

Supplementary Materials

FCCLnc: functional characterization of disease/comorbidity-associated long noncoding RNAs

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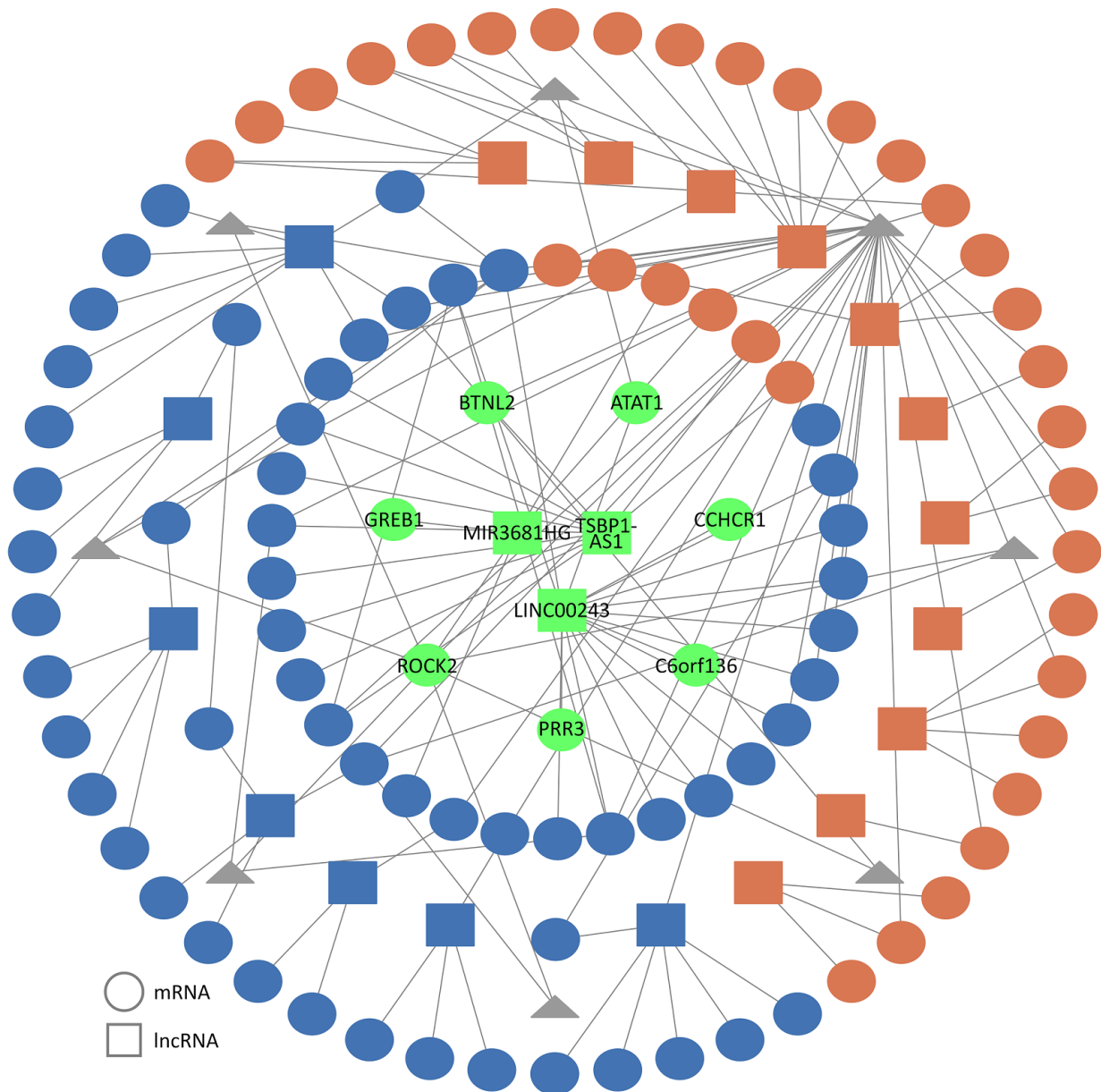
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Supplementary Figure S1. The co-expression network showing the regulation among lncRNAs and mRNAs in schizophrenia patients comorbid with bipolar disorder. Network was constructed using FCCLnc based on dataset GSE78936[1]. lncRNAs/mRNAs solely involved in schizophrenia or bipolar disorder were colored in orange or blue, respectively, while the common disease genes between schizophrenia and bipolar disorder were highlighted in green. Three common lncRNAs (green square) shared by both diseases were identified (including LINC00243, MIR3681HG and TSBP1-AS1).

Supplementary Table S1. KEGG pathways enriched by each of the studied methods together with the literature-reported relevance(s) between the enriched pathways and the studied disease (breast cancer). These enrichment results were based on the dataset TCGA-BC collected from The Cancer Genome Atlas[2]. All p-values were calculated using hypergeometric test, and only the pathways of p-value < 0.05 were enriched. The pathway classes were defined by KEGG database. The information of literature-reported relevance was fully referenced.

Pathway Class	Pathway Name (KEGG ID)	Methods Enriched (p-value)	Literature-reported Relevance(s) between the Enriched Pathway and the Studied Disease
Amino Acid Metabolism	Valine, leucine and isoleucine biosynthesis (hsa00290)	FCCLnc (3.5E-02)	VARS2 protein in valine, leucine, and isoleucine biosynthesis pathway is a prognostic factor for survival in patients with early breast cancer[3]
Carbohydrate Metabolism	Amino sugar and nucleotide sugar metabolism (hsa00520)	GADEA (4.0E-02)	Has not been reported so far
Carbohydrate Metabolism	Citrate cycle or TCA cycle (hsa00020)	GADEA (1.6E-02)	Citric acid cycle is closely associated with the anthracycline cardiotoxicity in the patients with breast cancer[4]
Carbohydrate Metabolism	Glyoxylate and dicarboxylate metabolism (hsa00630)	FCCLnc (3.4E-02)	Glyoxylate and dicarboxylate metabolism pathway gives an insight into the mechanism of aggressive breast cancer[5]
Cell Growth and Death	Apoptosis (hsa04210)	FCCLnc (3.9E-02)	Cancer cell apoptosis, especially the P53-miR-191-SOX4 regulatory loop, is key in the survival of breast cancer cells[6]
Cell Growth and Death	Cell cycle (hsa04110)	FCCLnc (1.2E-10)	Cell cycle pathway is reported to be essential for the hormone-receptor positive breast cancer (the luminal subtype)[7]
Cell Growth and Death	Oocyte meiosis (hsa04114)	FCCLnc (1.1E-02) GADEA (4.4E-02)	Oocyte meiosis pathway was found to be highly susceptible to the triple-negative breast cancer (TNBC)[8]
Cell Growth and Death	Signaling pathway p53 (hsa04115)	FCCLnc (5.0E-04)	Inactivation of p53 by the ways of mutations or other mechanism is a prominent feature of all subtypes of breast cancer[9]
Cellular Community in Eukaryote	Tight junction (hsa04530)	FCCLnc (3.1E-02) GADEA (2.6E-02)	Chromatin remodeling protein BRM regulates the transcription of tight junction proteins in breast cancer metastasis[10]

Circulatory System	Vascular smooth muscle contraction (hsa04270)	FCCLnc (2.4E-02) GADEA (4.9E-03)	Has not been reported so far
Endocrine System	Adipocytokine signaling pathway (hsa04920)	GADEA (1.0E-03)	Adipocytokine and its affiliated pathway play an important role in the development of postmenopausal breast cancer[11]
Endocrine System	GnRH signaling pathway (hsa04912)	GADEA (1.6E-02)	GNRH pathway was restrained in differential expression of proteins in SP cells (rare population of BCSCs) compared to non-SP cells[12]
Endocrine System	Insulin signaling pathway (hsa04910)	GADEA (1.8E-10)	Dysregulation of the insulin-like growth factor-1 receptor pathway was found to correlate with breast cancer progression[13]
Endocrine System	Melanogenesis (hsa04916)	FCCLnc (4.1E-02) GADEA (3.0E-02)	MDA-7 that is mediated by melanogenesis-associated transcription factor down-regulates DICER in breast cancer cells[14]
Endocrine System	Progesterone-mediated oocyte maturation (hsa04914)	FCCLnc (2.9E-02)	Progesterone-mediated oocyte maturation were significantly associated, suggesting that abnormal oogenesis occurs in breast cancer[15]
Energy Metabolism	Nitrogen metabolism (hsa00910)	GADEA (2.5E-02)	Nitrogen metabolism were identified as particularly associated with all three breast cancer cell lines after PARP inhibition[16]
Energy Metabolism	Oxidative phosphorylation (hsa00190)	FCCLnc (1.1E-02)	Oxidative phosphorylation (OXPHOS) was frequently indicated to be up-regulated in breast cancer[17]
Energy Metabolism	Sulfur metabolism (hsa00920)	GADEA (2.1E-02)	Sulfur metabolism was found to be involved in the doxorubicin-resistant breast cancer cells[18]
Excretory System	Aldosterone-regulated sodium reabsorption (hsa04960)	GADEA (3.6E-02)	Has not been reported so far
Excretory System	Vasopressin-regulated water reabsorption (hsa04962)	FCCLnc (4.9E-02)	Vasopressin-regulated water reabsorption was found significantly related to the KLK5 reconstituted expressing in breast cancer cells[19]
Glycan Biosynthesis and Metabolism	Glycosphingolipid biosynthesis (hsa00601)	FCCLnc (3.3E-02)	Several lncRNAs involved in the progression of breast cancer via participating in glycosphingolipid biosynthesis pathway[20]

