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CORRECTION

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Correction: Microenvironment of pancreatic inflammation: calling for nanotechnology for diagnosis and treatment

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Following publication of the original article [1], the cells in Table 1 were not formatted and aligned correctly.

The correct Table 1 is included in this Correction, and the original article has been corrected.

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The original article can be found online at https://doi.org/10.1186/s12951-023-02200-x.

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Table 1 Nanotechnology-based strategies for the diagnosis and treatment of acute pancreatitis

Category	Indicators	Nanoagents (Size)	Animal models	Mechanisms	Modes	Refs.
Inflammatory cells	Macrophage	Lip-DTPA@AuNP(17.2 ± 2.1 nm) M-Gd-NL (120.2 ± 8.5 nm)	Caerulein and LPS-induced AP L-Arginine-induced AP	Gd (III) contrast agents loading of AuNPs and localization to pancreatic tissue for MR imaging	Diagnosis	[47]
		Gd-DTPA-Cy5.5-PsLmAb (50 mm) CO-HbV (~280 nm)	Caerulein-induced AP, L-A-arginine-induced AP –	Targeting macrophages and increasing T1 Imaging ability	Diagnosis	[48]
		G4.5-COOH, G5-OH (5 nm) SPIO-clodronate-liposomes (100–200 nm) MU (175 nm)	Caerulein-induced AP	P-selectin-targeted MR/NIRF bimodal imaging improves spatial resolution and sensitivity.	Diagnosis	[49]
			Sodium taurocholate-induced SAP Caerulein-induced AP	Targeting macrophages and polarizing macrophages toward an M2-like phenotype	Therapy	[56]
				Inhibition of NF-κB nuclear translocation in macrophages and a reduction in inflammatory cells	Therapy	[57]
				Selectively inducing macrophage apoptosis and reducing the release of inflammatory mediators	Diagnosis and therapy	[59]
				Significantly inhibiting the secretion of pro-inflammatory cytokines TNF-α and IL-6 by macrophages	Therapy	[60]
	Neutrophil	tFNAs (~10 nm)	Sodium taurocholate-induced AP	Suppressing the secretion of inflammatory cytokines and regulating the expression of specific apoptotic and anti-apoptotic proteins	Therapy	[67]
		CQ-LPs/TAM-NPs(152.8 ± 2.26/153.2 ± 3.05 nm)	Caerulein and LPS-induce SAP	CQ in combination with TAM syner-gistically promoted iNOS/IDO expression	Therapy	[68]
		NNPs/CLT (61.4 ± 2.8 nm、156.8 ± 2.3 nm、303.7 ± 1.3 nm)	3% Sodium taurocholate-induce AP	Significantly downregulating the levels of serum amylase and pancreatic myeloperoxidase elevant pro-inflammatory cytokines	Therapy	[74]
Oxidative stress and ROS		CAPE-loaded-NL (309 ± 54 nm) RA-EMP (4.703 ± 0.114 nm)	L-Ornithine-induced AP	Modulating Nrf2 and NF-κB Signaling	Therapy	[83]
			L-Arginine-induced AP	Suppressing the effects of oxidative stress and proinflammatory cytokines	Therapy	[84]
		NC (82 ± 5.4 nm)	Caerulein-induced AP	Upregulation of Nrf2, SOD1 and NQO1, downregulating the iNOS, p65-NF-κB, Hsp27 and Hsp70	Therapy	[87]
		NY (159 ± 7.5 nm)	Caerulein-induced AP	Reducing mitochondrial and ER stress via modulation of Nrf2-NFκB pathway	Therapy	[88]

Table 1 (continued)

Category	Indicators	Nanoagents (Size)	Animal models	Mechanisms	Modes	Refs.
		Pbzyme (~ 110-nm)	Caerulein-induced AP	Inhibiting TLRs/NF-κB signaling pathways and scavenging ROS	Therapy	[17]
		MoSe2-PVP NPs (119.39 ± 13.94 nm) MoSe2@PVP NSs (86.278 ± 11.82 nm)	Caerulein-induced AP	Mimicking CAT, SOD, POD, GPx and eliminating a variety of ROS	Therapy	[89]
			Caerulein-induced AP	Mimicking the intrinsic multi-enzyme antioxidant activities of CAT, POD, GPx and SOD to scavenge ROS and RNS	Therapy	[90]
		Nano-Se (20–60 nm)	L-Arginine-induced AP	Anti-inflammatory, antioxidant and pro-apoptotic actions	Therapy	[91]
		CA-NPs (50–90 nm)	L-Arginine and gamma radiation-induced AP	Down-regulating NLRP3, NF-κB and ASK1/MAPK signal pathways and reducing malondialdehyde and caspase-3 levels.	Therapy	[92]
Enzymes	Lipase	Gd-DTPA-FA (–)	L-Arginine-induced AP	Upon enzymatic hydrolysis by lipase, the fat-soluble Gd-DTPA-FA is converted into a water-soluble Gd-DTPA complex, resulting in the changes of the signal intensities observed with MRI in vitro	Diagnosis	[96]
	Proteolytic enzymes	BRSNPs (268.65 ± 6.5 nm)	L-Arginine-induced AP	Inhibiting NF-κB pathway and activating the Nrf2/HO-1 pathway	Therapy	[97]
		LCNPs (89–127 nm)	AP	Extending the circulation half-life of the model peptide compound somatostatin	Therapy	[98]
	PLA2	MΦ-NP (L&K) (~ 100 nm)	Caerulein-induced AP Choline-deficient ethionine (CDE) diet-induced AP	Effectively inhibiting PLA2 activity and PLA2-induced pancreatic injury	Therapy	[99]
pH		Porous COS@SiO_2 nanocomposites (~ 110 nm)	Caerulein and LPS-induced SAP, L-arginine-induced SAP	Activating the Nrf2 signaling pathway to inhibit oxidative stress and reduce the production of NF-κB and NLRP3 and the release of inflammatory factors	Therapy	[105]
			L-Arginine-induced AP	Dramatically enhancing gene transfection efficiency showing high targeting efficiency in pancreas	Therapy	[106]
		Ca-CQ-pDNA-PLGA-NPs (~ 100 nm) FA-SF-NPs (186 nm)	Biliopancreatic duct ligation- induced AP	Suppressing the inflammation and oxidative stress	Therapy	[107]
Multi-targeting		TMSN@PM (~ 142 nm)	L-Arginine-induced AP	Scavenging the excess ROS, degrading, and releasing manganese ions for enhanced magnetic resonance imaging	Diagnosis and therapy	[13]

Reference

1. Liu L, Zhang Y, Li X, Deng J. Microenvironment of pancreatic inflammation: calling for nanotechnology for diagnosis and treatment. J Nanobiotechnology. 2023;21(1):44.

Publisher's Note