AUTHOR CORRECTION

Author Correction: Chemoprevention of Colorectal Cancer in High-Risk Patients: from Molecular Targets to Clinical Trials

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In the original version of this article, which published in *Current Colorectal Cancer Reports*, Volume 13, Issue 3, June 2017, the references in both Tables 1 and 2 were captured incorrectly. The tables provided are now correct.

The authors apologize to the readers for this error.

The online version of the original article can be found at <https://doi.org/10.1007/s11888-017-0364-7>

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Table 1 Molecular target in CRC chemoprevention

	Molecular target	Effect	Ref
FAP			
NSAIDs <i>Sulindac</i>	COX inhibition	Inhibition of PGE2 and inhibition of cell proliferation	[13]
	Beta-catenin down-regulation	Beta -catenin proteasome degradation and caspase activation; growth inhibition	[15]
	PPAR δ inhibition	Suppression of transcriptional activity	[16]
	RXR α inhibition	Activation of the death receptor-mediated apoptosis	[17]
	Inhibition of NF- κ B traslocation to the nucleus	Inhibition of tumor invasion suppressing miRNAs (miR-10b, miR-17, miR-21, miR-9)	[18]
<i>Sulindac</i> + <i>DFMO</i>	ODC and COX inhibition	Suppression of tumor growth	[31]
<i>Phosphosulindac</i> + <i>DFMO</i>	ODC inhibition without PGE2 alteration	Inhibiting cell proliferation and inducing apoptosis	[32]
<i>Sulindac</i> + <i>erlotinib</i>	EGFR inhibition	Inhibition of COX-2	[34••]
<i>Aspirin</i>	COX-1, COX-2 binding and acetylation	Reduced levels of PGE2	[36, 37••]
	PPA2A inactivation	Reduction of cytoplasmatic beta-catenin	[39]
<i>NO-ASA</i>	WNT signaling inhibition	Growth suppression and proapoptotic stimulation	[42, 43]
<i>Coxibs</i>	COX-2 inhibition	Reduction of conversion of arachidonic acid to prostanoids including PGE2	[54]
Omega-3 PUFAs	COX-2 inhibition	Reduction of PGE2 formation and increased level of anti neoplastic PGE3 series	[60]
	NF- κ B level decrease	Reducing inflammation	[62]
	Beta-catenin down-regulation	WNT pathway inhibition	[63]
	Decreasing Lipid peroxidation	Reducing inflammation	[63]
Curcumin	NF- κ B inactivation	Reducing inflammation	[65]
	COX-2 inhibition	Reduction PGE2, reduction of nuclear beta-catenin accumulation	[65]
LS			
NSAIDs <i>Aspirin</i>	Increase steady state level of MMR proteins	Induced apoptosis in MMR proficient and cell cycle arrest in MMR deficient cells	[68]
<i>NO-ASA</i>	MSI suppression	Increase microsatellite stability	[69]
<i>Mesalamine</i>	Activates replication checkpoint in MMR proficient and deficient cells	Slow down DNA replication and improve DNA replication fidelity	[73]
	Reduce mutations at TGFBR2 and ACVR2	Improve replication fidelity	[74]
IBD			
Mesalamine	Direct inhibition of COX-2 and indirect inhibition of TNF- α and IL-1 β	Reduce epithelial turnover, promotes apoptosis	[56]
	PPAR- γ stimulation	WNT/b-catenin inhibition	[57]
	Reducing formation of frameshift mutation	Reduce MSI	[58]
Omega-3	Reduction of beta catenin traslocation	Reduction of cell proliferation in early colorectal carcinogenesis	[111]
	Suppression of Notch signaling pathway	Reduction of cell proliferation	[111]
	Modulating IL-10/SOC3 axis	Decrase STAT3 activation	Unpublished data
UDCA	Hyperphosphorylation of ERK1/ERK2 kinase	Inhibition of proliferatory effects of Irs-1 gene	[115]
	Inhibition of apoptosome formation mediated by PI3K, MAPK or cAMP pathway	Reduced deoxycholic-induced apoptosis	[116]
	Interact with EGFR and caveolin-1	Enhances suppression of MAPK and cell growth	[117]
	Suppression of c-Cbl activity	Increased EGFR degradation	[117]
	Inhibition of COX-2, inducible NO synthase, cyclin D and E-cadherin in aberrant crypt foci of the colon	Reduced cell proliferation and ACF development	[118]
	Reducing expression of inducible NO synthase and TNF- α	Reduction in multiplicity of colonic adenocarcinoma	[119]

