Supplementary material

Somatic evolution of Cancer: A new synthesis

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We give here data and references demonstrating the nearly complete overlap between the pathways, hallmarks and phenomena involved in cancer and wound healing.

Table 1 exemplifies the parallels along with the genes/molecules/pathways/mechanisms that are common to both the processes. This is not an exhaustive list, but an illustrative one.

Table 2 uses a complete catalogue of genes, mutations in which have been causally implicated in cancer, to examine how many of them have evidence for a role in wound healing.

Table 3a and 3b examine the list of genes (coming from two different sources) differentially expressed during various phases of wound healing, for evidence of their involvement in cancers.

Supplementary table 1:

The phenomena or hallmarks of cancer that were once thought to be unique to cancers are now demonstrated to be involved in wound healing, along with their underlying mechanisms.

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| --- | --- | --- | --- |
| Phenomenon/ hallmark | Genes/molecules/pathway/ mechanism involved | Role in cancer | Role in wound healing |
| Oncogenes and oncoprotein | Small GTP binding and hydrolyzing proteins (GTPases)Ras | Mutations that affect some key oncoproteins (such as Ras and Raf) result ineither constitutive activation of proliferative signalling or failure ofnormal negative feedback mechanisms, both of which can drive uncontrolled cell proliferation without the need for mitogenic stimuli. (MacCarthy-Morrogh and Martin 2020) | Ras mediates re-epithelization of wound (Tscharntke et al 2005). Also regulates vascular permeability during angiogenesis, and blood vessels lacking R-Ras leak plasma proteins constantly. (Ketomäki et al. 2019) |
| RAF proto-oncogene serine/threonine-protein kinase, also known as proto-oncogeneRaf | Signaling through the Ras/Raf/MAPK regulates a variety of cellular functions that are important for tumorigenesis.Results in either constitutive activation of proliferative signaling or failure of normal negative feedback mechanisms, both of which can drive uncontrolled cell proliferation without the need for mitogenic stimuli (MacCarthy-Morrogh and Martin 2020), (Molina and Adjei 2006) | Conditional ablation experiments show that Raf -1 is necessary for wound healing (Ehrenreiter, et al 2005). The Ras/Raf/MAPK pathway can stimulate angiogenesis through changes in expression of genes directly involved in the formation of new blood vessels. (Molina and Adjei 2006)  |
| Receptor tyrosine kinases Mitogenic signalsEGF-EGFR signaling | Activation of mitogenic signals: Overall decline in mitogenic signals, detrimental to normal cells, likely drives the competition in favor of precancer clones carrying oncogenic mutations. Hence, if cancer is ‘a wound that does not heal’, combining ‘healing signaling’ and stress-targeted therapies might revert this pernicious dynamic, improving several aspects of cancer treatment (Dias and Bernards 2021) | EGF-EGFR signaling axis is a model of prosurviva (MacCarthy-Morrogh and Martin 2021) proliferative signaling for normal cells, playing a key role in development, homeostasis, and wound healing (Dias and Bernards 2021)There is complicated crosstalk between innate immunity and EGFR signaling in wound healing processes, and imbalance of this crosstalk may lead to impaired wound healing. (Chen et al. 2016) |
| Epidermal growth factor (EGF) and transforming growth factor (TGF) | Melanoma cells secrete TGF-β, PDGF, FGF2, and IL-8, which act in a paracrine manner to stimulate fibroblast activation and proliferation. (Foster et al. 2018) (Sundaram et al 2018) | In wound healing, TGF-β1, via the SMAD-4 pathway, stimulates wound healing activities includingwound contraction by fibroblasts and ECM component deposition,immune cell recruitment, angiogenesis, keratinocyte migration via integrins. TGF-β2 shares similar roles to TGF-β1 and is involved in stimulating ECM deposition, while TGF-β3 is involved in regulating TGF-β1 and decreasing collagen I deposition and scar formation (Foster et al. 2018) (López-Cortés et al. 2021). |
