Supplement Table S2. Association of metabolites (C2, serotonin, phosphatidylocholine) with other gastrointestinal malignancies; esophageal, stomach/gastric (GC), liver/ hepatocellular carcinoma (HCC), colon/colorectal (CRC) PubMed. 2015-2023

cancer	C2	serotonin	Phosphatidylcholine PC aa C34:1

esophageal squamous cell carcinoma (ESCC)	down-regulated carnitines in ESCC patients. PMID: 29631075		de novo synthesis of phosphatidylcholine (PC) in esophageal cancer MCTS [56] altered FA profiles (phosphatidylcholine) in plasma of the patients with squamous EC during neo-adjuvant chemoradiotherapy abnormal FA metabolism in cancer; altered synthesis de novo, b-oxidation, desaturation, and elongation [57] metabolomic alterations associated with ILF2 and ILF3 in ESCC tissues enriched phosphatidylcholine biosynthesis, fatty acid metabolism pathways, involved in the oxidation of fatty acids [58] enrichment of fatty acid metabolism in EAC pathogenesis progressive alterations (NE-BE-EAC) with increasing levels of phosphatidylcholine changes in phospholipid metabolism during EAC development altered significantly phosphatidyl-choline metabolism in ESCC [59] Phosphatidylcholine metabolism dysregulated serum of ESCC patients [60] significantly different serum levels of metabolites between patients with and without hematological or renal side effects

			higher level of phosphatidylcholines linked to hematological toxicity lower serum level of lysophosphatidylcholine linked to nephrotoxicity biomarkers for predicting hematological toxicity or nephrotoxicity after neoadjuvant chemoradiotherapy for ESCC [61] dysregulated lipid metabolism in ESCC patients significant reduction of phosphatidylcholines (PC) in ESCC serum phosphatidylcholines as novel biomarkers for ESCC [62]
stomach/gastric (GC)	higher level of the acylcarnitine: C2 in GC cancer vs of first-degree relatives increase C2 in negative H. pylori-GC. [59]	potent oncogenic effect 5-HT receptor 1D (HTR1D) on GC [64] FLX-induced apoptosis in AGS (Human gastric cell lines) connected with upregulation of death receptors, ROS generation, and activation of ER stress F [65] The expression of 5-HT significantly higher in GC than patients with chronic gastritis ($p < 0.05$). 5-HT partially co-localized with CgA, in gastric mucosa [66]	phospholipids more abundant in patients with early gastric cancer than in healthy controls reduction of the levels of phosphatidylethanolamine (36:2), phosphatidylcholine (32:0), after resected the cancerous lesions serum phospholipids pointed as potential biomarkers for the early diagnosis of gastric cancer [67] Increase of Phosphatidylcholine (34:3) AUC 0.978 phosphatidylcholine (36:1) in the cancerous than in the paracancerous tissues higher in the serum of EGC patients than healthy controls reduction of Phosphatidylcholine (32:0), (AUC 1.0) in the cancerous than in the paracancerous tissues lower in the serum of EGC patients than healthy controls.

			regions associated with EGC different lipid distributions from the paracancerous regions Serum phosphatidylcholine (32:0), (34:3) pointed as potential biomarkers for discriminating between EGC patients and healthy controls [68] changes with the phospholipids (mainly PC) between cancer tissue and nonneoplastic mucosa [69] Serum levels of phosphatidylcholine (PC) (34:2), PC(34:1), PC(36:4), PC(36:3), and PC(36:2) different and linked with pathophysiological states (colorectal cancer, gastric cancer) phospholipids significantly correlated with gender, physiological states, and cancer stages [70]
Liver (HCT) hepatocellular carcinoma (HCC)	upregulated carnitine (C2/C0) HCT vs. DNT [67] impact of lenvatinib therapy in the carnitine system in patients with hepatocellular carcinoma carnitine insufficiency, a common cause of fatigue during the treatment [68]	induction of cell death in hepatocellular carcinoma cell lines, by Fluoxetine (FLX), a selective serotonin inhibitor induction of apoptosis in hepatocellular carcinoma cell line Hep3B [88] association preoperative intra-platelet serotonin (IP5-HT) with early disease recurrence after liver resection high IP5-HT levels (>134ng/ml IP5-HT) linked with an increased incidence of early tumor recurrence, low IP5-HT levels (<73ng/ml IP5-HT) with a higher rate of morbidity [73]	an increase (2-fold) of phosphatidylcholines (PCs) in hepatic disease patients with HBV replication [81] (using of (PET)/x-ray computed tomography (CT) detection of HCC), based on imaging the initial steps of phosphatidylcholine synthesis high levels of saturated phosphatidylcholines in most HCC tumors fatty acid metabolism connected with for phospholipid membrane synthesis. imperfect diagnostic sensitivity of PET/CT identification of lipogenic tumors, connected with metabolic heterogeneity across HCC and a

