacket' model

The third model, organo-metallic complexing and the physical and/or chemical binding of tissues to mineral surfaces, is biochemically more complex, but involves some similar biochemical players, including endogenous haemoglobin and myoglobin, but also other organic molecules such as cytochromes and ferritin, proteins which manufacture and/or transport iron oxyhydroxide mineral nanoparticles (Schweitzer et al. 2013).

In this model, iron – sourced from either the haemoglobin and/or myoglobin, or other organic molecules that use iron such as ferritin – are precipitated directly onto the tissues after the death of the organism. The biochemical pathways that lead to this precipitation are very complex but have been shown to occur in actualistic experiments (Schweitzer et al. 2013). Essentially, the tissues in question get 'jacketed' with tiny iron nanoparticles (Figure 2A). These biochemical 'jackets' are said to impart organic preservation to the tissues in one of two ways: (1) The jackets *physically* protect the tissues from enzyme attack – and thus decay; and (2) The jackets *chemically* protect the tissues from reactive oxygen species (free radicals). These free radicals like to strip away electrons from tissues – this is called oxidative damage.

Schweitzer et al. (2013) succinctly and persuasively discuss this method of preservation, but it is not without its problems. The most obvious problem is that not all dinosaur soft tissues have these metallic jackets. They seem to be absent, for example, in the samples discussed by Schweitzer et al. (2005, 2007). Neither paper mentions these biochemical jackets, despite the micro-scale,

detailed descriptions of the tissues, including the use of scanning electron microscopy (SEM) and transmission electron microscopy (TEM).[8] The iron jackets were, however, clearly detected in the 2013 paper as electron-dense iron nanoparticles during TEM. One would think they would also have been detected in the 2007 paper had they been present.

The purpose of these fine-scale descriptions was important back in 2005 and 2007 when these findings were treated with community-wide scepticism. Schweitzer and her team had to ensure that their description of these tissues excluded every possible contaminant including pyrite framboids, fungal species, bacterial biofilms, glue, preservatives, etc. If metallic biochemical jackets were present, they would have been discussed. Schweitzer et al. (2007, p.191) make this explicit:

> As arguably the most labile and easily degraded of the structures we observed, the presence of soft vessels is enigmatic. They are *neither biomineralized nor have any obvious inherent characteristics that would favour preservation … (emphasis mine)*

It is possible that these iron particles dissolved during the 2005 and 2007 demineralization process, but, according to the supplementary material, all of the fossil tissues discussed in both the 2007 *and* the 2013 papers were demineralised using 0.5 M Ethylenediaminetetraacetic acid. Since this acid did not remove the iron particles from the 2013 samples, it would seem reasonable to assume that it also did not remove the iron particles from the 2007 samples.

If, however, examples of soft, pliable, stretchy tissues exist – *in the absence of biochemical jackets* – then these current findings are, at best, merely thought provoking. And, in fact, Schweitzer et al. (2005, 2007), as well as other researchers (Armitage and Solliday 2020; Bailleul et al. 2020), furnish many such examples. Unfortunately, Schweitzer et al. (2013) do not report the absence of iron jackets from their 2005 and 2007 research in their 2013 paper.

One of the most important papers, effectively neutralizing the efficacy of this model, was published by Schweitzer's team a few years later in 2020 (Bailleul et al. 2020) (already discussed above). In this paper, Schweitzer and her team found soft tissues in *cartilage* from an 80-million-year-old duck-billed dinosaur (Figure 1C). Cartilage lacks all of the iron-producing and/or -containing proteins found in bone[9] that act as catalysts in the jacket model. Alluding to the non-oxidative state of the cartilage, the team say (Bailleul et al. 2020, pp.817–818):

> Unlike dinosaur osteocytes that often present a reddish hue due to iron inclusions … *Hypacrosaurus* chondrocytes are transparent … , suggesting a different preservation mode.

Secondly, the presence of a biochemical jacket does not *necessarily* lead to soft tissue preservation over deep time. This is an assumption. It is true, the presence of iron-saturated haemoglobin around tissues does improve preservation substantially. This was confirmed when Schweitzer et al. (2013) immersed extant ostrich vessels into haemoglobin and left them at room temperature for two years. After that period of time, the ostrich vessels were still in excellent condition (although see my discussion above within the 'cross-linking model'). These data certainly work towards a model of tissue preservation that extends over periods of thousands of years, but not millions. In order to present a model for the preservation of tissues over hundreds of millions of years, the researchers would need to take a whole host of other variables into consideration (discussed below).