| Extracellular signal-regulated kinase 1/2 (ERK) belongs to the mitogen-activated protein kinase (MAPK) family | Nearly one-third of all types of cancers involve deregulated ERK. ERK was constitutively active in ~50 tumor cell lines  (Lu et al 2020).The mitogen-activated protein kinase (MAPK) a complex interconnected signaling cascade with frequent involvement in oncogenesis, tumor progression, and drug resistance. The MAPK family consists of a large number of kinases altered in cancers and against which many targeted therapies were developed. (Braicu et al 2019) | Expression of the metallo-proteinases, MMP-9 and MMP-2, induced by EGFR-dependent activation of ERK1/2 and PI3K/Akt signaling cascades are also important for promotion of airway epithelial cell migration and wound repair in response to airway epithelial cell injury. (Chen et al. 2016). MAPKAPK-2 signaling is critical for cutaneous wound healing (Thuraisingam et al 2010) |
| Transcription Factors | Core transcription factors *OCT4*, *SOX2*, *NANOG* | Increased expression of SOX2 may be involved in primary transformation and carcinogenesis of squamous tumors in the process of hyperplasia and dysplasia.Transcription factors in very early stage of carcinogenesis can greatly enhance detection of oral squamous cell carcinoma (OSCC). (Naini et al. 2019) | During wound healing, SOX2 induced proliferation of epithelial and connective tissue cells and promoted angiogenesis.( Uchiyama et al 2019). Olfactomedin-4 improves cutaneous wound healing by promoting skin cell proliferation and migration through POU5F1/OCT4 and ESR1 signalling cascades (Klaas et al 2022) Epidermal stem cells (EpSCs) with high expression of regulatory factor Nanog can promote wound healing (Yin et al 2022). |
| Signal Transducers and Activators of Transcription (STATs) | The transcription factors STAT1 and STAT3 appear to play opposite roles in tumorigenesis. While STAT3 promotes cell survival/proliferation, motility and immune tolerance and is considered as an oncogene, STAT1 enhances inflammation and innate and adaptive immunity, triggering in most instances anti-proliferative and pro-apoptotic responses in tumor cells (Pensa et al. 2013) | Stat 3 and Stat 6(Byun and Gardner 2013)Stat3 is activated by oncogenic signals such as Src and EGFR signaling as well as by cytokines and mitogens involved in wound healing such as interleukin-6 and hepatocyte-growth factor (HGF) (Dauer et al. 2005)Stat3 activation results upregulation of genes involved in cell invasion, chemotaxis, angiogenesis, and blood coagulation. (Dauer et al. 2005)Cell shape change during wound healing is also mediated in part by the JAK/STAT pathway (Lee et al. 2017). |
| *ETS1, ETS2* | ETS1 and/or ETS2 form an autoregulatory circuit with the MAPK pathway components ERK1, ERK2 and DUSP6 (Bushweller 2019) | Ets-1 plays an important role in angiogenesis in the early phase of ulcer healing. (Ito et al 1998) cdk10 and ETS2 involved during corneal wound healing (Zehra et al 2019) |
| Tumor suppressor genes | RetinoblastomaRb1 | Reduced expression of [retinoblastoma protein](https://www.sciencedirect.com/topics/medicine-and-dentistry/retinoblastoma-protein) pRb in cancer (Bartkova et al 2003). Mutational inactivation of *Rb1* causes the pediatric cancer retinoblastoma, while deregulation of the pathway is common in most human cancer. The *Rb1*-encoded protein (pRb) is well known as a general cell cycle regulator, and this activity is critical for pRb-mediated tumor suppression. (Goodrich, 2006; Goodrich 2022) | Rb1 enhances cutaneous wound healing process by increasing keratinocyte migration (Shin et al 2018) |