Immune System	Antigen processing and presentation (hsa04612)	FCCLnc (2.1E-11)	Antigen processing and presenting molecules are highly expressed in breast cancer metastasis[21]
Immune System	B cell receptor signaling pathway (hsa04662)	GADEA (1.8E-03)	B-cell receptor pathway is found to be dysregulated in triple negative breast cancer (TNBC)[22]
Immune System	Chemokine signaling pathway (hsa04062)	FCCLnc (2.3E-02) GADEA (4.4E-02)	CXCR4 triggered by chemokine activates chemokine signaling pathway in hormone-receptor positive breast cancer (the luminal subtype)[23]
Immune System	Cytosolic DNA-sensing pathway (hsa04623)	GADEA (4.0E-04)	Cytosolic DNA-sensing pathway was suggested to be taken as a guide for future research in breast cancer metastasis[24]
Immune System	Fc epsilon RI signaling pathway (hsa04664)	GADEA (7.0E-03)	Has not been reported so far
Immune System	Fc gamma R-mediated phagocytosis (hsa04666)	FCCLnc (5.0E-02)	Fc gamma R - mediated phagocytosis is speculated to substantially influence the function of calcium in breast cancer cells[5]
Immune System	Hematopoietic cell lineage (hsa04640)	FCCLnc (8.1E-04) GADEA (1.2E-04)	Modulation of hematopoietic cell lineage is reported to prevent the bone metastasis in breast cancer[25]
Immune System	Intestinal immune network for IgA production (hsa04672)	FCCLnc (8.0E-03) GADEA (1.9E-02)	Discovered pathways are related to intestinal immune network for IgA production, showing a key role of immune system in BD development[26]
Immune System	Leukocyte trans-endothelial migration (hsa04670)	GADEA (1.2E-02)	Up-regulated genes in breast cancer patients were particularly involved in leukocyte trans-endothelial migration[27]
Immune System	Natural killer cell mediated cytotoxicity (hsa04650)	FCCLnc (1.7E-12) GADEA (4.9E-11)	Breast cancer cells are sensitized to the natural killer cell-mediated cytotoxicity by proteasome inhibition[28]
Immune System	Toll-like receptor signaling pathway (hsa04620)	GADEA (1.2E-03)	Toll-like receptor 4 (TLR4) signaling pathway plays a crucial role in breast cancer[29]
Lipid Metabolism	Alpha-linolenic acid metabolism (hsa00592)	GADEA (1.4E-02)	Identified pathways related to alpha-linolenic acid metabolism were found to be closely associated with breast cancer[30]

Lipid Metabolism	Arachidonic acid metabolism (hsa00590)	GADEA (5.3E-03)	Arachidonic acid metabolite is proposed to be a novel therapeutic target in breast cancer metastasis[31]
Lipid Metabolism	Ether lipid metabolism (hsa00565)	GADEA (4.1E-02)	Has not been reported so far
Lipid Metabolism	Glycerolipid metabolism (hsa00561)	FCCLnc (1.1E-02)	A metabolic map of the breast cancer metabolome demonstrated a marked increase in purine and glycerolipid metabolism[32]
Lipid Metabolism	Glycerophospholipid metabolism (hsa00564)	GADEA (3.8E-03)	Glycerophospholipid metabolism is reported to be related to the metastasis phase of breast cancer[33]
Lipid Metabolism	Linoleic acid metabolism (hsa00591)	GADEA (3.2E-02)	Breast tumors is associated with changes in invasiveness and survival in linoleic acid metabolism form a factor in increasing cancer incidence[34]
Nervous System	Long-term depression (hsa04730)	GADEA (3.0E-02)	Breast cancer bears considerable morbidity and mortality is well known to increase the risk of major depression[35]
Nervous System	Long-term potentiation (hsa04720)	GADEA (3.2E-02)	Has not been reported so far
Nervous System	Neurotrophin signaling pathway (hsa04722)	GADEA (2.4E-02)	Neurotrophin signaling pathway plays an important role in the lapatinib-resistance of breast cancer through EMT pathway[36].

Supplementary Table S2. GO terms enriched by each of the studied methods together with the literature-reported relevance(s) between the enriched GO terms and the studied disease (breast cancer). These enrichment results were based on the dataset TCGA-BC collected from The Cancer Genome Atlas[2]. All p-values were calculated using hypergeometric test, and only the GO terms of [adjusted p-value](#) < 0.05 were enriched. The information of literature-reported relevance was fully referenced.

GO Term (GO ID)	Methods Enriched (adjusted p-value)	Literature-reported Relevance(s) between the Enriched GO Term and the Studied Disease
Striated muscle contraction (GO0006941)	FCCLnc (1.11E-30)	TPM1 gene plays essential role in and functions specifically for striated muscle contraction[37], and it has been reported to be significantly upregulated in human breast cancer cell lines[38]
Protein-DNA complex subunit organization (GO0071824)	FCCLnc (1.20E-30)	Abnormalities in the core human SWI/SNF components BAF170 are critical in the development of breast cancer as assessed by 21 breast cancer cell lines[39]
Skeletal muscle contraction (GO0003009)	FCCLnc (1.30E-30)	Exercise contributes to recovery from breast cancer treatment by resetting the sympathetic tone of lymphatic vessels and propelling lymph flow by skeletal muscle contraction[40]
Regulation of muscle contraction (GO0006937)	FCCLnc (1.10E-27)	Suppression of the transformed phenotype of breast cancer by tropo-myosin-1, while tropo-myosin has been known to function in regulation of muscle contraction[41]
Chromatin remodeling at centromere (GO0031055)	FCCLnc (1.20E-27) GADEA (3.69E-02)	Chromatin remodeling at centromere and DNA amplification were identified in a total of 940 primary breast tumors and 39 breast cancer cell lines[42]
Lymphocyte activation (GO0046649)	FCCLnc (3.10E-26)	Exercise improves the immune functions by increasing lymphocyte activation in patients with breast cancer following treatment[43]
Muscle contraction (GO0006936)	FCCLnc (3.30E-26)	Changes in shoulder muscle size and activity following treatment (muscle contraction) are closely associated to the development of breast cancer[44]
Chromatin assembly or disassembly (GO0006333)	FCCLnc (3.40E-26) GADEA (1.60E-02)	Chromatin assembly or disassembly were differentially expressed in BRCA1mut/+ epithelium relative to BRCA1+/+ epithelium in breast cancer cell lines[45]
Chromatin assembly (GO0031497)	FCCLnc (1.20E-25)	Chromatin assembly was identified and reported to be among the differentially expressed pathways in breast cancer cell lines[46]
Actin-mediated cell contraction (GO0070252)	FCCLnc (1.10E-24)	The suppression of genes involved in the actin mediated cell contraction process is commonly found in cancer, especially the ionizing radiation in breast cancer[47]

Defense response (GO0006952)	FCCLnc (1.40E-24)	Nrf2-mediated defense response is upregulated in the popular MCF-7/DOX cells (which belongs to one typical type of breast cancer cell lines)[48]
Regulation of defense response (GO0031347)	FCCLnc (2.10E-24)	Genes deregulated in blood of breast cancer patients are found to be closely related to the functional process of defense response[49]
Interferon-gamma-mediated pathway (GO0060333)	FCCLnc (2.60E-24)	Interferon-gamma is identified to exert antiproliferative effects on various tumor cells including breast cancer and melanoma[50]
Cellular protein complex assembly (GO0034622)	FCCLnc (2.70E-24)	Attenuation of full-length CSMD1 expression on T47 cells confirmed that endogenously expressed CSMD1 inhibition of the membrane attacked the complex assembly at the level of C7[51]
Adaptive immune response (GO0002250)	FCCLnc (2.80E-24)	Genetic variants in the adaptive immune response pathway are closely associated with breast cancer risk, which appears to differ by ancestry groups, menopausal status and ER status[52]
Muscle structure development (GO0061061)	FCCLnc (1.10E-23)	Muscle structure development is identified to be associated with the biological process of anomalies in individual breast cancer[53]
Nucleosome organization (GO0034728)	FCCLnc (1.30E-23)	In breast cancer cells, a global reconfiguration of nucleosome-depleted regions at distal regulatory elements coupled with a substantial reorganization of the cancer methylome was observed[54]
Cell activation (GO0001775)	FCCLnc (1.40E-23)	Smoothed (SMO) activates breast cancer stem-like cell and promotes tumorigenesis and metastasis of breast cancer[55]
Regulation of immune response (GO0050776)	FCCLnc (1.60E-23)	High immune response and low macrophages were a hallmark of C3, and the patients had a better event-free survival than patients, characterized by low immune response[56]
Multicellular organismal movement (GO0050879)	FCCLnc (1.10E-22)	Tea-shirt zinc finger homeobox 3 (TSHZ3), which was associated with multicellular organismal movement, is novel tumor suppressor gene in breast tumors[57]
Lymphocyte mediated immunity (GO0002449)	FCCLnc (1.20E-22)	Blockage of LILRB1 can restore the cytotoxicity function of NK cells, which was associated with the regulation of lymphocyte mediated immunity in triple negative breast cancer[58]
Regulation of lymphocyte mediated immunity (GO0002706)	FCCLnc (1.30E-22)	Blockage of LILRB1 can restore the cytotoxicity function of NK cells, which was associated with the regulation of lymphocyte mediated immunity in triple negative breast cancer[58]
Negative regulation of epigenetic gene expression (GO0045814)	FCCLnc (1.70E-22)	Agents targeting ER and HER2 were found to be associated with negative regulation of gene expression, and tamoxifen and trastuzumab have been the most extensively used therapeutics for breast cancer[59]
Natural killer cell mediated immunity (GO0002228)	FCCLnc (1.80E-22)	CISH mice were resistant to breast cancer metastasis in vivo, and this was intrinsic to NK cell activity which was associated with natural killer cell mediated immunity[60]

