Corresponding Author	Iván Alvarez-Twose
List all other authors	Morales-Cabeza C, Bodero-Sánchez J, Caligaris H, Henriques A, Sánchez Muñoz L, Navarro-Navarro P, Jara-Acevedo M, Orfao A, Alvarez-Twose I.
Email-Address	ivana@sescam.jccm.es
Institute/Department	Institute of Mastocytosis Studies of Castilla La Mancha (CLMast), Virgen del Valle Hospital, Toledo, Spain
Address of Institute	Ctra. Cobisa, s/n, 45071 Toledo
Title of Presentation	Mast Cell Clonality, but Not Hereditary Alpha-Tryptasemia, Predicts Severe Food-Induced Anaphylaxis
abstract-hint	
Abstract of Presentation	Background: Hereditary alpha-tryptasemia (HαT) has been proposed as a genetic modifier of anaphylaxis severity, although its role in food-induced anaphylaxis remains uncertain. We investigated the contribution of HαT (and other factors) to the severity of food-induced anaphylaxis. Methods: We retrospectively analyzed 303 consecutive adults with food-induced anaphylaxis evaluated at the Institute of Mastocytosis Studies of Castilla-La Mancha (Toledo, Spain). Anaphylaxis severity was classified using the FASS-5 scale, with severe reactions defined as grades 4-5. A multivariable logistic regression model included HαT, mast cell clonality, comorbidities, age at onset and oropharyngeal symptoms during anaphylaxis. In patients without bone marrow evaluation, the absence of mast cell clonality was assumed when the REMA score was <2 and KIT D816V was undetectable in blood. Results: The cohort included 154 men (51%) and 149 women (49%). HαT status was available in 167 patients. Mast cell clonality was independently associated with severe anaphylaxis (OR 2.96, 95% CI 1.37-6.39; p=0.006). In contrast, HαT (OR 0.44, 95% CI 0.21-0.92; p=0.03) and oropharyngeal involvement (OR 0.26, 95% CI 0.11-0.62; p=0.002) were independently associated with less severe reactions. The remaining factors were not

	associated with severity. Conclusions: In our cohort of patients with food-induced anaphylaxis, mast cell clonality emerged as the strongest independent predictor of severe reactions, whereas HαT was associated with lower risk of severity. These findings suggest that HαT alone should not be considered as a marker of severe food-induced anaphylaxis and underscore the importance of assessing underlying clonal mast cell disorders during risk stratification