| FoxO3a | FOXO3a acts as a tumor suppressor in cancer. FOXO3a is frequently inactivated in cancer cell lines (Liu, *et al.*2018). | FoxO play a crucial role in wound healing. FoxO1 and FoxO3 primarily function in epithelial wound healing. re-epithelialisation and keratinocyte migration. (Miao, C., Li, Y. and Zhang, X. (2019),FOXO1 and FOXO3 possess biological functions such as morphogenesis, maintenance and tissue regeneration. FOXOs important target to enhance wound healing (Rajendran et al. 2019) |
| P53 | Importasnt tumor suppressor. Loss or mutation of p53 in cancers can affect the recruitment and activity of myeloid and T cells, allowing immune evasion and promoting cancer progression. p53 can also function in immune cells, resulting in various outcomes that can impede or support tumour development. (Blagih et al 2020). Prevents dedifferentiation (Di Fiore et al 2014) | p21 and p53 activity induced during human wound healing.(Blagih et al 2020).  |
| PTEN |  loss of PTEN function is common in different types of tumours(Álvarez-Garcia et al 2019) | Injury to the corneal epithelium downregulates the expression of PTEN at wound edges, allowing increased PI3K/Akt signaling, thereby contributing to a significant enhancement of cell migration and wound healing. (Cao et al 2011).  |
| Adenomatous polyposis coli (APC) | Mutations in APC are found in around 80% of sporadic colonic tumors.(Minde et al 2011).  | Promotes chronic wound healing (Zhao et al 2019) |
| Collagen | Both promoting and tumor suppressing roles Upregulated collagen in tumour growth (Xue and Jackson 2015, Xu et al 2019, Bhattacharjee et al 2021)ECM as well as immune modulator (Mutolo et al 2012, Romer et al 2021) | Collagen, a key component of the extracellular matrix, plays critical roles in the regulation of the phases of wound healing either in its native, fibrillar conformation or as soluble components in the wound milieu.(Mathew-Steiner et al 2021) |
| Tumor suppressor genes p16INK4A | p16Ink4a is a negative regulator of cell proliferation. Close to 50% of all human cancers show p16Ink4a inactivation (Romagosa et al. 2011) | P16INK4A is expressed by epidermal and oral keratinocytes at the migrating fronts of healing wounds (Natarajan et al. 2005) (Chikenji et al. 2019) |
| FAT1, FAT4 | Fat cadherins are extremely large cell adhesion molecules, with >30 cadherin repeats, including FAT1, FAT2, FAT3, FAT4. FAT1 is an important *trans*-membrane protein involved in the regulation of cell adhesion and growth, migration, actin dynamics and orientation, playing critical roles in tumor development. It is often regarded as a tumor-suppressor gene or oncogene in different types of human cancer ( Hu et al 2018) | FAT1 depletion led to a significant increase in cell migration and invasion abilities in the YSE2 and Colo680N cell lines which were also confirmed by the wound healing migration assay( Hu et al 2018) |
| SMAD proteins | SMAD4, which serves as the central mediator of TGF-βsignaling, is specifically inactivated in over half of pancreatic duct adenocarcinoma, and varying degrees in many other types of cancers. DPC4/Smad4 is a critical tumor suppressor involved in the progression of pancreatic cancer(Zhao et al 2018).  | Extracellular matrix modelling and wound healing (Schiller et al 2004). SMAD3 null mice had excessive proliferation. Smad knockout leads to aberrant wound healing (Owens et al 2010) |