Innate immune response (GO0045087)	FCCLnc (1.10E-21)	Activation of STING-dependent innate immune pathway is responsible for chemokine production in response to DNA damage, resulting in an inflammatory microenvironment in DDRD breast tumors[61]
Protein heterodimerization activity (GO0046982)	FCCLnc (1.20E-21)	Heterodimerization of Her2 and TrkB receptors gives breast cancer cells a survival advantage in the brain and that dual inhibition of these receptors may hold therapeutic potential[62]
T-cell activation (GO0042110)	FCCLnc (1.10E-19)	Elevated T-cell activation score is identified to be closely associated with the improved survival of breast cancer patients[63]
Chromatin organization (GO0006325)	FCCLnc (2.00E-19) GADEA (1.89E-02)	There is an obvious connection between the higher-order chromatin organization of the major HLB and its regulation during breast cancer progression[64]
Muscle system process (GO0003012)	FCCLnc (2.30E-19)	Genes correlated with male sexual characteristics are actively involved in the muscle system process in breast cancer[65]
Actin filament-based movement (GO0030048)	FCCLnc (1.00E-18)	GIPC1 KD in MDA-MB231 human breast cancer cells found the key roles of the regulation of actin filament-based movement and JAK-STAT signaling cascade[66]
Leukocyte mediated immunity (GO0002443)	FCCLnc (1.30E-18)	Tumor antigen-induced inhibition of leukocyte adherence was adapted and modified for use in glass test tubes for the study of cell-mediated antitumor immunity to human adenocarcinoma of the breast[67]
Chromatin organization in regulation of transcription (GO0034401)	FCCLnc (1.40E-18)	BRCA1 gene mutations account for about 25-28% of hereditary breast cancer as BRCA1 is included in the category of chromatin organization involved in regulation of transcription[68]
Leukocyte mediated cytotoxicity (GO0001909)	FCCLnc (1.50E-18)	Messenger RNA upregulation of leukocyte mediated cytotoxicity was found to be closely associated with the overall survival and disease-free status in patients with breast cancer[69]
Histone exchange (GO0043486)	FCCLnc (1.60E-18) GADEA (4.89E-03)	Over two hundreds proteo-forms of histone H4 in two breast cancer cell lines were found to be significantly associated with histone exchange[70]
Regulation of cell cycle phase transition (GO1901987)	FCCLnc (1.90E-17)	Significantly delayed regulation of the cell cycle phase transition in human breast cancer cell lines was observed by experimental validations[71]
DNA conformation change (GO0071103)	FCCLnc (2.00E-17) GADEA (2.64E-03)	Methylation modulator genes were identified to be extensively associated with DNA conformation changes, and played a crucial role in aberrant methylation of CpG sites in breast cancer[72]
Response to interferon-gamma (GO0034341)	FCCLnc (2.50E-17)	Different members of the TP53 family can drive the transcription of genes involved in response to interferon-gamma in different breast cancer subgroups[73]
Regulation of immune system process (GO0002682)	FCCLnc (2.60E-17)	Thymosin alpha 1-fc modulates the immune system and down-regulates the progression of breast cancer with a prolonged half-life[74]

Cytokine production (GO0001816)	FCCLnc (1.10E-16)	Avelumab, an IgG1 anti-PD-L1 immune checkpoint inhibitor, triggers NK cell-mediated cytotoxicity and cytokine production against triple negative breast cancer cells[75]
Negative regulation of cell cycle process (GO0010948)	FCCLnc (1.20E-16)	High expression of ED-induced growth arrest, which involved in negative regulation of cell cycle process, was associated with better prognosis in breast cancer patients[76]
Muscle filament sliding (GO0030049)	FCCLnc (1.30E-16)	Downregulation of the differentially expressed genes was involved in the muscle filament sliding in breast cancer patients[77]
Regulation of adaptive immune response (GO0002819)	FCCLnc (1.02E-15)	RelB functions in the regulation of adaptive immune response[78]. High nuclear levels of nuclear RelB was found to be significantly elevated in nuclei from ER- inflammatory breast cancer tissue[79, 80]
Protein heterotetramerization (GO0051290)	FCCLnc (1.30E-15)	Has not been reported so far
Negative regulation of defense response (GO0031348)	FCCLnc (1.30E-04)	Pathway of negative regulation of defense response was identified and reported to be substantially associated with breast cancer[81]
Positive regulation of leukocyte cell-cell adhesion (GO1903039)	FCCLnc (1.30E-04)	Has not been reported so far
Exogenous peptide antigen via MHC class I (GO0042590)	FCCLnc (1.60E-04)	Has not been reported so far
Negative regulation of DNA damage checkpoint (GO2000002)	FCCLnc (1.70E-04)	A cluster of 13 TNBC addiction genes is found to be frequently co-upregulated that includes genes regulating cell cycle checkpoints, and malignant cell selective mitotic genes[82]
Differentiation of leukocyte (GO0002521)	FCCLnc (1.70E-04)	Has not been reported so far
Regulation of system process (GO0044057)	FCCLnc (2.20E-04)	Regulation of system process is among the top pathways in TMX2-11 and TMX2-28, which were breast cancer cells, affected by tamoxifen selection[83]
Myofibril assembly (GO0030239)	FCCLnc (2.20E-04)	Radiation-induced gene signature predicts pathologic complete response in myofibril assembly to neoadjuvant chemotherapy in breast cancer patients[84]
Regulation of response to external stimulus (GO0032101)	FCCLnc (2.40E-04)	Regulation of response to external stimulus is among the top-3 pathways identified to be significantly associated with the developmental mechanism of MCF-7 breast cancer cells[85]
Non-recombinational repair (GO0000726)	FCCLnc (2.40E-04)	Has not been reported so far

Cell cycle G2/M phase transition (GO0044839)	FCCLnc (2.50E-04)	Expression of UBAP2L, which was associated with cell cycle G2/M phase transition, was significantly up-regulated in breast cancer tissues and cell lines[86]
Antigen receptor-mediated signaling pathway (GO0050851)	FCCLnc (2.60E-04)	Co-expressed genes associated with PD-1 in breast cancer patients were found to be mainly enriched in antigen receptor-mediated signaling pathway[87]
Embryonic skeletal system development (GO0048706)	FCCLnc (2.60E-04)	Embryonic skeletal system development is among the top pathways found to be much more significantly associated to breast cancer cell lines than non-tumorigenic human breast epithelial cell lines[88]
Natural killer cell activation (GO0030101)	FCCLnc (2.60E-04)	Natural killer T-cell activation overcomes immunosuppression to enhance clearance of postsurgical breast cancer metastasis in mice[89]
Heart process (GO0003015)	FCCLnc (3.10E-04)	In nickel-induced cDNA library, 6% among the 68 EST-related nickel-induced breast cancer genes were related to heart process and muscle cell development[90]
Natural killer cell lectin-like receptor binding (GO0046703)	FCCLnc (3.10E-04)	Expressions of MICA/B, which are associated with natural killer cell lectin-like receptor binding, were inversely associated with the tumor node metastasis stage[91]
Organ or tissue specific immune response (GO0002251)	FCCLnc (3.20E-04)	Profiling results of 45,000 immune cells from eight breast carcinomas suggest that tumor-resident T-cells are exposed to inflammation response[92]
Skeletal muscle organ development (GO0060538)	FCCLnc (3.40E-04)	Skeletal muscle-derived cytokines (myokines) exert endocrine-like effects on multiple organs in breast cancer initiation and progression[93]
Inflammatory response (GO0006954)	FCCLnc (3.40E-04)	Connexin hemichannels are major players in modulating the inflammatory response[94]. Inflammation is strongly linked to angiogenesis, invasiveness, metastasis, and resistance in breast cancers[94]
Negative regulation of innate immune response (GO0045824)	FCCLnc (3.40E-04)	Has not been reported so far
Positive regulation of cell-cell adhesion (GO0022409)	FCCLnc (3.60E-04)	IGF-I receptor-induced cell-cell adhesion of MCF-7 breast cancer cells requires the expression of junction protein ZO-1[95]
Regulation of cell-cell adhesion (GO0022407)	FCCLnc (3.80E-04)	Simultaneous knockdown of uPA and MMP9 can reduce breast cancer progression by increasing cell-cell adhesion and modulating EMT genes[96]
Response to biotic stimulus (GO0009607)	FCCLnc (3.90E-04)	Has not been reported so far
Regulation of leukocyte differentiation (GO1902105)	FCCLnc (5.50E-04)	Has not been reported so far

