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Deranged expression of smooth muscle molecules in colonic tunica muscularis of DD patients

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The pathogenesis of diverticular disease (DD) seems to be a result of a complex interaction between exposure to a low-fibre diet, possible genetic influence, coexistence of other bowel disease and impact of medicine use. These conditions may lead to alterations in colonic pressure and motility [1]. To date, histopatological studies in the field of disturbances in intestinal motility have been mainly focused on the enteric nervous system and interstitial cells of Cajal, which have been found to be both variously affected in different conditions of gut dysmotility. By contrast, although smooth muscle cells (SMCs) are well recognized as the final effectors of the enteric neuromuscular units and have been extensively studied by electrophysiological experiments in colonic motility dysfunction, scarce attention has been paid to the molecular abnormalities of enteric musculature in gut dysmotility, and, in particular, data on SMCs in DD are fairly lacking. Accordingly, the aim of the present study was to evaluate the expression patterns of molecules involved in the contractile functions of SMCs within the colonic *tunica muscularis* from patients with DD. In particular, we examined the expression of the following molecules by immunohistochemistry and image analysis: Cx26 and Cx43, which are prominent components of gap junctions in human colonic SMCs [2], and an array of signaling molecules, which are known to regulate the functions of gap junctions and the contractile activity of SMCs [3,4,5].

The immunohistochemical analysis revealed significant abnormalities in DD samples, concerning both the expression and distribution patterns of most of the investigated molecular factors. Indeed, Cx26, Cx43, PKCps and RhoA appeared to be markedly deranged in all DD colonic samples compared to normal ones, except for α-SMA and pS368-Cx43.

The present results provide the first evidence of an altered pattern of factors involved in smooth muscle contractility at level of *tunica muscularis* SMCs of DD patients. These abnormalities may contribute to the altered motility function, which characterize the colonic *tunica muscularis* of patients with DD.

References

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