Thirdly, many of the iron jackets that circumscribed the tissues in Schweitzer et al. (2013) were not fully 'enclosed'. Visible 'holes' in the jackets were obvious at several locations around the tissues (Figure 2A). Yet no data was presented to discuss the limitations such holes would have on preservation. How do we *know* that these holes would not compromise tissue preservation, especially over time frames in the tens of millions of years? The fact that the tissues are still present does not answer the question because the tissues may not, in fact, be tens of millions of years old – the young-age creationist position.

Finally, Wiemann et al. (2018, p.2) also weigh in on the efficacy of this model:

> Such preservation has been attributed to … physical or chemical binding to mineral surfaces [*the 'jacket' model] …, but none of these models provides an explanation for patterns of originally proteinaceous soft tissue preservation in vertebrate hard tissues in deep time. (emphasis mine)*

4. N-heterocyclic polymer or 'scaffold' model

The fourth major model proposed for the preservation of organic tissues over deep time, although having an auxiliary place in previous models, has been pushed into first position with the publication of a paper by Wiemann et al. (2018). (See also Boatman et al. 2019.) At a basic level, this model proposes that original

proteins are transformed, molecule by molecule, by altered sugars and lipids (Figure 2E). These biochemical pathways are not a new discovery and are presently at work within our bodies altering our DNA and proteins into Advanced Glycoxidation[10] End Products (AGEs) and Advanced Lipoxidation End Products (ALEs).

Simplistically, sugars and lipids found within our tissues can bind to proteinaceous amino acids using glycoxidation and/or lipoxidation pathways. These 'new' molecules are called AGEs and ALEs, in that they are the 'end products' of these reactions. AGEs and ALEs damage tissues by altering their structure, often causing them to become hard and brittle. It is for this reason that doctors warn humans about consuming AGE- and ALE-rich foods, as these foods are known to promote hardening of arteries (atherosclerosis), liver disease, Alzheimer's, arthritis, kidney failure and high blood pressure.

It turns out that glycoxidation and lipoxidation reactions can occur even after an organism dies.

It turns out that glycoxidation and lipoxidation reactions can occur even after an organism dies. This is called non-enzymatic glycoxidation and lipoxidation because the chemical reaction occurs without the aid of enzymes. In other words, dead tissues can continue to change after death. Given the right conditions, large portions of proteins can be altered by AGEs and ALEs, leaving behind non-proteinaceous scaffolds that resemble the original proteins – much like a mineralised bone; what is left is a three-dimensional representation of the bone, but not the bone itself. In other words, fossil tissues that have been exposed to advanced levels of glycoxidation and/or lipoxidation processes might only contain remnants of the original protein molecule, even though they retain the original organic *shape*.

Like the 'iron jacket' model (model 3) put forward by Schweitzer et al. (2013), this model sounds plausible and is quite persuasive. The major problem with this model, however, is closely related to the problems associated with the jacket model. Although the preservational conclusions associated with AGE and ALE research are robust (Wiemann et al. 2018), the *application* of those conclusions to the preservation of *all* soft tissues over deep time is not. That's because *all* of the dinosaur tissues in this study were *brittle* and lacked elasticity, a property conferred to tissues by *organic* elastin (Figure 2B). These conclusions are confirmed in other studies:

> Some of the biological effects are due to the loss of function of the target proteins under-going the covalent modification, such as in the case of extracellular matrix proteins that lose their elastic and mechanical functions when modified as AGEs/ALEs and in particular, when cross-links are involved … (Vistoli et al. 2013, p.3)

So, although these AGEs and ALEs mimic the *shape* of the original proteins, they do not mimic the *roles* of the proteins they replace. All of the dinosaur samples studied by Wiemann et al. (2018) contained greater than 50% AGEs and ALEs, so this only makes sense. Their fragility is, therefore, diagnostic of protein-wide glycoxidation and/or lipoxidation.

Yet if we find examples of *soft* tissues preserved *without* the aid of glycoxidation and/or lipoxidation, then, like the jacket model proposed above, we are back to square one – how did this happen? And, in fact, this is precisely what we do find. There are multiple examples of soft, transparent tissues liberated from bones that retain almost perfect elasticity (Schweitzer et al. 2005, 2007; Bailleul et al. 2020; Armitage and Solliday 2020; Armitage 2021). These samples are also often clear; a fact that, according to Wiemann et al. (2018), suggests a reducing environment that is *not suitable* for the formation of AGEs and ALEs. All of their samples were a charred colour indicative of oxidation. This dark colour is thus diagnostic for heavily altered proteins (Figure 2B). The clear cartilage from the 80-million-year-old duck-billed dinosaur discussed above (Bailleul et al. 2020) is equally applicable here. Remember that, alluding to the non-oxidative state of the cartilage, the team say (pp.817–818):

> Unlike dinosaur osteocytes that often present a reddish hue due to iron inclusions … *Hypacrosaurus* chondrocytes are transparent … , suggesting a different preservation mode.