Actomyosin structure organization (GO0031032)	FCCLnc (5.60E-04)	Has not been reported so far
Mitotic cell cycle checkpoint (GO0007093)	FCCLnc (6.80E-04)	Reduced abundance of PI3K-C2 α in breast cancer models initially impairs tumor growth but later leads to the convergent evolution of fast-growing clones with mitotic cell cycle checkpoint[97]
Mitotic cell cycle (GO0000278)	FCCLnc (6.80E-04) GADEA (1.20E-03)	Loss of CEP55 sensitizes breast cancer cells to anti-mitotic agents through premature CDK1/cyclin B activation which is associated with mitotic cell cycle[98]
Antibacterial humoral response (GO0019731)	FCCLnc (7.10E-04)	Antibacterial humoral response demonstrated the significantly differential expression in breast cancer cells, which was considered to be essential in breast cancers[99, 100]
Regulation of inflammatory response (GO0050727)	FCCLnc (7.80E-04)	Connexin hemichannels are major players in modulating the inflammatory response[94]. Inflammation is strongly linked to angiogenesis, invasiveness, metastasis, and resistance in breast cancers[94]
Cytokine secretion (GO0050663)	FCCLnc (8.10E-04)	Positive lymph-node breast cancer patients - activation of NF- κ B in tumor-associated leukocytes stimulates cytokine secretion that promotes metastasis via C-C chemokine receptor CCR7[101]
Regulation of leukocyte proliferation (GO0070663)	FCCLnc (8.10E-04)	Has not been reported so far
Translational elongation (GO0006414)	GADEA (8.30E-04)	Eukaryotic elongation factor 2 kinase, which is associated with translational elongation, is an atypical calcium/calmodulin-dependent serine/threonine kinase that promotes breast cancer tumorigenesis[102]
Regulation of catabolic process (GO0009894)	GADEA (8.30E-04)	A novel regulating mechanism of redox homeostasis involving with regulation of catabolic process, and provided a potential candidate for the anti-metastatic therapy of breast cancer[103]
Establishment of protein localization to organelle (GO0072594)	FCCLnc (9.80E-04) GADEA (4.98E-02)	Overexpression of BAX sensitizes breast cancer MCF-7 cells to cisplatin and etoposide, which was associated with establishment of protein localization to organelle[104]
Protein targeting to membrane (GO0006612)	FCCLnc (9.80E-04)	CXCR4 membrane staining was considered a strong prognostic factor for both 10-year metastasis-free- ($p < 0.0001$) and overall survival ($p < 0.0001$) in node-negative breast cancer patients[105]
Nuclear-transcribed mRNA catabolic process (GO0000184)	FCCLnc (9.80E-04)	RPL28 possess ribosome-independent functions, especially in tumorigenesis, immune signaling and development in breast cancers[106]
Co-translational protein targeting to membrane (GO0006613)	FCCLnc (9.80E-04)	CXCR4 membrane staining was a strong prognostic factor for both 10-year metastasis-free- ($p < 0.0001$) and overall survival ($p < 0.0001$) in node-negative but not in node-positive breast cancer patients[105]
Tumor necrosis factor-mediated signaling pathway (GO0033209)	FCCLnc (9.80E-04)	Has not been reported so far

Regulation of lymphocyte differentiation (GO0045619)	FCCLnc (1.00E-03)	Breast cancers with a high number of TILs contained CD8+ T-cells and these CD8+ TRM cells expressed high levels of immune checkpoint molecules and effector proteins[107]
Negative regulation of mediator of immune response (GO0002701)	FCCLnc (1.01E-03)	Has not been reported so far
Regulation of defense response to virus (GO0050688)	FCCLnc (1.03E-03)	Regulation of defense response to virus is among the top GO biology processes enriched with genes that co-express with CD147 in breast cancer cell lines[108]
Cellular component assembly involved in morphogenesis (GO0010927)	FCCLnc (1.09E-03)	Heparanase enzyme promotes normal morphogenesis and possibly pre-malignant changes, which was associated with cellular component assembly involved in morphogenesis, in breast cancer[109]
Metabolic regulation of nucleobase-containing compound (GO0019219)	GADEA (1.20E-03)	Has not been reported so far
Regulation of cell cycle G2/M phase transition (GO1902749)	FCCLnc (1.21E-03)	Apigenin, which control the regulation of cell cycle G2/M phase transition, can be used in breast cancer prevention[110]
Regulation of T cell receptor signaling pathway (GO0050856)	FCCLnc (1.22E-03)	PLAC1 T cell receptor (TCR)-transduced CD8+ T cells efficiently and specifically identified and annihilated the HLA-A2+/PLAC1+ breast cancer cell lines in a lactate dehydrogenase activity assay[111]
Cardiac muscle tissue development (GO0048738)	FCCLnc (1.23E-03)	Has not been reported so far
Negative regulation of lymphocyte mediated immunity (GO0002707)	FCCLnc (1.33E-03)	Has not been reported so far
Regulation of phosphatidylinositol 3-kinase signaling (GO0014066)	FCCLnc (1.36E-03)	Has not been reported so far
Processing of non-coding RNAs (GO0034470)	GADEA (1.57E-03)	Has not been reported so far
Negative regulation of leukocyte cell-cell adhesion (GO1903038)	FCCLnc (1.60E-03)	It is verified that miR-155, which related to ncRNA processing, was a direct target of XIST in breast cancer treatment[112]
Establishment of protein localization to endoplasmic reticulum (GO0072599)	FCCLnc (1.64E-03)	Has not been reported so far
Translational termination (GO0006415)	GADEA (1.82E-03)	Apoptosis factor EI24/PIG8 is a novel endoplasmic reticulum-localized Bcl-2-binding protein which is

		associated with suppression of breast cancer invasiveness[113]
Negative regulation of RNA biosynthetic process (GO1902679)	FCCLnc (1.99E-03)	Has not been reported so far
Sequence-specific double-stranded DNA binding (GO1990837)	FCCLnc (2.00E-03)	Probing the sequence specific modified DNA structural stability and DNA amplification using the breast cancer suppressor gene 1 (BRCA1) of human breast and colon adenocarcinoma cell lines as models[114]
Regulation of cytokine production in immune response (GO0002718)	FCCLnc (2.12E-03)	Bufalin promotes the inflammatory response in vitro and in vivo by increasing COX-2 and IL-8 (cytokines) production in breast cancer cells[115]
Negative regulation of type I interferon production (GO0032480)	FCCLnc (2.16E-03)	IFIX is the newest member of the human HIN family and is often downregulated in breast tumors and breast cancer cell lines[116]
Negative regulation of adaptive immune response (GO0002820)	FCCLnc (2.16E-03)	Has not been reported so far
Organization of the sarcomere (GO0045214)	FCCLnc (2.17E-03)	Has not been reported so far
Sarcoplasmic reticulum lumen (GO0033018)	FCCLnc (2.17E-03)	Has not been reported so far
Tumor necrosis factor receptor superfamily binding (GO0032813)	FCCLnc (2.29E-03)	Tumor necrosis factor receptor-associated death domain protein is involved in the neurotrophin receptor-mediated antiapoptotic activity of nerve growth factor in breast cancer cells[117]
Regulation of cell adhesion (GO0030155)	FCCLnc (2.29E-03)	Retinoic acid induces nuclear FAK translocation and reduces breast cancer cell adhesion through moesin, FAK, and paxillin[118, 119]
Regulation of T-cell differentiation (GO0045580)	FCCLnc (2.31E-03)	Interference SNHG1 could inhibit the differentiation of Treg (regulatory T cells) cells by promoting miR-448 expression and reducing IDO level, thereby impeding the immune escape of breast cancer[120]
DNA-templated transcription initiation (GO0006352)	FCCLnc (2.51E-03)	MiR-129-5p sensitized Her-2-positive breast cancer to trastuzumab by downregulating rpS6. rpS6 always participates in DNA-templated transcription initiation[121]
Positive regulation of leukocyte differentiation (GO1902107)	FCCLnc (2.76E-03)	ER(+) and TNBC breast cancer cell lines were distinctly influenced by the presence of macrophages (a leukocyte cell)[122]
Cellular protein complex disassembly (GO0043624)	GADEA (2.81E-03)	Activity of cellular protein complex disassembly as a functional unit in breast cancer is supported by the co-expression of these molecules in multiple subtypes of human breast tumors[123]

Striated muscle cell differentiation (GO0051146)	FCCLnc (2.92E-03)	TNF α -dependent miRNA circuitry in muscle differentiation and survival pathways in cancer, especially breast cancers[124]
Positive regulation of lymphocyte differentiation (GO0045621)	FCCLnc (3.05E-03)	Breast cancers with a high number of TILs contained CD8+ T-cells and these CD8+ TRM cells expressed high levels of immune checkpoint molecules and effector proteins[125]
Negative regulation of T-cell receptor signaling pathway (GO0050860)	FCCLnc (3.05E-03)	Tumors with negative regulation of T-cell receptor signaling were smaller, more spherical, and had a more heterogeneous texture upon contrast enhancement in invasive breast cancers[126]
Protein localization to membrane (GO0072657)	FCCLnc (3.07E-03)	Overexpression of BAX sensitizes breast cancer MCF-7 cells to cisplatin and etoposide, which was associated with protein localization to membrane[104]
Establishment of protein localization to membrane (GO0090150)	FCCLnc (3.07E-03)	Localization of the human breast cancer resistance protein (BCRP/ABCG) in lipid rafts/caveolae and modulation of its activity by cholesterol in vitro[127]
Synaptic vesicle exocytosis (GO0016079)	FCCLnc (3.09E-03)	Messenger RNA expression of SNIP/p140Cap, which was associated with synaptic vesicle exocytosis, is an favorable prognostic factor in breast cancer[128]
Synaptic vesicle membrane (GO0030672)	FCCLnc (3.09E-03)	Synaptic vesicle membrane protein VAT-1 homolog showing significant differences (p-value < 0.05) between control and treated MDAMB231 breast cancer cells[129]
Synaptic vesicle cycle (GO0099504)	FCCLnc (3.09E-03)	Synaptic vesicle cycle pathway is identified and reported to be substantially involved in the live metastases of breast cancer[130]
Synaptic signaling (GO0099536)	FCCLnc (3.09E-03)	Endocannabinoids are responsible for retrograde synaptic signaling[131]. Suppression of nerve growth factor by endocannabinoids leads to inhibition of human breast cancer cell proliferation[132]
Calcium ion-regulated exocytosis of neurotransmitter (GO0048791)	FCCLnc (3.09E-03)	Has not been reported so far
Response to ionizing radiation (GO0010212)	FCCLnc (3.17E-03) GADEA (1.70E-02)	Histamine increased radiation response of breast cancer cells, suggesting that it can be used as an adjuvant to enhance the efficacy of radiotherapy[133]
Nuclear-transcribed mRNA catabolic process (GO0000956)	FCCLnc (3.25E-03)	RPL28 possess ribosome-independent functions, especially in tumorigenesis, immune signaling and development in breast cancers[106]
Transition between fast and slow fiber (GO0014883)	FCCLnc (3.92E-03)	Has not been reported so far
Interspecies interaction between organisms (GO0044419)	FCCLnc (3.94E-03)	Overexpression of BAX sensitizes breast cancer MCF-7 cells to cisplatin and etoposide, which was associated with interspecies interaction between organisms[104]

