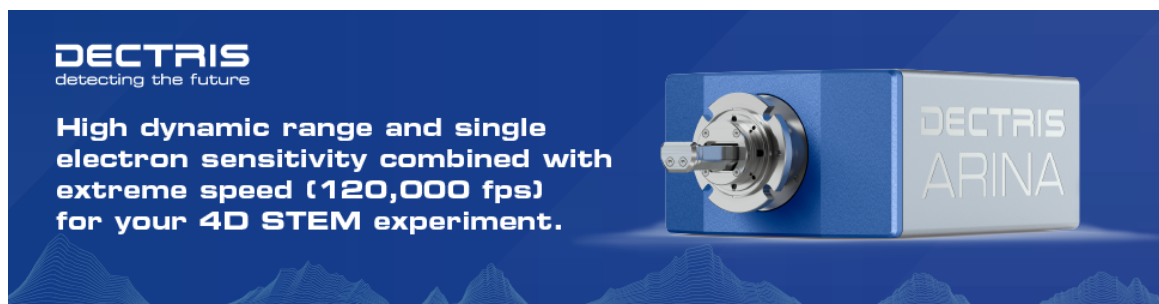


Blood Clots in Dinosaur Bones: Seemingly Permanent Organic/Mineral Interfaces in Once-living Structures

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Blood Clots in Dinosaur Bones: Seemingly Permanent Organic/Mineral Interfaces in Once-living Structures

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Blood clots which occlude microvascular bone canals have been reported in Cretaceous dinosaur remains, including separate individuals of *Triceratops* and *Nanotyrannus* [1–3], and *Dimetrodon*, (an early Permian synapsid) [4] using UV auto-fluorescence microscopy (UVFL). We report newly discovered blood clots in *Edmontosaurus* (another Cretaceous animal), *Camarasaurus*, (a Jurassic sauropod dinosaur), and four Permian early tetrapod individuals *Cacops*, *Eryops*, *Varanops* and *Captorhinus*.

We collected *Edmontosaurus* post-cranial rib, vertebrae and scapula elements at Hell Creek Formation in Glendive, MT, USA, and limb bone elements from *Camarasaurus* at the Morrison Formation in CO, USA. Bones were subjected to fixation in formalin at the site for transport to the lab. Additionally, we secured Permian museum limb specimens of *Cacops*, *Eryops*, *Varanops* and *Captorhinus* from the Oklahoma Sam Noble Museum of Natural History, which were also fixed upon arrival. Bones were rinsed in pure water, air-dried and ground thin sections were made to 40 and 80 micron thickness, Sections were affixed to glass slides and were examined without coverslip under reflected light UVFL for the presence of auto-fluorescing clots within microvascular bone canals.

Clots were imaged in brightfield (Figures 1,3,5,7,9) and UVFL (Figures 2,4,6,8,10). All clots auto-fluoresced brightly under UV illumination indicating massive presence of iron- probably from heme in blood, especially *Cacops* (Figure 2) and *Edmontosaurus* (Figure 6). Despite deep-time environmental factors, (erosion, water infiltration, annual freeze-thaw cycle, radiation) and predation (by bacteria, fungi, microbes, insects and worms), clots adhered tenaciously to bone matrix walls and often completely occluded blood canals for the entire depth of sections. We were stunned that fixation, washing, dehydration and mechanical vibration (during intense grinding for sectioning), did not dislodge clots or even separate them from canal walls. They form tight and uniform junctions against the hydroxyapatite walls of bone they are lodged in. Clots are present in Volkmann canals as well. Intrusion of calcite or silica was unobserved under polarized light.

We also cut rectangular planks of bone and partially decalcified them in EDTA, which exposed solid tubes of clots extending several mm from within the remaining bone (unpublished).

Clots were characterized by embedded, non-fluorescing, dark crystalline structures within the brightly fluorescing pooled iron, (Figures 2,4,6).

We reason that clotted microvascular bone channels are characteristic of disseminated intravascular coagulation, a process initiated by trauma during asphyxiation leading to death [2, 5, 6]. We also note that many histological dinosaur bone studies reveal unreported clots, thus we encourage workers to examine their sections for iron auto-fluorescence response under UVFL. Further work is required to establish the taxonomic and temporal ranges, and prevalence of clots in ancient remains.