| promyelocytic leukemia protein (PML)PML–RARα | PML tumor suppressor in multiple complex ways (Hsu and Kao 2018). Blocks differentiation; (Bushweller 2019)PML—RAR *α* is the hallmark protein of acute promyelocytic leukaemia, a highly malignant subtype of acute myeloid leukaemia that accounts for approximately 10% of all AML cases.(Saeed et al. 2011) | Both PML and STAT1 are important regulators of ITGB1 expression in ECs. Furthermore, antibody experiments show that blocking ITGB1 delays wound healing even in the presence of TNFα and IFNα.( Cheng et al 2012) ATRA inhibits PC regeneration, and decreased RARα expression in wounds after E14 inhibits myoblast migration (Takaya et al 2022) |
| ARID1A | ARID1A loss-of-function mutations are commonly found in human cancer  (Sun et al 2016) | Knockouts faster would closure (Rahmanto et al 2020, Sun et al 2016)Arid1a is physiologically suppressed during liver regeneration and wound healing, and that complete ablation leads to improved regeneration (Sun et al 2016). |
| NOTCH | Notch signaling pathway’s oncogenic or tumor-suppressor abilities are highly context dependent. (Lobry et al 2014) | Wnt and Notch signaling pathway involved in wound healing by targeting *c-Myc* and *Hes1* (Shi et al 2015) |
| Evading immune mechanisms | CD47 also known as integrin associated protein is a transmembrane protein*CD47* | A key anti-phagocytic molecule, enables various types of cancer cells to evade phagocytosis by macrophages (Chikenji et al. 2019) | Epithelial CD47 expression regulates mucosal wound closure in vivo by promoting signaling through a β1 integrin-dependent FAK-Src-p130 Cas pathway (Reed et al. 2019) |
| Immune checkpoint receptors  | programmed cell death 1 (PD-1) and cytotoxic T lymphocyte associated protein 4 (CTLA-4) serve as immune checkpoint receptors inactivating macrophages (Barrueto et al. 2020). | Immune regulation by PD-L1 promotes tissue repair (Su et al 2019, Wang et al 2022) |
| Fibroblast activity | platelet-derived growth factorsPDGFRα and -β(PDGFRαalsoknownasCD140α) | Involved in recruitment and phenotypic remodeling of CAFs. Found to be robustly expressed by CAFs in mouse squamous cell carcinoma.(Foster et al. 2018) | PDGFs induce fibroblast reactivity and fibrosis.Expression associated with fibroblasts involved in organ fibrosis and wound healing in miceand humans. Expressed by dorsal, scar-formingfibroblasts in mice.(Reuterdahl et al. 1993). |
| FSP1 (fibroblast specific protein-1,also known as S100A4) | FSP1-positive cells were increased in human and mouse experimental liver injury including liver cancer. (Österreicher et al. 2011) | FSP1 and α1β1 integrin expression are associatedwith a quiescent fibroblast phenotype in mice.FSP1+ fibroblasts are predominant cell types withinexperimental granulation tissue during wound healing at day 22 in a mouse model. (Foster et al. 2018).  |
| Vim (vimentin) | Expression necessary for fibroblast initiation of EMTin tumor cells. Also expressed by epithelial cells thathave undergone EMT. | Expressed by quiescent and activated fibroblasts.Expression critical for fibroblast proliferation. Invim-deficient mouse wounds, fibroblasts do notproliferate, which inhibits TGF-β1 signaling andSlug, yielding dysfunctional wound healing. Also expressed by other mesenchymal cells. (Foster et al. 2018) |
| Col-I and Col-III  | Upregulation in cancer-associated fibroblast CAFs. Mesenchymal stem cells candifferentiate into fibroblastic cells expressing α-SMA and Col-I in human lung tissue. (Foster et al. 2018) | Upregulated in fibroblasts involved in scarformation and fibrosis. Secreted by En1-expressingfibroblasts in mouse wound healing Majorpopulation of human fibroblasts expressingSFRP2/DPP4 associated with collagen bundles (Foster et al. 2018) |
| Postn (periostin) | Expressed by CAFs in colon cancer. Upregulatedin human pancreatic stellate (fibroblastic) cells;deposition seen at carcinoma cell infiltration sites (Foster et al. 2018) | A tissue repair product; levels in wounds correlatewith activated fibroblast presence in granulationtissue. (Foster et al. 2018) |