Protein localization to endoplasmic reticulum (GO0070972)	FCCLnc (3.94E-03)	Apoptosis factor EI24/PIG8 Is a novel endoplasmic reticulum–localized Bcl-2–binding protein which is associated with suppression of breast cancer invasiveness[113]
Mitochondrial translational termination (GO0070126)	GADEA (3.94E-03)	A role of enhanced mitochondrial ROS release was established, which was related to mitochondrial translational termination, in 3,3'-diindolylmethane-induced p21 up-regulation in breast cancer[134]
Interleukin-12 production (GO0032615)	FCCLnc (3.98E-03)	Mononuclear cells of patients affected by breast cancer have a defective IL-12 production capability while generating higher amounts of IL-10[135]
Macromolecule catabolic process (GO0009057)	FCCLnc (4.22E-03) GADEA (8.32E-03)	Has not been reported so far
Cellular response to oxygen-containing compound (GO1901701)	FCCLnc (4.46E-03)	Has not been reported so far
CD8-positive, alpha-beta T cell activation (GO0036037)	FCCLnc (4.55E-03)	In 7 out of 16 breast cancer patients' lymph nodes, there is a dominant V beta 18 T-cell clonal expansion in the CD8+ T cell subset[136]
DNA repair (GO0006281)	FCCLnc (4.59E-03) GADEA (1.18E-03)	DNA damage response (DDR) involves the activation of repair DNA lesions, and the breast cancer cells sensitive to specific DNA damage response inhibitors[137]
Regulation of cell cycle G1/S phase transition (GO1902806)	FCCLnc (4.68E-03)	Regulation of G1/S transition by PANDARPANDAR/Bmi1/p16(INK4A) axis could serve as novel targets for breast cancer therapy[138]
Chromosome segregation (GO0007059)	FCCLnc (4.68E-03) GADEA (1.30E-03)	MASTL, which causing chromosome segregation errors, is most commonly amplified and overexpressed which correlates with increased chromosome instability in breast cancer[139]
DNA integrity checkpoint (GO0031570)	FCCLnc (4.68E-03)	In breast cancer, several pathways maintaining DNA integrity checkpoint are distinctly altered, including some that have been successfully exploited for therapeutic targeting[140]
Chromosome organization (GO0051276)	FCCLnc (4.90E-03) GADEA (3.50E-04)	Breast tumors and cell lines frequently display major epigenetic instability of the inactive X chromosome, with highly abnormal 3D nuclear organization[141]
DNA strand renaturation (GO0000733)	FCCLnc (5.40E-03)	Reactive oxygen species (ROS) can induce the breakage of DNA strands[142]. PARP-1 as a mechanism to protect DNA from further damage has become a current target of breast cancer drug strategies[142]
Positive regulation of cell activation (GO0050867)	FCCLnc (5.84E-03)	Smoothed (SMO) activates breast cancer stem-like cell and promotes tumorigenesis and metastasis of breast cancer[55]
Antimicrobial humoral immune response (GO0061844)	FCCLnc (5.95E-03)	Antimicrobial humoral immune response mediated by antimicrobial peptide influences immune response genes paly critical roles in human breast cancer[143]

Regulation of skeletal muscle contraction (GO0014819)	FCCLnc (6.22E-03)	Exercise contribute to recovery from the impact of breast cancer treatment by resetting the sympathetic tone of lymphatic vessels, and propelling lymph flow by skeletal muscle contraction[40]
Nucleosome positioning (GO0016584)	FCCLnc (6.33E-03)	Relationship between nucleosome positioning and progesterone-induced alternative splicing in breast cancer cells was revealed[144]
Circulatory system process (GO0003013)	FCCLnc (6.47E-03)	Technological developments for the isolation and analysis of circulating and disseminated tumor cells continue to emerge, creating new opportunities to monitor progression of breast cancer[145]
Mitotic DNA integrity checkpoint (GO0044774)	FCCLnc (7.34E-03)	Genomic integrity is closely guarded by several mechanisms, including DNA damage checkpoints, the DNA repair machinery, and the mitotic checkpoint in breast cancer[140]
Regulation of antigen receptor-mediated pathway (GO0050854)	FCCLnc (7.35E-03)	MTT assay indicated that GNP-NKCT1 reduced proliferation in the estrogen receptor induced ER negative breast cancer cell line (MDA-MB-231)[146]
Negative regulation of cell-cell adhesion (GO0022408)	FCCLnc (7.42E-03)	Restoring E-cadherin-mediated cell-cell adhesion increases PTEN protein level and stability in human breast carcinoma cells[147]
Positive regulation of synaptic transmission (GO0050806)	FCCLnc (7.56E-03)	TAC1, involved in positive regulation of synaptic transmission and expression, is found to occur in breast cancer[148]
Synaptic vesicle localization (GO0097479)	FCCLnc (7.56E-03)	Detection of autoantibodies against the 128-kd synaptic protein (localized in neurons and concentrated at synapses) in patients with this syndrome is considered an indication to find an occult breast cancer[149]
Neurotransmitter transport (GO0006836)	FCCLnc (7.56E-03)	Pathways such as neuropeptide signaling pathway, neurotransmitter transport were enriched with genes that analyzed using the triple negative breast cancer cell line[150]
Calcium-ion regulated exocytosis (GO0017156)	FCCLnc (7.56E-03)	In mammals, miR-7a which was associated with calcium-ion regulated exocytosis, is a widely studied miRNA and extensive studies have shown that it functions as a tumor suppressor in breast cancer[151, 152]
Signal release from synapse (GO0099643)	FCCLnc (7.56E-03)	Has not been reported so far
Cytoskeleton of presynaptic active zone (GO0048788)	FCCLnc (7.56E-03)	Has not been reported so far
Antimicrobial humoral response (GO0019730)	FCCLnc (7.57E-03)	Pathways involved in chemotaxis and antimicrobial humoral response showed significant differences in breast cancer[153]

Chromosome condensation (GO0030261)	FCCLnc (7.69E-03)	Nano-encapsulation of a novel anti-Ran-GTPase peptide for blockade of regulator of chromosome condensation 1 (RCC1) function in MDA-MB-231 breast cancer cells[154]
Negative regulation of antigen receptor-mediated signal (GO0050858)	FCCLnc (7.82E-03)	Has not been reported so far
Homeostasis of number of cells (GO0048872)	FCCLnc (7.92E-03)	Several ECM components and ECM remodeling enzymes are specifically induced in breast cancer or during tissue regeneration while healthy tissues under homeostasis express exceedingly low levels[155]
Heart development (GO0007507)	FCCLnc (7.94E-03)	Manageability and reversibility of trastuzumab-related cardiac complications in a patient who had developed severe heart failure complicated with trastuzumab therapy for breast cancer[156]
Regulation of DNA damage checkpoint (GO2000001)	FCCLnc (8.24E-03)	Mediator of DNA damage checkpoint protein 1 (MDC1) expression as a prognostic marker for nodal recurrence in early-stage breast cancer patients treated with breast-conserving surgery[157]
Regulation of skeletal muscle adaptation (GO0014733)	FCCLnc (8.34E-03)	Exercise training during chemotherapy preserves skeletal muscle fiber area, capillarization, and mitochondrial content in patients with breast cancer[158]
Protein targeting (GO0006605)	FCCLnc (8.74E-03)	Agents that target ER and HER2 which were associated with negative regulation of gene expression, epigenetic, such as tamoxifen that has been extensively used for breast cancer[59]
Regulation of synaptic plasticity (GO0048167)	FCCLnc (8.89E-03)	Higher expression in the adult brain could be associated with a potential role of miRNA in neuron maintenance or in regulation of synaptic plasticity or long-term memory in breast cancer[159]
Positive regulation of hemopoiesis (GO1903708)	FCCLnc (9.04E-03)	Screening for clonal hematopoiesis as a predictive marker for development of therapy-related myeloid neoplasia (t-MN) following neoadjuvant therapy for breast cancer[160]
RNA binding (GO0003723)	FCCLnc (9.20E-03) GADEA (1.82E-03)	RNA binding protein RNPC1 inhibits breast cancer cell metastasis via activating STARD13-correlated ceRNA network[161]
Galactose metabolic process (GO0006012)	FCCLnc (9.28E-03)	When the level of galactose was reduced, transactivation defects could be revealed, suggesting that some breast cancer-related mutants can have subtle changes in transcription[162]
Interleukin-12 biosynthetic process (GO0042090)	FCCLnc (9.28E-03)	IL-12, which was associated with interleukin-12 biosynthetic process, induces autophagy in human breast cancer cells through AMPK and the PI3K/Akt pathway[163]
Negative regulation of developmental process (GO0051093)	FCCLnc (9.83E-03)	ASAP1's tags in the metastatic library were found to be 6.7 times more prevalent than in the nonmetastatic library and it had been linked to metastatic activity of breast cancer[164, 165]
Antigen processing and presentation of peptide antigen (GO0048002)	FCCLnc (1.00E-02)	Western blots of NE-treated breast cancer cells confirm that the expression of the antigen-processing machinery proteins TAP1, LMP2 does not change following NE treatment[166]