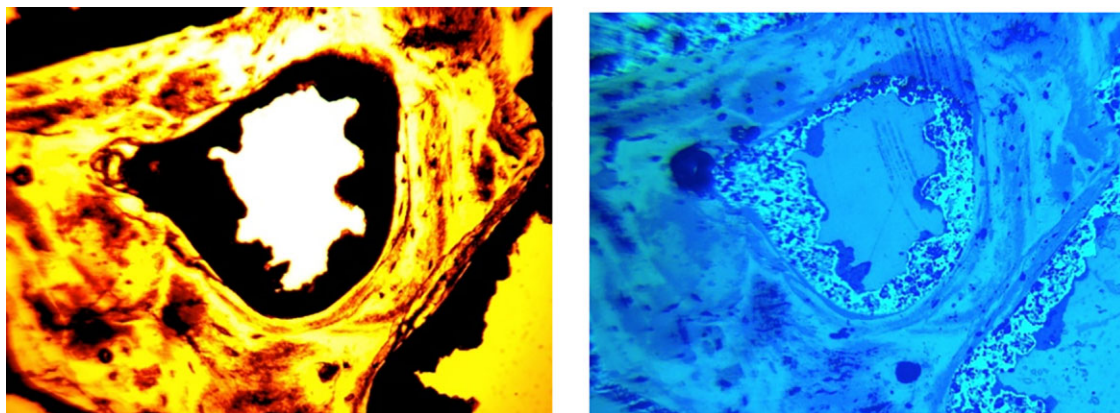


Fig.s 1,2 Brightfield (BF) and UV Fluorescence (UVFL) clots in *Cacops* limb microvascular canal.

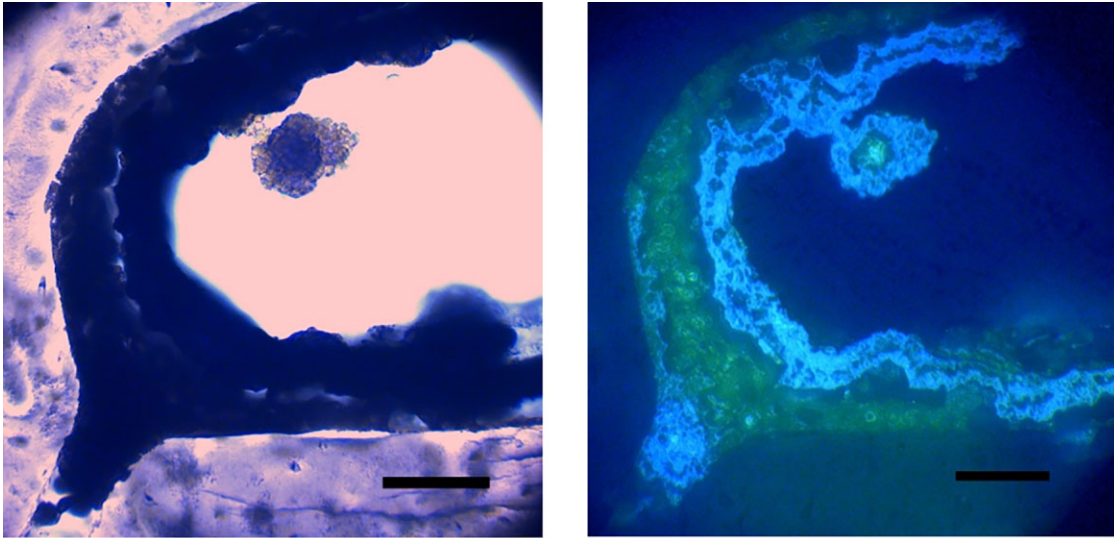


Fig.s 3,4 (BF) and (UVFL) clots in *Camarasaurus* limb microvascular canal.

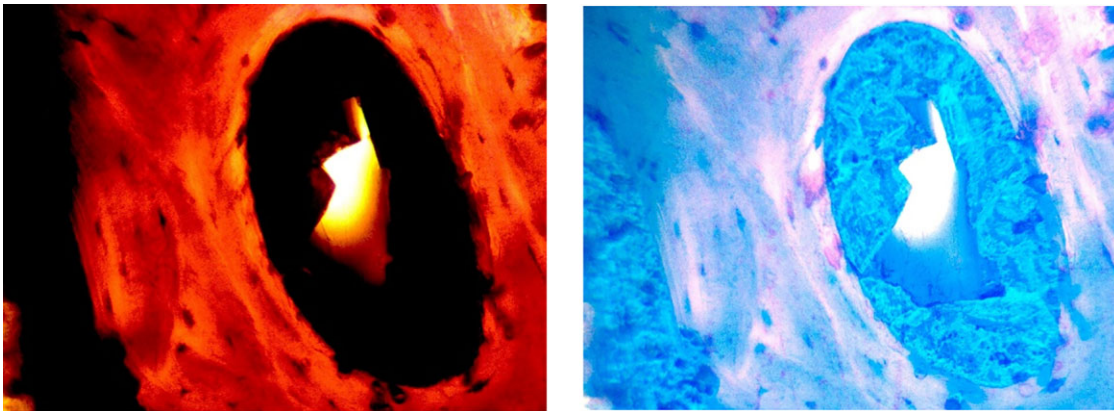


Fig.s 5,6 Brightfield (BF) and UV Fluorescence (UVFL) clots in *Edmontosaurus* limb microvascular canal.

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