TABLE 1. Overview of *in vitro* and *in vivo* studies.

Obesity model	Diet, duration	Cancer model	Obese tumor phenotype/ proposed mechanism (keywords)	Ref
Breast cancer				
DIO	HFD (60% kcal from fat), 8 weeks + special diet	T (MMTV-Wnt-1, 5x10^4 cells, orthotopic)	Calorie restriction and rapamycin inhibit mammary tumor growth in postmenopausal obesity	[120]
DIO	HFD (60% kcal from fat)	T (E0771/MDA-MB-231, 1×10^6 cells, orthotopic; LLC/ID8, 1x10^6 cells, subcutaneous)	Obesity-induced expanded adipose stromal cells promote tumor growth	[121]
DIO	Western diet (21% fat), 45 days	CICM (N-methylnitrosourea)	Obesity promotes cancer stemness phenotype via leptin-STAT3-G9a histone methyltransferase signaling axis	[40]
DIO	HFD (36% kcal from fat), 10 weeks	T (Py8119, 1×10^5 cells / E0771, 2×10^5 cells, orthotopic)	Obesity-associated NLRC4 inflammasome activation/ interleukin (IL)-1 signalling promotes breast tumor growth and angiogenesis	[108]
DIO	HFD (60% kcal from fat), 15 weeks	T (86R2 or 99LN, 1.5×10^6 cells, orthotopic)/metastasis assay (99LN, 2×10^6 cells, tail vein)	Obesity-associated inflammation promotes breast cancer metastatic progression	[13]
DIO	HFD (60% kcal from fat), 13 weeks	Metastasis assay (metM-Wntlung, 2,5x10^3, tail vein)	HFD fed mice have reduced overall survival and higher incidence of lung macrometastases	[122]
DIO	HFD (60% kcal from fat), 5-6 months, GEMM (45% kcal from fat), 14 months	T (E0771, 5×10^5 cells + limiting dilution, orthotopic), GEMM (MMTV-TGFα)	A-FABP promotes tumor stemness and aggressiveness through activation of the IL-6/STAT3/ALDH1 pathway	[90]
DIO	HFD (60% kcal from fat), 9 - 11 weeks	T (E0771 or Py230, orthotopic)	Obesity induces hypoxia, neutrophil infiltration and EMT, leading to the faster growing tumors and an increase in metastasis-initiating cells	[103]
DIO	HFD (60% kcal from fat), 12 weeks or 4 days	T (E0771 (1×10^4 - 2×10^5 cells) /PY8119, (2,5 - 5×10^3 cells), orthotopic)	Metabolically activated adipose tissue macrophages link obesity to triple-negative breast cancer	[20]
DIO	HFD (60% kcal from fat), 12 weeks	T (E0771, 5×10^5 cells, orthotopic)	Heparanase regulates macrophage functions to promote tumor progression	[86]
DIO	HFD (60% kcal from fat), 5 weeks	T (E0771, 2 x 10^5 cells, orthotopic)	A non-canonical function of BMAL1 metabolically limits obesity-promoted triple-negative breast cancer	[95]
DIO	HFD (60% kcal from fat), 12 weeks	T (E0771, 5×10^5 cells, orthotopic)	Enhanced resistin secretion in obese mammary adipose issue via FFA/PPARγ/TAZ axis promote breast tumorigenesis	[64]
Colon/small intestine cancer				
DIO	HFD (56.7% kcal from fat), until endpoint	CICM (azoxymethane)	Adiponectin supresses colorectal carcinogenesis under the HFD condition	[56]
DIO, ob/ob, db/db	HFD (56.7% kcal from fat), until endpoint	CICM (azoxymethane)	Leptin acts as a growth factor for colorectal tumors at stages subsuquent to tumor initiation	[32]

TABLE 1 (continued). Overview of *in vitro* and *in vivo* studies.