Type I interferon production (GO0032606)	FCCLnc (1.05E-02)	Type I interferon/IRF7 axis instigates the chemotherapy-induced immunological dormancy in the patients of breast cancer[167]
Negative regulation of chromatin silencing (GO0031936)	FCCLnc (1.06E-02)	FOXA1 repression is associated with loss of BRCA1 and increased promoter methylation and chromatin silencing in breast cancer[168]
Negative regulation of biosynthetic process (GO0009890)	FCCLnc (1.06E-02)	SALL4, which was associate with negative regulation of biosynthetic process, expression has been detected in breast cancer[169]
Cell-cell adhesion (GO0098609)	FCCLnc (1.12E-02)	Changes in cell-cell adhesion depend on the level of phosphatidylinositol 4-phosphate [PI(4)P] in the Golgi apparatus in breast cancer cells[170]
Secretion (GO0046903)	FCCLnc (1.12E-02)	Interleukin-like epithelial-to-mesenchymal transition inducer activity is controlled by proteolytic processing-regulated secretion during breast cancer progression[171]
Organic cyclic compound catabolic process (GO1901361)	FCCLnc (1.15E-02)	Overexpression of BAX sensitizes breast cancer MCF-9 cells to cisplatin and etoposide, which was associated with organic cyclic compound catabolic process[104]
Regulation of myeloid dendritic cell activation (GO0030885)	FCCLnc (1.16E-02)	The number of myeloid dendritic cells was markedly reduced in breast cancer patients, and exposure of dendritic cells to spermine promoted their activation, and impaired their function[172]
Susceptibility to natural killer cell mediated cytotoxicity (GO0042271)	FCCLnc (1.18E-02)	Blockage of LILRB1 can restore the cytotoxicity function of NK cells, which was associated with lymphocyte mediated immunity in triple negative breast cancer[58]
Cell cycle G1/S phase transition (GO0044843)	FCCLnc (1.18E-02)	DSCAM-AS1 participates in the cell cycle G1/S phase transition is a promising clinical therapeutic target in breast cancer patients[158]
Neuron differentiation (GO0030182)	FCCLnc (1.25E-02)	Neuron differentiation is among the top pathways in functional annotation clustering of hypermethylated genes in HER2+ breast cancer using DAVID bioinformatics tool[173]
Chemical synaptic postsynaptic transmission (GO0099565)	FCCLnc (1.25E-02)	Has not been reported so far
Regulation of cell differentiation (GO0045595)	FCCLnc (1.28E-02)	LncRNA SNHG1 regulates the differentiation of Treg cells and affects the immune escape of breast cancer via regulating miR-448/IDO[120]
Regulation of hemopoiesis (GO1903706)	FCCLnc (1.31E-02)	Human breast tumors is depicted as highly heterogeneous, with numerous haematopoietic cell-related programs seemingly involved in patient survival[174]
Regulation of calcium ion-dependent exocytosis (GO0017158)	FCCLnc (1.32E-02)	Regulation of calcium ion-dependent exocytosis such as KIF1A and KIF14 have been reported to be responsible for the progression of breast cancer[175]

Negative regulation of leukocyte proliferation (GO0070664)	FCCLnc (1.34E-02)	A significantly greater number of WGA bright lymphocytes, which was associated with negative regulation of leukocyte proliferation in PBL was found in patients with breast cancer[176]
Regulation of DNA metabolic process (GO0051052)	GADEA (1.39E-02)	Horizontal transfer of mtDNA, which was participated in regulation of DNA metabolic process, from EVs leads to endocrine therapy resistance in OXPHOS-dependent breast cancer[107]
Protein catabolic process (GO0030163)	GADEA (1.39E-02)	Has not been reported so far
Regulation of response to biotic stimulus (GO0002831)	FCCLnc (1.40E-02)	Has not been reported so far
Positive regulation of gene expression (GO0010628)	FCCLnc (1.42E-02)	DNA methylation has a substantial impact on positive regulation of gene expression, affecting the prognosis of breast cancer patients dependent on molecular subtypes[177]
Positive regulation of RNA biosynthetic process (GO1902680)	FCCLnc (1.42E-02)	Expression of METTL3, which was associated with positive regulation of RNA biosynthetic process, was positively related to that of HBXIP in clinical breast cancer tissues[178]
Respiratory burst (GO0045730)	FCCLnc (1.43E-02)	Has not been reported so far
Taurine metabolic process (GO0019530)	FCCLnc (1.45E-02)	Pathways of metabolism of amino acids, organic acids and taurine metabolic process were found to be dysregulated in breast cancer[179]
Protein-containing complex disassembly (GO0032984)	GADEA (1.45E-02)	Has not been reported so far
Negative regulation of cell adhesion (GO0007162)	FCCLnc (1.46E-02)	In basal-A TNBC cells, a major effect of p63 is the negative regulation of cell adhesion, a process important in metastasis and invasion of tumor cells[180]
Regulation of calcium-mediated signaling (GO0050848)	FCCLnc (1.46E-02)	Calcium-based mechanism is established as crucial for executing the cell death process, and suggest that simvastatin with doxorubicin may be an effective regimen for the treatment of breast cancer[181]
Defense response to Gram-positive bacterium (GO0050830)	FCCLnc (1.48E-02)	Defense response to Gram-positive bacterium and transcriptomic pathways analysis of urokinase receptor were associated in breast cancer cells[182]
Regulation of protein catabolic process (GO0042176)	GADEA (1.49E-02)	Novel interaction via expression patterns of EphA10s and EphA10, which associated with regulation of protein catabolic process, promotes the malignant transformation of breast cancer[183]
Metabolic process of non-coding RNA (GO0034660)	GADEA (1.49E-02)	Functions of SNHG15, which associated with the metabolic process of non-coding RNA, have been revealed and found to be essential in breast cancer[184]
RNA polyadenylation (GO0043631)	GADEA (1.49E-02)	An immune-specific APA (RNA polyadenylation) signature in breast cancer is utilized as a prognostic

		marker of early stage breast cancer[185]
Cellular response to prostaglandin stimulus (GO0071379)	FCCLnc (1.55E-02)	Long term use of aspirin and similar agents, which cellular response to prostaglandin stimulus, can decrease the incidence of certain malignancies, including breast cancers[186]
Embryo development ending in birth or egg hatching (GO0009792)	FCCLnc (1.56E-02)	Has not been reported so far
ATP-dependent chromatin remodeling (GO0043044)	GADEA (1.60E-02)	Has not been reported so far
Protein-containing complex assembly (GO0065003)	GADEA (1.60E-02)	Has not been reported so far
T-cell homeostasis (GO0043029)	FCCLnc (1.64E-02)	Persistent fatigue in breast cancer survivors is associated with a chronic inflammatory process involving the T-cell homeostasis[187]
Negative regulation of cell population proliferation (GO0008285)	FCCLnc (1.67E-02)	Progesterone receptor status of proliferating cancer cells was associated with histological grade and recurrence score, and a potent prognostic factor in estrogen-receptor-positive breast cancers[188]
Cellular response to ionizing radiation (GO0071479)	GADEA (1.75E-02)	Clone genic tests and cellular senescence assays was conducted to monitor for radiation-induced effects which observed in breast cancer cells[189]
Mitochondrial translation (GO0032543)	GADEA (1.75E-02)	RB1 deficiency promotes cancer cell proliferation in part by enhancing mitochondrial translation for RB1-deficient Triple-negative breast cancer[190]
Ubiquitin-like protein binding (GO0032182)	GADEA (1.79E-02)	Has not been reported so far
Regulation of cell cycle (GO0051726)	GADEA (1.82E-02)	Regulation of cell cycle and tumor suppressor role of ERR β in breast cancer cells have been discovered as essential and critical[191]
Protein folding (GO0006457)	FCCLnc (1.82E-02)	Huaier could inhibit the proliferation of breast cancer cells through modulating linc00339/miR-4656/CSNK2B, which associated with protein folding signaling pathway[192]
Endomembrane system organization (GO0010256)	GADEA (1.83E-02)	Has not been reported so far
Regulation of ryanodine-sensitive calcium-release channel (GO0060314)	FCCLnc (1.85E-02)	Has not been reported so far

