Reference


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Reactivity of the rat distal colon to autoantibodies targeting angiotensin type I receptors

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Aim: To describe the reactivity of the rat distal colon to AT1R-Abs and to compare it to that of Ang II.

Introduction: Agonistic IgG (IgG1 and IgG3 subclasses) autoantibodies against the angiotensin II type 1 receptor (AT1R-Abs) have been associated with hypertension, preeclampsia, placental ischemia, renal-allograft rejection and systemic sclerosis. It is though that AT1R-Abs mimic the action of angiotensin II (Ang II) and contribute to the physiopathology of several diseases and the associated complications.

Methods: Male Wistar rats (9–12 weeks of age) were killed by decapitation and strips of the distal colon were mounted in organ baths along their longitudinal axis. Tissues were stretch to 1 g of resting force and isometric responses to AT1R-Abs (25, 50 and 100 mg/dl) obtained from sera of systemic sclerosis and renal-allograft rejection patients and to Ang II (10 ng/M-1 g M) were recorded on a polygraph. The response of Ang II were expressed as % of the response to 125 mM potassium chloride (KCl).

Results: AT1R-Abs caused a long-lasting response. Very often, AT1R-Abs induced an increase in the frequency and amplitude of distal colon spontaneous contractions. Occasionally, AT1R-Abs caused a slight decrease in the resting tone and, more rarely, they caused colonic contraction. The effects of the AT1R-Abs seem to be attenuated by candesartan. The pattern of the response to Ang II was different; Ang II caused a fast developing contraction of the colon with an Emax of 64.37 ± 12.68 (%KCl) and EC50 of 1.22 ± 0.20 nM.

Conclusion: AT1R-Abs change the normal rhythm of spontaneous contractions of the rat distal colon but more studies are necessary to evaluate whether this reactivity is mediated by AT1 receptors. Moreover, Ang II cause a marked AT1 receptor-mediated contraction of the rat distal colon.

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Intestinal colonization by antibiotic-resistant Gram negatives in children

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Aim: This study aims to further the knowledge of antibiotic-resistance in the commensal intestinal flora of children by studying the intestinal colonization by antibiotic-resistant Gram negative bacteria in Portuguese children.

Introduction: Although it is known resistance to antibiotics is a growing problem worldwide, this scenario which constitutes a risk factor for infectious disease is an under-characterized reality in Portugal.

Methods: Faecal samples of 29 healthy children (4 months to 12 years-old) were collected from randomly selected localities of Portugal: Viana do Castelo (n = 8), Porto (n = 6), Braga (n = 14), Leiria (n = 1), from September 2016 to March 2017. Risk factors were assessed by questionnaire, namely antibiotic usage history and direct contact with dependent elders. Isolates were selected by spreading saline suspension (100 µL) on MacConkey agar and MacConkey agar with ampicillin (100 µg/mL), cefotaxime (2 µg/mL), and meropenem (1 µg/mL). Susceptibility profiles to β-lactam and non-β-lactam antibiotics were assessed by disk-diffusion methods according to the EUCAST. Presumptive identification of the isolates was performed with CHROMagar-Orientation culture media.

Results: In a total of 29 isolates (lactose fermenters (n = 22) and lactose non-fermenters (n = 8)), 28 showed resistance to amoxicillin and 13 to amoxicillin with clavulanic acid. Of the 29 children analysed, 17 showed resistance to at least one of the antibiotics studied. Four children were colonized with bacteria resistant to cephalosporins (n = 8), two of which have daily contact with elders.

Conclusion: The results indicate that young children might be an important reservoir of commensals with clinically relevant resistance mechanisms. The clarification of this reality in Portugal could prove essential in the fight against silent dissemination of these threats and persistent infections.

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Is the oral mycobiome of young adults influenced by the delivery mode?

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Aim: To investigate whether the mode of delivery influences the oral yeast colonization in young adults.