Obesity model	Diet, duration	Cancer model	Obese tumor phenotype/ proposed mechanism (keywords)	Ref
Colon/small intestine cancer				
DIO	HFD (40% kcal from fat), 8 weeks	GEMM (ApcMin/+)	HFD alter expression of inflammatory markers and increase tumorigenesis	[123]
DIO	HFD (60% kcal from fat), 22 weeks/until endpoint	GEMM (KrasG12Dint)	HFD mediated dysbiosis promotes carcinogenesis independently of obesity	[102]
DIO	HFD (60% kcal from fat), 9-14 months	-	HFD enhances stemness and tumorigenicity of intestinal progenitors	[12]
DIO	HFD (60% kcal from fat), 8 - 10 weeks	T (MC38, 1×10^5 cells, subcutaneous)	Fatty acid metabolism impair T cells infiltration and function and promote cancer growth	[92]
Kidney cancer				
DIO	HFD (60% kcal from fat), 20 weeks	T (RenCa-Luc, 2 × 10^5 cells, orthotopic)	HFD promotes dendritic cell infiltration, which suppress T cell expansion and enhanced tumor growth	[104]
DIO, ob/ob	HFD (60% kcal from fat), 20 weeks	T (CRL-2947-Luc, orthotopic)	Elevated leptin during diet-Induced obesity reduces the efficacy of tumor immunotherapy	[41]
Liver cancer				
DIO, ob/ob	HFD (59% kcal from fat)	CICM (Diethylnitrosamine)	Obesity induced low-grade inflammation promoted the hepatic procarcinogen DEN-induced HCC	[100]
DIO, ob/ob	HFD (60% kcal from fat), until endpoint	CICM (7,12-dimethylbenz(a)anthracene, DMBA)	Obesity-induced gut microbial metabolite promotes liver cancer through senescence secretome	[11]
DIO	HFD (59% kcal from fat), until endpoint	GEMM (MUP-uPA)	ER stress cooperates with hypernutrition to trigger TNF-dependent spontaneous HCC development	[101]
DIO	HFD (60% kcal from fat), until endpoint	CICM (7,12-dimethylbenz(a)anthracene, DMBA)	Gut microbiota promotes obesity-associated liver cancer through PGE2-mediated suppression of antitumor immunity	[124]
DIO	HFD (43% kcal from fat, 40 weeks)	Alb-Cre;Ptpn2fl/fl	Obesity drives STAT-3 dependent hepatocellular carcinoma	[82]
Melanoma				
DIO	HFD (60% kcal from fat)	T (B16,1*10^6, subcutaneous)	Paradoxical effects of obesity on T cell function during tumor progression and PD-1 checkpoint blockade	[109]
Myeloma				
DIO	HFD (60% kcal from fat), 15 weeks	Vk12598 (5x10^5, intrafemorally), 5TGM1 (1x10^6, intravenous)	Acetyl-CoA synthetase 2 - a critical linkage in obesity-induced tumorigenesis in myeloma	[125]
Oral carcinomas				
HFD	HFD (60% kcal from fat), 7 days	T (Detroit-562 cells, orthotopic,)	HFD is able to boost the metastatic potential of CD36+ metastasis-initiating cells to promote cancer metastasis	[88]

TABLE 1 (continued). Overview of *in vitro* and *in vivo* studies.