Response to interleukin-12 (GO0070671)	GADEA (1.89E-02)	Balance between pro-angiogenic factors (TP and VEGF) and IL-12 (response to interleukin-12) in the breast cancers was reported[193]
Cleavage of messenger RNA (GO0006379)	GADEA (1.89E-02)	A hammerhead ribozyme cleaves human telomerase (hTERT) mRNA (mRNA cleavage) in the human breast tumor line MCF-7 was discovered[194]
Regulation of mRNA 3'-end processing (GO0031440)	GADEA (1.89E-02)	Has not been reported so far
Metabolic process of messenger RNA (GO0016071)	GADEA (1.89E-02)	Has not been reported so far
B-cell activation (GO0042113)	FCCLnc (2.03E-02)	Molecular mechanism of DBC1 (deleted in breast cancer 1) inhibition of alternative NF-κB activation in suppressing B cell activation was defined[195]
Regulation of prostaglandin secretion (GO0032306)	FCCLnc (2.04E-02)	Protein p53 is a negative regulator of aromatase in the breast and its inhibition by PGE2 (prostaglandin E2) which provides a novel mechanism for aromatase regulation in obesity and breast cancer[196]
Positive regulation of skeletal muscle tissue development (GO0048643)	FCCLnc (2.08E-02)	Skeletal muscle-derived cytokines (myokines) exert endocrine-like effects on multiple organs, including the mammary glands, which showed the role of macrophages and T-cells in breast cancer progression[93]
Viral gene expression (GO0019080)	FCCLnc (2.27E-02)	Several viral systems have drawn attention for the treatment of breast cancer with impressive results, which has been considered as promising therapeutics in clinical development[197]
Spindle localization (GO0051653)	GADEA (2.28E-02)	Spindle cell type of MBC with myoepithelial features exhibits a higher frequency of PIK3CA mutation than other types of metaplastic or basal-like breast cancer[198]
Ubiquitin binding (GO0043130)	GADEA (2.28E-02)	Elevated PPIL2 inhibited EMT and breast cancer invasion by interacting with the SNAI1 to enhance its ubiquitin binding[199]
Proteasomal protein catabolic process (GO0010498)	GADEA (2.28E-02)	ECM29 mRNA levels, which associated with proteasomal protein catabolic process, are predictive of relapse-free survival in breast cancer patients[200]
DNA-dependent DNA replication maintenance of fidelity (GO0045005)	GADEA (2.28E-02)	A compelling relationship exists between high CDK2AP1 mRNA expression, which is related to DNA-dependent DNA replication maintenance of fidelity, and lower TNM classification of breast cancer[201]
Skeletal muscle adaptation (GO0043501)	FCCLnc (2.32E-02)	Exercise training during chemotherapy preserves skeletal muscle fiber area, capillarization, and mitochondrial content in patients with breast cancer[158]
COPII-coated ER to Golgi transport	FCCLnc (2.33E-02)	Anks1a acts as an important regulator of COPII-mediated EphA2 ER export. The Anks1a ankyrin repeat

vesicle (GO0030134)		domain binds EphA2 (target of breast cancer) and causes it to accumulate at sites of ER exit[202]
Positive regulation of gene expression, epigenetic (GO0045815)	FCCLnc (2.33E-02)	Matrix metalloproteinase 9 (MMP-9) gene expression regulation by intragenic epigenetic modifications in breast cancer[203, 204]
Regulation of DNA-dependent DNA replication (GO0090329)	FCCLnc (2.36E-02)	Both PARP and DNA-PK are activated by DNA strand breaks and are implicated in DNA replication and transcription, which were associated with breast cancer[205]
Energy derivation by oxidation of organic compounds (GO0015980)	FCCLnc (2.36E-02)	Reactive oxygen species (produced by metabolic by-products of energy production) can induce DNA breakage, and PARP-1 as a molecule protecting DNA becomes a target of breast cancer[142]
Positive regulation of mitochondrion organization (GO0010822)	GADEA (2.44E-02)	Has not been reported so far
Regulation of multi-organism process (GO0043900)	FCCLnc (2.47E-02)	Has not been reported so far
Positive regulation of NLRP3 inflammasome assembly (GO1900227)	FCCLnc (2.58E-02)	Has not been reported so far
Hexose catabolic process (GO0019320)	FCCLnc (2.65E-02)	Expression and function of hexose transporters GLUT1, GLUT2, and GLUT5 in breast cancer-effects of hypoxia[206]
Hindbrain development (GO0030902)	FCCLnc (2.66E-02)	Gastrulation brain homeobox 2 (GBX2) involved in mid/hindbrain region identified having played a role in breast cancer development due to its association with the Wnt/ β -catenin pathway[207]
DNA-binding transcription factor binding (GO0140297)	FCCLnc (2.66E-02)	Ets-1, one of the crucial members of transcription factor which share a unique DNA binding domain, knock down in MCF-7 and MDA-MB-231 breast cancer cells showed reduced cell invasion[208]
Telomere organization (GO0032200)	GADEA (2.67E-02)	Telomere length, which associated with telomere organization, could be a valuable breast cancer prognostic marker[209]
Transcription of non-coding RNA (GO0098781)	GADEA (2.67E-02)	Strategies to decrease MIAT, which is related to ncRNA transcription and expression levels can improve sensitivity to therapy in breast cancer[210]
Cranial nerve morphogenesis (GO0021602)	FCCLnc (2.69E-02)	Has not been reported so far
Negative regulation of response to stimulus (GO0048585)	FCCLnc (2.73E-02)	Has not been reported so far

Mast cell mediated immunity (GO0002448)	FCCLnc (2.78E-02)	HER2/neu IgE-sensitized ADMC (adipose-derived mast cells) induced breast cancer cell (SK-BR-3) death through apoptosis[211]
Cell cycle arrest (GO0007050)	FCCLnc (2.82E-02)	Anti-tumor effect of CEP on breast cancer cells and suggests that CEP and cell cycle arrest can be a potential new clinical therapy for breast cancer[212]
Receptor internalization (GO0031623)	FCCLnc (2.82E-02)	EGF induces programmed death ligand 1 (PD-L1) and receptor programmed cell death protein 1 (PD-1) interaction, receptor internalization in breast cancer[213]
Regulation of neurotransmitter transport (GO0051588)	FCCLnc (2.87E-02)	Has not been reported so far
Organonitrogen compound catabolic process (GO1901565)	GADEA (2.96E-02)	Has not been reported so far
Intracellular protein transport (GO0006886)	FCCLnc (3.01E-02) GADEA (3.96E-02)	Role of miR-124-3p in breast cancer development and miR-124-3p/PDCD6 signaling is found, which is related to intracellular protein transport, and therapeutic strategy for advanced breast cancer[214]
Nucleic acid phosphodiester bond hydrolysis (GO0090305)	GADEA (3.01E-02)	Has not been reported so far
Cellular response to fatty acid (GO0071398)	FCCLnc (3.02E-02)	FFA1 and FFA4 agonists, which were associated with cellular response to fatty acid, can inhibit proliferation and migration of prostate and breast cancer cells[215]
Formation of extrachromosomal circular DNA (GO0001325)	FCCLnc (3.07E-02)	PIP gene, which were associated with formation of extrachromosomal circular DNA can be the target of genomic variability processes in breast cancer[216]
Organelle fission (GO0048285)	FCCLnc (3.07E-02)	Upregulation of dynamin related protein-1 (Drp1), which was associated with organelle fission, was accompanied with increased organelle fission markers in breast cancer cells[217]
Meiotic cell cycle (GO0051321)	FCCLnc (3.07E-02)	Knocking down ALDOA blocked breast cancer cells in the G0/G1 phase, which was associated with meiotic cell cycle, under minimized glycolysis[218]
Cellular response to lipid (GO0071396)	FCCLnc (3.07E-02)	Cellular response to lipid, adipocytes, and the processes and pathways in which they are involved, could be effective therapeutic targets for breast cancer[219]
Negative regulation of interferon-beta production (GO0032688)	FCCLnc (3.11E-02)	TR is post-transcriptionally induced by the IFN-beta-all-trans-retinoic acid combination, which is associated with negative regulation of interferon-beta production, in breast carcinoma[220]
Response to interleukin-2 (GO0070669)	FCCLnc (3.11E-02)	NK cell expansion and trastuzumab-mediated increased NK cell killing of breast cancer targets (which is related with response to interleukin-2) in a HER2-specific manner[221]

Response to interleukin-15 (GO0070672)	FCCLnc (3.11E-02)	IL-15 has a role in mammary cancer immunosurveillance, further supporting the potential use of IL-15 as adjuvant in immunological strategies against tumors[222]
Interferon-beta production (GO0032608)	FCCLnc (3.19E-02)	IFN β -hUCMSCs, which was associated with interferon-beta production, inhibited the growth of breast cancer cells through apoptosis[223]
Cranial nerve development (GO0021545)	FCCLnc (3.24E-02)	(BRCA1 germline mutation and expression of the Her2 seem to contribute to an increased rate of development of CNS metastases, which were associated with cranial nerve development[224]
Positive regulation of molecular function (GO0044093)	FCCLnc (3.24E-02)	Endocrine therapy, the mainstay of treatment for hormone-responsive breast cancer involves use of estrogen receptor modulators, which is related to positive regulation of molecular function[59]
Morphogenesis of a branching structure (GO0001763)	FCCLnc (3.24E-02)	Paracrine signaling, which is related to morphogenesis of a branching structure, these interactions play crucial role in mammary gland homeostasis and when deregulated breast cancer[225]
Regulation of autophagy (GO0010506)	FCCLnc (3.24E-02)	Inhibition of the autophagic flux in dormant breast cancer cells leads to the accumulation of damaged mitochondria and reactive oxygen species (ROS), resulting in cell apoptosis[226]
Plasma membrane bounded cell [227]projection cytoplasm (GO0032838)	FCCLnc (3.24E-02)	Has not been reported so far
Cellular response to external stimulus (GO0071496)	FCCLnc (3.34E-02)	Has not been reported so far
Pallium development (GO0021543)	FCCLnc (3.37E-02)	Anti-cancer activity of a novel palladium (II) complex, which was associated with the pallium development pathway, on human breast cancer cells[228]
Signaling cascade of p38MAPK (GO0038066)	FCCLnc (3.47E-02)	Increased activation of p38-MAPK in MCF-7TN-R cells shows that this signaling pathway through activation of NF-kappa B is key route for control of resistance to cell death in breast cancer[229]
Regulation of CD8-positive alpha-beta T-cell activation (GO2001185)	FCCLnc (3.47E-02)	Has not been reported so far
Leukocyte apoptotic process (GO0071887)	FCCLnc (3.47E-02)	Cell sialylation controls Gal-8-mediated cell adhesion, and a central role of either ALCAM (which activated leukocyte cell adhesion molecule) in controlling triple negative breast cancer[227]
Annealing helicase activity (GO0036310)	FCCLnc (3.49E-02)	Annealing helicase activity is reported to be significantly associated with the greatly increased risk of breast cancer[230]
Protein modification by small protein	FCCLnc (3.59E-02)	Pathway of protein modification by small protein conjugation is found to play the important role in breast