| Desmin | Dysregulated in many types of cancers (Mittal 2020) | Expressed by fibroblasts in hypertrophic stars and fibrosis in humans. (Foster et al. 2018) |
| Podoplanin | Marks a specific CAF population. Prognostically relevant in lung, breast, and SCC. Also expressed by lymphatic endothelial cells. (Foster et al. 2018). | Displayed by human dermal fibroblasts in sclerosis (along with CD 90). (Foster et al. 2018). |
| CD26 (also known as dipeptidylpeptidase-4 [DPP4]) | Fibroblasts of En1 lineage (expressing CD26/DPP4) contribute to tumor stroma in mouse melanoma.When these fibroblasts are ablated (via diphtheriatoxin), tumor burden decreases.(Foster et al. 2018) | Associated with mouse papillary fibroblastspopulation. Expressed by En1+ fibroblast in themouse dermis. Expressed by human dermal SFRP2+fibroblasts (Foster et al. 2018). |
| CD90 (also known as thymocyteantigen 1 [Thy1]) | High CD90-expressing CAFs promote tumorsmorethan low CD90-expressing CAFs in human gastric andprostate cancer.(Foster et al. 2018) | Expressed by human dermal fibroblasts. Displayedby dermal fibroblasts in sclerosis (along withPodoplanin) in humans. (Foster et al. 2018). |
| NG2 (neuron glial antigen-2) | Expressed on normal and intratumoral pericytes. Pericytes distinguished by regulator of G-protein signaling 5 (RGS5) expression, which associates with CD31 rather than α-SMA. (Foster et al. 2018).  | Expressed by migratory fibroblasts in response toinjury in vitro. (Foster et al. 2018) |
| Tenascin-C | CAF expression involved in regulating carcinoma cell adhesion.(Foster et al. 2018) | Involved in recruiting fibroblasts to wounds. (Foster et al. 2018) |
| ED-A  | Expressed by CAFs, as well as carcinoma cells and tumor blood vessels. Increased urinary ED-A associated with poor prognosis in human bladder cancer.(Foster et al. 2018) | A mesenchymal marker, upregulated in activated fibroblasts (Kohan et al. 2010).  |
| E to M transition (EMT) | KLF8, SIX1 RUNX2 | KLF8, SIX1 RUNX2 Regulate EMT (Bushweller 2019) |  Runt-related transcription factor 2 (RUNX2) is required for mesenchymal stem cells to differentiate to osteoprogenitor cells. (Sato and Takaoka 2015) |
| platelet-derived growth factorsPDGFRα and -β(PDGFRαalsoknownasCD140α) | Autocrine PDGF signaling has been implicated in various types of malignancies such as gliomas and leukemia. In contrast, paracrine signaling was found in cancers that originate from epithelial cells, where it may be involved in stromal cell recruitment, metastasis, and EMT ( Liu et al 2011) |  PDGFRβ is also expressed by normal pericytes. An increased expression of PDGF beta receptor protein was prominent in vessels in the proliferating tissue zone in wounds as early as 1 d after surgery (Reuterdahl et al. 1993) |
| Cell proliferation migration and metastasis | L1CAML1 cell adhesion molecule  | L1CAM+ cells in human colorectal cancer (CRC) have metastasis-initiating capacity (Ganesh et al. 2020) | L1CAM, appears to be necessary and sufficient in both tissue repair and cancer contexts in the gut (MacCarthy-Morrogh and Martin 2020) |
| Rho small guanosine triphos-phatase (GTPase) | Misregulated in cancer (MacCarthy-Morrogh and Martin 2020) | Rho family small GTPases are absolutely required for wound re-epithelialization (MacCarthy-Morrogh and Martin 2020) |
| increased production of several proteases | The production of metastasis appears to involve a number of different proteases including the urokinase form of plasminogen activator, cathepsin B, cathepsin D and various metalloproteases (MJ 1992) | wound granulation tissue requires the increased production of several proteases, particularly matrix metalloproteinase 1(MMP1), which may facilitate integrin-matrix adhesion dynamics by locally cleaving various extracellular matrix (ECM) and ECM-associated proteins |