In other words, glycoxidation and lipoxidation, and thus AGEs and ALEs, were not involved in the preservation of these soft tissues. This *single* example actually neutralises the applicability of all the haemoglobin/iron-based models discussed above. This is because the tissues were found in cartilage, not bone. Cartilage lacks the calcium, apatite and haemoglobin (and thus the iron) that acts as catalysts in the preservation of soft tissues.

There is also the assumption that AGE- and ALE-affected tissues are actually impervious to microbial metabolism over geological time.[11] This is just assumed, however, and not proven. Yes, these 'non-proteinaceous scaffolds' should impart a greater level of preservation to what is left of the original tissues, but where are the experimental

results that show that these polymers can inhibit microbial degradation for time frames as great as 200 million years? This is especially important because scientific studies have shown that even the most robust polymers can be broken down by microbial metabolism in short time frames (Gu 2003; Thomas et al. 2019).

In summary, all current scientific models seeking to solve the 'puzzle' of soft tissue survival over geological time are found wanting. And this is not a young-age creationist interpretation. Saitta et al. (2019, p.3), themselves unconvinced about the likelihood of soft tissue preservation over deep time, say:

> Reports of dinosaur protein and complex organic structure preservation are problematic for several reasons. Firstly, it remains unclear how such organics would be preserved for tens of millions of years. If endogenous, putative dinosaur soft tissues should contain diagenetically unstable proteins and phospholipids …, vulnerable to hydrolysis …, although the released fatty acid moieties from phospholipids could be stabilized through in situ polymerization into kerogen-like aliphatic structures. At 25°C and neutral pH, peptide bond half-lives from uncatalyzed hydrolysis are too short to allow for Mesozoic peptide preservation, although hydrolysis rates can be decreased through terminal modifications and steric effects on internal bonds … Estimates based on experimental gelatinization suggest that, even when frozen (0°C), relatively intact collagen has an upper age limit of only 2,700,000 years … The youngest non-avian dinosaur bones are 66 million years old; on both theoretical and empirical grounds, it seems exceptional that original proteins could persist for so long.
>
> Furthermore, a long-term trend of protein loss and increasing contamination in ancient organismal remains, such as bone, has been shown … Fossil bones are open systems capable of organic and microbial flux … Such a system might lead not only to the loss of endogenous organics, but also to the influx of subsurface microorganisms that could complicate the detection of any surviving organics, as well as potentially metabolizing them. …
>
> Since there are theoretical and empirical reasons to believe that dinosaur organics are unlikely to persist for tens of millions of years, and given the potential for contamination, we argue that the null hypothesis is that complex biomolecules … recovered from dinosaur bones are not original material, more likely representing recent contamination.

Interestingly, this claim of 'contamination' is not new. When Schweitzer and her team published their original results back in the late 1990s and early 2000s, they were blasted with just such an objection from the secular scientific community. Since then, however, Schweitzer, her team, and many other researchers in this field have been vigilant in their methodology so as to exclude contaminants. See my comments in Figure 1 that convincingly exclude microbial contamination.

Using archaeological proxies as a stepping-stone to preservation over geological time

All of the recent models proposing solutions to the preservation of organic material in dinosaur remains (for example by Schweitzer and Wiemann and their teams) often rely on older studies in biomolecular preservation which based their conclusions on the potential of soft tissues to preserve over *archaeological* time (the near present back to about two million years ago), not *deep* time (for example, Lindahl 1993; Collins et al. 2000; Smith et al. 2001; Buckley and Collins 2011; Allentoft et al. 2012). This makes sense since most soft tissues are found in bones that come from the Upper Pleistocene and the Holocene. Given the kinetics of soft tissue degradation, scientists were interested in how these tissues kept turning up in bones that they thought were sometimes hundreds of thousands of years old. This is why the temperatures targeted in most of these older papers are somewhat specific to *archaeological* palaeotemperatures. As I read through these and other papers, only rarely, and then only briefly, did the authors discuss the applicability of their experiments to the preservation of organic material in *dinosaur* remains. And there is a big difference between the two. Archaeological remains are typically found at or near the Earth's surface where conditions are relatively dry, and where temperatures averaged about 10˚C. In contrast, dinosaurs are conventionally thought to have lived in very hot and humid Mesozoic climates where temperatures may have averaged about 30°C (Spalletti et al. 2003; Preto et al. 2010). These temperatures, although dropping a little after the Cretaceous hothouse, are said to have remained relatively high well into the Palaeogene where Global Mean Surface Temperatures (GMST) still averaged over 20˚C, and even shot up to nearly 30˚C during the Palaeocene-Eocene Thermal Maximum (PETM) (Inglis et al. 2020). Dinosaurs are not thought to have lived during Palaeogene time in the conventional model, but their

fossils, buried in near-surface sediments, would have been exposed to these temperatures for supposedly millions of years before being sequestered in deep sedimentary basins.