conjugation (GO0032446)		cancer[231]
Positive regulation of cytokine in immune response (GO0002720)	FCCLnc (3.60E-02)	Inhibition of leptin-cytokine crosstalk, which was associated with positive regulation of cytokine production involved in immune response, serves as a preventative target of breast cancer[232]
Monosaccharide catabolic process (GO0046365)	FCCLnc (3.60E-02)	High levels of FGFR4 can increase glucose metabolism, which is related to monosaccharide catabolic process, and lead to chemo resistance in breast cancer[233]
Negative regulation of interleukin-12 production (GO0032695)	FCCLnc (3.68E-02)	Negative regulation of interleukin-12 production associated with ERx early transcriptional signature in breast cancer cell line[234]
Cytokine metabolic process (GO0042107)	FCCLnc (3.68E-02)	Gene ontologies included cytokine metabolic process, significantly upregulated and downregulated among AA compared to EA triple negative breast cancer[235]
Cell-cell adhesion mediated by integrin (GO0033631)	FCCLnc (3.68E-02)	Processes whose associated proteins were enriched are related to cell-cell adhesion mediated by integrin in triple negative parental human breast cancer cell lines[236]
Prostaglandin transport (GO0015732)	FCCLnc (3.68E-02)	COX-2, which was associated with prostaglandin transport, was identified statistically overexpressed in breast cancer[237, 238]
Negative regulation of PI3K signaling (GO0014067)	FCCLnc (3.68E-02)	PTEN (phosphatase and tensin homolog deleted from chromosome 10), involved in negative regulation of phosphatidylinositol 3-kinase signaling, expression in breast cancer[239, 240]
Catalytic activity acting on DNA (GO0140097)	GADEA (3.68E-02)	Has not been reported so far
Regulation of cell cycle checkpoint (GO1901976)	FCCLnc (3.68E-02)	Cell cycle checkpoint kinase gene dysfunction in chemotherapeutic drug resistance of triple negative breast cancer cells[241]
Response to steroid hormone (GO0048545)	FCCLnc (3.79E-02)	Enzymes with the provision of additional steroids as hormone sources were associated with response to steroid hormone, in breast cancer cells[242]
Protein modification by small protein conjugation or removal (GO0070647)	FCCLnc (3.84E-02)	Protein modification by small protein conjugation or removal is significantly related to the development of breast cancer[243]
Positive regulation of protein targeting mitochondrion (GO1903749)	GADEA (3.94E-02)	Has not been reported so far
Regulation of regulated secretory pathway (GO1903305)	FCCLnc (4.00E-02)	Enhanced expression of Rab27A gene, which involved in regulation of regulated secretory pathway, plays a key role in promoting the invasiveness of breast cancer[244]

Regulation of synaptic transmission, glutamatergic (GO0051966)	FCCLnc (4.00E-02)	Regulation of synaptic glutamatergic transmission was identified to demonstrate significant relation to breast cancer[245]
Lymph node development (GO0048535)	FCCLnc (4.08E-02)	Malignancy-risk gene signature performed well to show the statistically significant association with metastasis and lymph node development in breast cancer patients[246]
Activation and regulation of cytolysis (GO0042268)	FCCLnc (4.08E-02)	Has not been reported so far
Adult locomotory behavior (GO0008344)	FCCLnc (4.08E-02)	Adult locomotory behavior was associated with the different molecular subtypes of inflammatory breast cancer[247]
Regulation of membrane potential (GO0042391)	FCCLnc (4.08E-02)	Breast cancer cells, regulation of membrane potential is key to many other processes including determination of membrane potential and the rate of proliferation and apoptosis[248]
Neurogenesis (GO0022008)	FCCLnc (4.08E-02)	Biological phenomenon about cancer related neurogenesis in breast cancer were discovered to be significantly associated with the disease development[249]
Neuron projection extension (GO1990138)	FCCLnc (4.08E-02)	A network diffusion approach to inferring sample-specific function reveals functional changes included neuron projection extension associated with breast cancer[250]
Cell division (GO0051301)	FCCLnc (4.24E-02)	BRCA1 suppression regulates the cell division axis in proliferating and non-transformed human mammary epithelial cells[251]
Response to camptothecin (GO1901563)	GADEA (4.25E-02)	Obtained LCF NCs, which associated with response to camptothecin, were shown to be able to suppress growth of breast cancer[252]
Messenger RNA export from nucleus (GO0006406)	GADEA (4.25E-02)	Has not been reported so far
Cell projection organization (GO0030030)	FCCLnc (4.26E-02)	Significance of the first fifteen annotated functions included cell projection organization in breast cancer cells[253]
Skeletal system development (GO0001501)	FCCLnc (4.26E-02)	It is a powerful modality for skeletal and nodal staging, which is associated with skeletal system development, in breast cancer patients with important impact on therapy[254]
Cytoskeleton organization (GO0007010)	FCCLnc (4.26E-02)	Global transcriptome changes in breast cancer cells following knockdown of AmotP130 identified pathways related with the cytoskeleton and cell motility that involved Rho GTPase family[255]
Negative regulation of T cell proliferation (GO0042130)	FCCLnc (4.32E-02)	CTLA4 49A/G (rs231775), which plays an important role in the negative regulation of T-cell proliferation, was a candidate cancer susceptibility marker for breast cancers[256]

Ion transmembrane transport (GO0034220)	FCCLnc (4.33E-02)	Observed antimigratory effects in breast cancer cells are directly related to the inhibition of Na(+)/K(+) transport, which was associated with ion transmembrane transport[257]
Cellular response to ATP (GO0071318)	FCCLnc (4.46E-02)	Overexpression of RABL6, which involved in cellular response to ATP, plays a role in breast cancer tumorigenesis[258]
Positive regulation of fatty acid transport (GO2000193)	FCCLnc (4.46E-02)	Has not been reported so far
Regulation of protein localization to mitochondrion (GO1903747)	GADEA (4.52E-02)	Has not been reported so far
Vesicle localization (GO0051648)	FCCLnc (4.60E-02)	PLD2 inhibitor treatment inhibited the plasma membrane localization of MT1-MMP while increased its vesicle localization in breast cancer cells[259]
Regulation of chromatin silencing (GO0031935)	FCCLnc (4.80E-02)	FOXA1 repression is associated with loss of BRCA1 and increased promoter methylation and chromatin silencing in breast cancer[168]
Regulation of cofactor metabolic process (GO0051193)	FCCLnc (4.80E-02)	Transcriptional positive cofactor 4, associated with regulation of cofactor metabolic process, promotes breast cancer proliferation and metastasis through c-Myc mediated Warburg effect[260]
Signal release (GO0023061)	FCCLnc (4.80E-02)	Blocking signal release by TSLP without affecting IL-7 function was a potentially interesting option for treating tumors, and TSLP was also a determinant of tumor progression in breast cancer[261]
T-cell migration (GO0072678)	FCCLnc (4.84E-02)	COX2 expression manipulates the immune profile in a syngeneic, and immune-competent, which is associated with T cell migration, mouse model of breast cancer[262]
Negative regulation of T cell mediated immunity (GO0002710)	FCCLnc (4.84E-02)	B7-H4, a co-signaling molecule involved in the negative regulation of T-cell mediated immunity, has been found to be overexpressed in breast cancer[263, 264]
Regulation of icosanoid secretion (GO0032303)	FCCLnc (4.84E-02)	Has not been reported so far
Protein localization to mitochondrion (GO0070585)	GADEA (4.88E-02)	Interrupting mitochondrial functions of G1P3, which associated with protein localization to mitochondrion, could improve clinical outcomes in breast cancer patients[265]
Nerve development (GO0021675)	FCCLnc (4.97E-02)	Breast cancer growth and progression were accelerated following stimulation of sympathetic nerves in tumors, which is related to nerve development[266]
Facial nerve structural organization (GO0021612)	FCCLnc (4.97E-02)	Has not been reported so far

Cellular response to organic cyclic compound (GO0071407)	FCCLnc (4.97E-02)	Cellular response to organic cyclic compounds has an influence in breast cancer, and most genes may be related with viral carcinogenesis[267]
Double-strand break repair (GO0006302)	FCCLnc (4.98E-02)	Breast cancer risk is associated with the genes encoding the DNA double-strand break repair Mre11/Rad50/Nbs1 complex[268]

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