Table 2 Chemoprevention of CRC: relevant in vivo and clinical studies

	In vivo and clinical studies	Ref
FAP		
NSAIDs <i>Sulindac</i>	Sulindac 320 ppm treatment in Pirc rat determines a 62% tumor reduction	[20•]
	Sulindac treatment for 6 months in FAP patient, determines a reduction in epithelial cell proliferation and polyps regression in duodenum and rectum.	[21]
	Sulindac treatment at 150 mg twice a day for 9 months in FAP patients determines a 44% decrease in mean number of polyps and a 35% reduction in their mean diameter	[22]
	Sulindac treatment at 75 or 150 mg twice a day in FAP patients for 48 months does not prevent the development of adenoma	[29]
<i>Sulindac + DFMO</i>	Sulindac + DFMO treatment in ApcMin/+ mice determines an 80% reduction in numbers of tumors	[31]
<i>Phosphosulindac + DFMO</i>	Phosphosulindac 100 mg/Kg + DFMO 2% in drinking water treatment in ApcMin/+ mice reduces the 90.2% of the number of intestinal tumors	[32]
<i>Sulindac + Erlotinib</i>	Sulindac + erlotinib treatment for 6 months in FAP patients reduces the polyp burden by 71.2% and the number of duodenal polyps	[34••]
<i>Aspirin</i>	Lifetime continual exposure of Aspirin in ApcMin/+ mice delayed the onset of intestinal neoplasia	[40]
	Aspirin 25 mg/Kg daily treatment for 4 weeks in ApcMin/+ mice have no effect on tumor number and size	[41]
	Aspirin 600 mg daily treatment in FAP patients with intact colon for 17 months does not lead to reduce the polyps number (CAPP-1)	[47]
<i>NO-ASA</i>	Intrarectal treatment of ApcMin/+ mice with NO-Aspirin reduces the 60% of the number of polyps	[44]
<i>Coxibs</i>	Celecoxib treatment in ApcD ⁷¹⁶ mice determines a 86% reduction of polyps	[54]
	Celecoxib treatment at 800 mg or 200 mg daily for 6 months in FAP patients determines a 28% and 11% reduction in mean polyps number and 30% and 14% reduction in polyp burden, respectively	[55]
	Celebrex 800 mg + DFMO 0.5 g/m ² daily treatment for 6 months in FAP patients is not superior to Celebrex alone	[59••]
Omega-3	EPA-FFA 2.5% or 5% treatment for 12 weeks in ApcMin/+ mice determines a 71% and 78% reduction in polyps number, respectively	[63]
	EPA FFA 2g daily treatment for 6 months in FAP patients determines a 22% reduction in polyp number and a 29% reduction in sum of polyp diameters	[64]
Curcumin	Curcumin 0.1% on diet of ApcMin/+ mice determines a 64% reduction of tumor numbers	[65]
	Curcumin 2000 ppm + DIM 250 ppm for 8 months in Pirc Rat diet determine a 50% reduction in colon tumors	[20•]
	Curcumin 480 mg + Quercetin 20 mg 3 times/day for 6 months in FAP patients diet who underwent colectomy with IRA or IPAA, determined a 60.4 % reduction in polyp number and a 50.9% reduction in polyp size	[66]
LS		
NSAIDs <i>Aspirin</i>	Aspirin 600 mg daily treatment on LS patients for 2 years determines a reduction in cancer incidence (CAPP-2)	[72]
<i>NO-ASA</i>	ASA and NO-ASA treatment in MMR-deficient cell lines determines a dose-dependent suppression up to 67% of MSI sequences after 19 to 20 weeks of treatment	[69]
	ASA 400 mg/Kg and NO-ASA 72 mg/Kg treatment in Msh2flox/floxVpC mice increase the life span by 18 % to 21%	[70]
<i>Mesalamine</i>	Mesalamine treatment for 43 weeks in Msh2loxP/loxP Villin-Cre mice determines a 25% reduction in tumor incidence	[75•]
IBD		
Mesalamine	Mesalamine treatment at 75, 150, 225 mg/Kg in AOM-DSS mice reduces the number and burden of dysplastic lesions	[99]
	5-ASA treatment in UC-patients have a protective role for CRC transformation and for CRC/dysplasia transformation	[100,101]
	5 years treatment with 5-ASA in UC-patients have no effect on dysplasia or on low grade dysplasia	[102]
	Mesalamine treatment in IBD patients have no protective effect on neoplastic transformation	[103]
	5-ASA treatment for 12 months in IBD patients have no effect in neoplastic transformation	[104, 105]
Immunomodulators	AZA/6-MP treatment in UC-patients have no effect on the development of neoplastic transformation in the colon	[109]
	Thiopurine in IBD patients is associate with a significant decrease risk to develop advanced neoplasia	[110]
Omega-3	EPA-FFA treatment for 47 days in CAC mice determines a reduction in polyps incidence, multiplicity and size	[112••]
UDCA	UDCA treatment in CAC/PCS patients determines a 10-40% reduction in cancer development	[120, 121]
	UDCA treatment at high dose for long use in UC/PCS patients determines a 4 times increase risk to develop CRC	[122]