This is very important because even slight increases in temperature can have exponentially significant effects on soft tissue preservation. Nielsen-Marsh (2002), for example, drops the preservation potential of collagen from 180,000 years at 10°C to only 15,000 years at 25°C. Similarly, Allentoft et al. (2012), drop the preservation potential of DNA from 131,000 years at 15°C to only 22,000 years at 25°C. This means that the *archaeological* proxies determined for soft tissue preservation in much earlier papers, but later referenced by those papers published in the last six years or so, should not be used as a foundation upon which to add more geological time to the preservation potential of *dinosaur* soft tissues. Doing so could lead the reader to make unwarranted jumps. Consider this quote from Schweitzer et al. (2013, p.2):

> Multiple lines of evidence support the endogeneity of these recovered molecules in Cretaceous specimens, despite hypothesized temporal limits on molecular preservation of less than 1 Myr for proteins and approximately 100 000 years for DNA …

This conclusion gives the impression that earlier 'benchtop' scientific experiments verified soft tissue preservation over hundreds of thousands to perhaps a million years for soft tissues found in dinosaur remains – which, given Mesozoic temperatures, would be inaccurate. I am not saying that there was any intellectual dishonesty on the part of the team, nor that they were trying to pull the wool over the eyes of their readers, but, given the typical 10˚C temperatures associated with prior research, the authors could have added some qualifiers. Saitta et al. (2019, p.3) seem to do this in their assessment of Mesozoic soft tissue preservation:

> At 25°C and neutral pH, peptide bond half-lives from uncatalyzed hydrolysis are too short to allow for Mesozoic peptide preservation …

Of course, things get even more complicated when other variables are added to the mix – variables which are not discussed in earlier papers on preservation. High temperatures, abundant water and the presence of microorganisms are the three greatest threats to soft tissue preservation. Yet according to the secular scientific community, dinosaur remains were not only subject to near-surface temperatures for millions of years, they were then slowly buried to depths of greater than two kilometres (over 6,000 feet) in North American basins where the geothermal gradient increased temperatures to as high as 100°C (Morgan and Scott 2014) and where water was abundant in the form of deep subterranean aquifers.[12]

And what about microorganisms? Saitta et al. (2019, p.21) concluded that microorganisms are actually much more abundant in fossil bones than previously thought:

> More recent microbial colonization of fossil bone will occur as it nears the surface during uplift and erosion in the late stages of the taphonomic process. Furthermore, given that microbes can inhabit the crust kilometres below the surface …, it might be predicted that bone remains a biologically active habitat even when buried hundreds of meters deep for millions of years.

Given these data, is a time frame of around one million years for proteins and a hundred thousand years for DNA even close to the mark for the preservation potential of *dinosaur* soft tissues?[13]

So, how long would dinosaur proteins and DNA last in the humid, hot environments that characterised the Mesozoic? And what happens to these proteins and DNA molecules during their eclectic burial journey into deep sedimentary basins? And what effects do 200 million years of microbial attack, tectonic subsidence, sedimentary diagenesis, earthquakes, groundwater infiltration, changing pH, radioisotope decay and subsequent exhumation have on these proteins and DNA? Well, no one knows because no one has factored these variables into their *experimental* scientific models.

bone remains a biologically active habitat even when buried hundreds of meters deep for millions of years.