		regulation of liver cancer cell steatosis, cells survival with serotonin potential promotion of liver carcinogenesis by activation of Notch signaling and autophagy enhanced cancer cell proliferation/survival and drug resistance by serotonin. upregulation of the expression of lipogenic proteins and increase of steatosis in liver cancer cells with serotonin treatment [74] serotonin, essential for HSC survival and activation higher synthesis and accumulation in males than in females promotion of HCC carcinogenesis in Serotonin-activated HSCs increase in serotonin expression synthesis [75] significant increase of serotonin in early histological stage of HCC development (rat model, post DENA injection) potential role of serotonin as a biomarker used for diagnosis of early stage HCC [76] very good serotonin discriminating power (sensitivity 100%, specificity 92.3%) in early-stage HCC from cirrhosis Serotonin level rapid, sensitive, non-	weaker lipogenic phenotype in some tumors [82] altered lipid profile in human HCC specific lipid metabolic pathways linked with hepatocytes proliferation positive correlation of monounsaturated PC with hallmarks of cell proliferation and hepatic carcinogenesis [83] relatively poor prognosis, a low concentration of the degradation products of phosphatidylcholine connected with subtype S1 of (HCC) [84] disorder of lipid metabolism in (HCC) altered phosphatidylcholine (PC) [85] phospholipids connected with tumour progression metabolic tissue alterations associated with plasmatic modifications elevated concentrations of phosphatidylcholine (PC) 16:0/16:1, PC 16:0/16:0 , PC 16:0/18:1 , low concentrations of lysophosphatidylcholine 20:4 HCC patients phospholipid profiles related to HCC risk in liver cirrhotic patients potential of some phospholipids in predicting HCC patient mortality [86] lipids (with phosphatidylcholine) significantly downregulated lipids were in HCC patients compared to chronic liver disease CLD patients, and in HCC tissues compared to nontumor hepatic tissues [87]

		invasive diagnostic biomarker for the detection of early-stage HCC [77] selective serotonin reuptake inhibitors (SSRIs) associated with a 34% lower risk of HCC, dose-dependent [78] SSRIs, mianserin potentially antitumor activity in HCC [79] impact of Serotonin on the tumorigenesis of HCC serotonin - dependable marker for the diagnosis of HCC (AUC 0.942) for screening for HCC in cirrhotic patients with chronic hepatitis CHC [80]	
colon/colorectal (CRC)	cytotoxic effect of C2, C3, C5, C7 on CRC cell lines (HCT116 and SW480) significant reduction in adherent cells and inhibition of HCT116 cell invasion with C2 [85] Ffar2 deficiency (human colon cancer cell lines; SW480 and HT29) significantly altered profiles of fatty acid metabolites 9 fatty acid oxidation) and gut microbiome, potentially connected with promotion of colorectal cancer development [86] significant reduction of C3, C0, C5:1 before chemotherapy. reduction of C0, and C2 on the second day of chemotherapy (+1 day), increase of C3 [87]	induction of cell death in colon cancer cell line, by Fluoxetine (FLX), a selective serotonin inhibitor antiproliferative effects apoptosis-independent by FLX in colon cancer [65] plasma serotonin level markedly higher in patients with colorectal cancer, compared with healthy controls, and patients with benign colorectal higher plasma serotonin associated with nodal metastasis, disease recurrence, increased risk of mortality and shorter recurrence-free and overall survival higher stages of colorectal cancer	CRC cell line nonmetastatic (HCT116): increase of phosphatidylcholine (PC) 34 : 1, from exosomes from primary cancer patients and nonmetastatic cells (HCT116) compared with healthy person decreased in the metastatic cell line (SW620), and patients, linked to changes in the metastatic cell membrane [96] potential roles of lipid metabolism in the early stage of colorectal carcinogenesis (pre-diagnostic plasma metabolome of adenomas, serrated polyps) higher levels of C36:3 phospha-tidylcholine (PC) plasmalogen associated with lower risk of conventional adenomas [98] lipid metabolism associated with colon cancer prognosis and incidence

		associated with higher plasma level of serotonin polymorphisms of one of the serotonin transporters, SLC6A4, independently linked with shorter overall survival and disease specific survival of patients with colorectal cancer association serotonin with intra-tumor angiogenesis in colorectal cancer and serotonin deficiency PMID: 34289794 diverse roles in CRC, promoting the development of CRC association with more severe signs and symptoms, expansion [92] association of Serotonin (5-hydroxytryptamine, 5-HT) with initiation and progression of CAC colitis-associated cancer (CAC) 5-HT/5-HT2B/TGF-β signaling as a critical tumor-suppressing axis during CAC initiation, promoting cancer progression in the late-stage of CAC [93] reduction of serotonin in colorectal cancer [94] 5-HT signaling correlated with CRC severity	specific lipid composition critical for CSC (cancer stem cell) maintenance. lower phosphatidylcholine (PC; p-18:0/18:1) in CSCs (cancer stem cells) than in bulk cancer cells (BCCs) SCD1 inhibition increased specific PC a in CSCs [99] alterations in lipid composition in cancer tissues compared with normal tissue cancer tissues significantly elevated levels of phospholipids overexpression of genes associated with fatty acid oxidation, and the synthesis of phospholipids reprogramming of lipid metabolism in CRC tissues, for energy production and enhanced synthesis of membrane lipids, necessary for the rapid proliferation of cancer cells [100] reduction of PCs in serum of CRC patients compared with healthy one [101]

		inhibition of the self-renewal of colorectal cancer stem cells (CSCs) and therapeutic efficacy against CRC tumors connected with blocking 5-HT signaling in mice [95]	

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