Obesity model	Diet, duration	Cancer model	Obese tumor phenotype/ proposed mechanism (keywords)	Ref
Ovarian cancer				
-	-	T (coinjection: SKOV3ip1 cells + human adipocytes, subcutaneous)	Adipocytes promote ovarian cancer metastasis and provide energy for rapid tumor growth	[89]
Pancreas cancer				
DIO	HFD (60% kcal from fat)	GEMM (KC, 48Cre-K-rasLSL-G12D/+)	Inflammation (TNFa signalling) and increased fatty acid mitochondrial beta-oxidation links obesity to tumor promotion	[99]
ob/ob, db/db	-	T (Pan02, 2,5*10^5, sub.c)	Altered adipokine milieu and insulin resistance promotes cancer growth and dissemination	[126]
DIO	HFD (60% kcal from fat), >30 days/until endpoint	GEMM (KC, KrasG12D, LSL-Kras/Ela-CreERT and LSL-Kras/PDX1-Cre mice)	Activation of Kras via COX2 leads to pancreatic inflammation and fibrosis and developement of PanINs and PDAC	[98]
DIO	HFCD (40% kcal from fat), 3 months	GEMM (KC, PDX1-Cre;LSL-KRASG12D)	Increase in inflammatory cells, cytokines, chemokines, and stromal fibrosis accelerates early pancreatic neoplasia	[127]
DIO	HFD (60% kcal from fat), 10 weeks	T (Pan02, AK4.4, graft, orthotopic)	PIGF/VEGFR-1 signaling promotes macrophage polarization and accelerated tumor progression in obesity	[97]
DIO, ob/ob	HFD (60% kcal from fat), 10 weeks	T (Pan02, AK4.4, graft, orthotopic)	Obesity-induced inflammation and desmoplasia promote pancreatic cancer progression and resistance to chemotherapy	[42]
DIO	HFD (60% kcal from fat), >50 days/until endpoint	GEMM (KC, KrasG12D/Cre) and T (cells from KPC model)	Depletion LCN2 diminishes ECM deposition, immune cell infiltraton, PanIN formation, and tumor growth	[128]
DIO	HFD (60% kcal from fat), 8 weeks	T (xenograft: 10^5 to 10^6 cells, syngeneic (KPC): 2,5-5*10^4, orthotopic), GEMM (KPC)	Mitochondrial arginase (ARG2) is induced upon obesity and scilencing or loss suppresses tumorigenesis	[94]
DIO	HFCD (40% kcal from fat), >3 months	GEMM (KC,P48+/Cre;LSL-KRASG12D)	HFCD increase cancer incidence, fibrosis and inflammation of KC mice in addition to reducing autophagic flux of PanIN lesions.	[129]
DIO	HFD (60% kcal from fat), 3 months	NA (CK19-RasV12-GFP)	Obesity suppresses cell-competition-mediated apical elimination of RasV12-transformed cells from epithelial tissues	[130]
DIO	HFD (61,6% kcal from fat), 10 weeks	GEMM (KC, KRASG12D/+)	HFD heightens aerobic glycolysis through hyperactivation of oncogenic KRAS	[93]
DIO	HFD (60% kcal from fat), 10 weeks	GEMM (KC, fElasCreERT;KrasLSL-G12D/+, Ptf1aCreERT;KrasLSL-G12D/+)	KRAS reduces expression of FGF21 in acinar cells to promote tumorigenesis in mice on HFD	[131]
Ob/ob, db/db	-	GEMM (KC crossed with Ob/ob)	Endocrine-exocrine signaling drives obesity-associated PDAC	[7]

TABLE 1 (continued). Overview of *in vitro* and *in vivo* studies.

Obesity model	Diet, duration	Cancer model	Obese tumor phenotype/ proposed mechanism (keywords)	Ref
Prostate cancer				
DIO	HFD (42% kcal from fat), 4 months	-	HFD promotes prostatic basal-to-luminal differentiation and accelerates intiation of protstate epithelial hyperplasia originated from basal cells	[132]
DIO	HFD (60% kcal from fat), from 3 weeks until endpoint	GEMM (FVB-Tg(ARR2/Pbsn-MYC)7Key/Nci)	High-fat diet fuels prostate cancer progression by rewiring the metabolome and amplifying the MYC program	[117]

CICM – chemical induced cancer model; DIO – diet-induced obesity; GEMM – Genetically engineered mouse model; HFD – high fat diet; T – transplant model; unless otherwise listed, duration of feeding indicates feeding pattern prior to transplantation or induction of cancer (e.g. administration of agents to induce expression of tumor promoters or exposure to carcinogens).