CONCLUSION

The conclusions of recent research papers strongly suggest that certain biochemical interactions do inhibit degradation of soft tissues over extended periods of time but extrapolating these results over geological time is unjustified. The most parsimonious explanation for the presence of still stretchy, white-to-transparent, pliable tissues is that they are not millions and millions of years old. This is despite the claim by many anti-creationists who use these recent models to harshly criticise the young-age creationist interpretation without giving any thought to *obvious* shortcomings (see, for example, Buchanan 2015 and Senter 2021). As it turns

out, some scientists continue to have grave doubts about the claims for exceptional preservation of dinosaur soft tissues (Demarchi et al. 2016; Buckley et al. 2017; Saitta et al. 2019). These scientists openly acknowledge the incredible odds of finding soft tissues in dinosaur remains given countless natural mechanisms that should prevent such preservation occurring. Interestingly, many of these scientists seem to be swinging back to a microbial origin for these tissues. Personally, this seems stunning given the many images now published by Schweitzer et al. (2005, 2007, 2013), Wiemann et al. (2018), Bailleul et al. (2020), Armitage and Solliday (2020), and Armitage (2021).[14]

Secondly, given the hot and humid temperatures proposed in conventional Mesozoic climate models, models proposing novel mechanisms should not rely on past experiments of soft tissue kinetics where temperatures were *archaeologically* determined. Quoting from these sources for the purpose of building a bridge to geological time is not appropriate unless the obvious disparity between Mesozoic and Pleistocene temperatures, as well as other palaeoenvironmental factors, is addressed.

Finally, and perhaps more importantly, Christians should recognise that this particular issue is not going to go away. Geological time is a fundamental part of a naturalistic paradigm.[15] Christians believe in a *supernatural* Creator that requires a commitment to faith – 'By faith we understand that the universe was created by the word of God' (Hebrews 11:3, ESV). Even Jesus said, 'If they do not hear Moses and the Prophets, neither will they be convinced if someone should rise from the dead' (Luke 16:31, ESV). The Bible claims that we must 'believe' God, *not* test him, especially when that testing pits our experience with the present against Scripture (scientism). God has created an authoritative source of knowledge (evidence) that must be used as the standard for all other sources of knowledge (e.g., scientific evidence) – the natural realm included. That standard is his Word.

ENDNOTES

1. The DNA was fragmented into pieces, but some of those pieces were still six base-pairs long, and perhaps even longer.
2. A new paper just released this year has actually found original blood clots in a supposedly 300 million-year-old Permian synapsid. See Armitage (2022).
3. Simplistically, functional groups are just a group of atoms that perform a specific 'function' when attached to an amino acid.
4. Also commonly called a condensation reaction.
5. All complex animals have trillions of bacteria, for example in their gut.
6. Original to the organism.
7. Sugar-protein cross-linking is different than metal-catalysed cross-linking but the effects are similar.
8. Only used in the 2007 paper.
9. Especially the long bones crucial to red blood cell production.
10. Also called glycation.
11. Keep in mind that these non-proteinaceous scaffolds still retain large chains of *original*, and thus degradable, amino acids.
12. I suggest similar conditions for other basins on other continents.
13. As a final recourse, most researchers rely on the inviolability of radioisotope dating to expand upon and establish the conclusions of these empirical studies: 'However, evidence from radiometric dating shows that dinosaur fossils are indeed millions of years old' (Senter 2021, p.298). I will be the first to admit that the conclusions drawn from radioisotope dating techniques are difficult to address from a young-age creationist perspective, but they are not conclusive. For Christians, the last word must come from the Scriptures. For unbelievers, however, God's *supernatural* creative act – making a universe in literally six days – will only lead those who reject that revelation to reinterpret the processes involved in that *supernatural* act in terms of millions and billions of years. Think about it: what's the alternative if the universe wasn't created in six days? From a Christian and fully supernatural perspective, certain areas of radioisotope decay must fall into this Creation Week category (Baumgardner 2000; Snelling 2005; Coulson 2020). Evidence in other areas, such as the very topic under discussion, do challenge a commitment to deep time. Another example related to this topic, and actually discussed in a secular paper, is the presence of radiocarbon (^{14}C) in dinosaur soft tissues: 'However, the organic carbon content in the *Centrosaurus* bones was significantly lower than the 82–71 ka Yarnton bovine bone sample known to contain well-preserved (radiocarbon-dead) collagen … TOC [*Total Organic Carbon – KC*] in the *Centrosaurus* bone was not found to be radiocarbon dead …' (Saitta et al. 2019, p.14). Interestingly, the presence of this ^{14}C was used to argue *against* the endogenous origin of the dinosaur soft tissues in question (presently a minority position). It is true, these particular organics may have had a microbial origin (hence the presence of ^{14}C), but, from a young-age creationist perspective, the ^{14}C could also be

original to the dinosaur – such a finding being in full accord with young-age creationism.

14. See also my comments in the caption for Figure 1.

15. For more resources, please go to my website, www.creationunfolding.com, or visit my YouTube channel: www.youtube.com/channel/UCmgBaYvK_E29HT4xH-KAm0tA. Go to YouTube and then search for 'Creation Unfolding'.

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