| α-SMA (α-smooth muscle actin,also known as acta2) | Alpha-SMA is expressed by tumor cells carcinoma. Tumor cells that express α-SMA are predicted to be the cells that have the invasive nature, tend to metastasize, and have poorer prognosis.(Anggorowati et al. 2017) | An intracellular protein expressed by fibroblasts transiently in mouse wound healing. Associated with increased fibroblast contractility. Also expressed by normal fibroblasts, smooth muscle cells, and pericytes. In healing tissues, fibroblasts acquire a contractile phenotype, characterized by formation of microfilament bundles, and by de novo expression of α-smooth muscle actin (α-SMA). These activated cells, termed “myofibroblasts” participate in the reparative response, by secreting large amounts of extracellular matrix proteins and may be responsible for contraction of healing wounds (Shinde, Humeres, and Frangogiannis 2017)(Tomasek et al. 2005) |
| Nrf2 | The constitutive activation of Nrf2 in various cancers induces pro‐survival genes and promotes cancer cell proliferation by metabolic reprogramming, repression of cancer cell apoptosis, and enhancement of self‐renewal capacity of cancer stem cells.(S. Wu, Lu, and Bai 2019) |  |
|  | aberrant YAP/TAZ activity: *WWTR1* and *YAP1* Hypermethylation of the promoter region of *LATS1, STK3*, *WWC1* and *TAOK2* | YAP/TAZ are also implicated in metastatic progression — the leading cause of cancer-related death — which involves several events that participate in enhancing the migratory potential of aggressive cancers. (Dey, Varelas, and Guan 2020) | Roles for YAP/TAZ in basal stem cell control are apparent in epidermal development and skin wound repair.(Dey, Varelas, and Guan 2020) YAP/TAZ promotes wound healing and tissue regeneration, and, under physiological conditions, YAP/TAZ activity is restricted mainly by the LATS1/2 kinases. |
| Micro RNA’s*MiR-31* | Down-regulated [miR-31](https://www.sciencedirect.com/topics/medicine-and-dentistry/microrna-31) in human cancer shows tumor repressive function.UP-regulated [miR-31](https://www.sciencedirect.com/topics/medicine-and-dentistry/microrna-31) in human cancer showing tumor promotive function.diverse [signaling pathways](https://www.sciencedirect.com/topics/medicine-and-dentistry/signal-transduction) in which [miR-31](https://www.sciencedirect.com/topics/medicine-and-dentistry/microrna-31) is involved to clarify its different effects on cancer.(Yu et al. 2018)  | expression of miR-31 is gradually upregulated in wound edge keratinocytes in the inflammatory(D et al. 2015) (Shi et al. 2018) |
| Heat shock proteins Hsp27 (HSPB1), Hsp72 (HSPA1A HSPA1B), Hsp90 alpha (HSP) | Hsp70 is secreted from prostate carcinoma cells through such a route along with accompanying lysosomal marker proteins. Intracellular HSPs—most notably Hsp27, Hsp72 (the combined product of the HSPA1A and HSPA1B genes) and Hsp90 (product of the HSPC2 and HSPC3 genes)—have been shown to accumulate to high levels in many types of cancer(Calderwood 2018) | Hsp90α is required for normal wound closure. (Bhatia et al. 2018)(Bellaye et al 2014 and Song et al 2014) |
| GADD45 expression | Defects in the GADD45 pathway can be related to the initiation and progression of malignancies.(Tamura et al. 2012) |  GADD45 appears to increase the access of repair machinery to damaged DNA, is produced as a consequence of inflammation. (MacCarthy-Morrogh and Martin 2020) |
| Cell fusion | Cell fusion | Two way effects. Hybrids of malignant cells with normal cells become non-tumorigenic Cooper 2000) “wolf in sheep's clothing” model. The model suggests that a tumor cell becomes metastatic by fusion to normal cells that travel throughout the body freely, such as lymphocytes or macrophages.(Duelli and Lazebnik 2003) | Giant syncytium involved in re-epithelization in drosophila (Losickcurr boil 2013). Cell fusion may enable the stem cells to acquire the program/properties of the target tissue (Dorner et al Int J MolSci 2020) |
