

Interactions of different Urolithins with Human Serum Albumin

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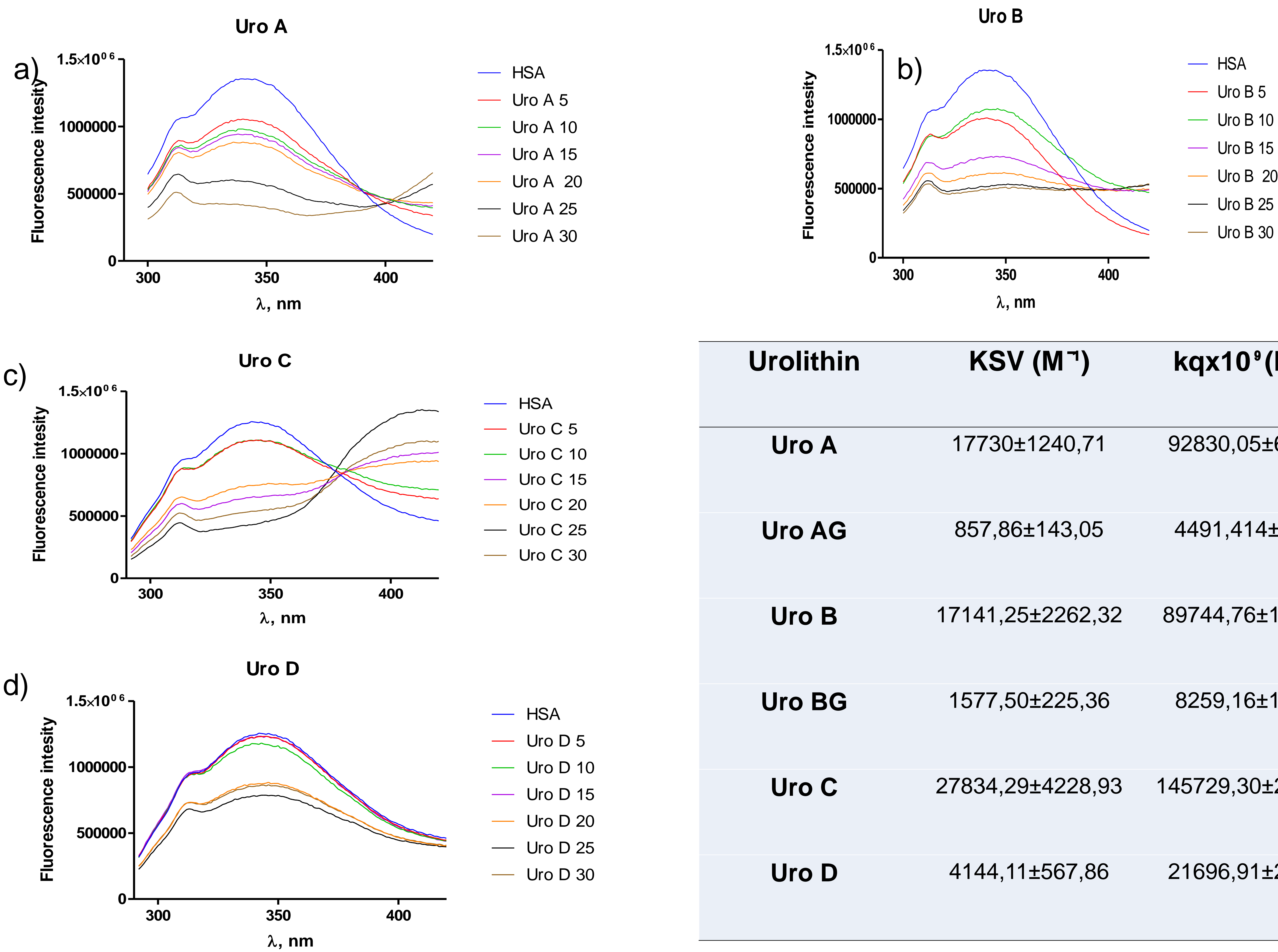
Introduction

Urolithins are metabolites derived from ellagic acid (EA) and ellagitannins (ETs) by gut microbiota after consumption of different ETs. Once produced these catabolites can be absorbed, circulate in plasma and accumulate in urine as glucuronide and sulphate conjugates while aglicones can be directly excreted in faeces. The health effects attributed to urolithins are numerous and diverse, ranging from antimalarial properties to anticancer activities and regulation of gene expression.

Aim

The aim of this work was to study binding of Urolithins: Urolithin-A (Uro A); Urolithin A-glucuronide (Uro AG); Urolithin-B (Uro B) and Urolithin-B-glucuronide (Uro BG) to Human Serum Albumin by fluorescence quenching of protein intrinsic fluorescence.

Results



Urolithin	KSV (M ⁻¹)	kqx10 ⁹ (M ⁻¹ S ⁻¹)
Uro A	17730±1240,71	92830,05±6495,86
Uro AG	857,86±143,05	4491,414±748,95
Uro B	17141,25±2262,32	89744,76±11844,61
Uro BG	1577,50±225,36	8259,16±1179,90
Uro C	27834,29±4228,93	145729,30±22140,99
Uro D	4144,11±567,86	21696,91±2973,09

Figure 1. Emission spectra of HSA (3μM) at λ_{ex} 282 nm (pH 7.0) in the presence of different concentration of increasing concentration of **a)** Uro A; **b)** Uro B; **c)** Uro C; **d)** Uro D

Table 1. Stern-Volmer (KSV) and Bimolecular (kq) Quenching Constants, for the Interaction of URO with HSA.

Conclusion

Higher hydrophobicity increases the binding affinity to HSA and could also be a reason for lower bioavailability of aglicons in sera noted in previous studies since higher rate of phenol binding to proteins is linked to reduced bioavailability.

Acknowledgment

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