| Inducing angiogenesis | Proangiogenic signals, primarily thrombospondinand vascular endothelial growth factor (VEGF)Wnt7a signals | vascular endothelial growth factor (VEGF) primarily regulated by the transcription factor nuclear factor NF-kB, which is active in most tumors and is induced by carcinogens (Wu and Chen 2014) | VEGF is unique for its effects on multiple components of the wound healing cascade, including angiogenesis and recently shown epithelization and collagen deposition (Bao et al. 2009) |
| hypoxia inducible factor HIF | HIF-1α is overexpressed in human cancers as a result of intratumoral hypoxia as well as genetic alterations, such as gain-of-function mutations in oncogenes (for xample, *ERBB2*) and loss-of-function mutations in tumour-suppressor genes (MacCarthy-Morrogh and Martin 2020) (Semenza 2003). | Expressed (Cañedo-Dorantes and Cañedo-Ayala 2019). Hypoxia upregulates tissue expression of VEGF and its receptors, which in turn promote an angiogenic response. (Bao et al. 2009) |
| TNF | TNF is secreted by inflammatory cells, which may be involved in inflammation-associated carcinogenesis. TNF exerts its biological functions through activating distinct signaling pathways such as NFκB and JNK (WANG and LIN 2008) | Expressed (Cañedo-Dorantes and Cañedo-Ayala 2019)macrophages induce angiogenesis, in part by releasing TNF-α, which may in turn induce VEGF expression in keratinocytes and fibroblasts (Bao et al. 2009) |
| Co-opting cells, coordination and cross talk |  | Cross talk between malignant cells and many other types of cells in the microenvironment, often mediated by microRNA (Pascut et al 2020, Su et al 2021) | Cross talk between many types of cells during wound healing (Brazil et al 2019, Bird et al 2021) |
|  | Neurons interact with tumor cells and facilitate tumor growth (Hanahan and Monje 2023) | Neurons contribute to tissue repair and fine tune the microenvironment (Beura et al 2022) |
| Inflammation and its regulation | receptor CXCR2 | chemokines, including those binding to the receptor CXCR2, act as attractants for neutrophils to clones of preneoplastic cancer cells (MacCarthy-Morrogh and Martin 2020) | chemokines, including those binding to the receptor CXCR2, act as attractants for neutrophils to wounds (MacCarthy-Morrogh and Martin 2020) |
| TGF- β 1 and 2 | TGF-β1 has more complex roles in cancer, being both positively and negatively associated with tumor progression. TGF Beta 1 overexpression of TGF-1 has also been linked to multiple cancers (MacCarthy-Morrogh and Martin 2020) | The TGF-β–related growth factor, activin, also has both protumorigenic activity and considerable effects in a wound repair scenario.(MacCarthy-Morrogh and Martin 2020) |
| CSF-1 (colony-stimulating factor–1)  | CSF1 is involved in breast cancer progression through inducing monocyte differentiation and homing (Ding et al 2016) | CSF1 is essential to macrophage survival and function and there are profound macrophage deficiencies in op/op mice carrying an inactive Csf1 gene. (van den Boorn and Hartmann 2014) |
| high mobility group box 1 HMGB1 | Leukocytes to cancer cells (MacCarthy-Morrogh and Martin 2020) | Leukocytes to acute wounds cells (MacCarthy-Morrogh and Martin 2020) |
| Duox | blocking Duox prevents immune cells from homing to growing clones of precancer cells (MacCarthy-Morrogh and Martin 2020) | Ca2+ signal activates the nicotinamide adenine dinucleotide phosphate oxidase (NOX), Duox to generate hydrogen peroxide (MacCarthy-Morrogh and Martin 2020) |
| IL-12 and IL-23  | IL-12 also induces the production of large amounts of IFNγ which itself is cytostatic/cytotoxic, anti-angiogenic and can upregulate MHC I and II expression on tumor cells for enhanced recognition and lysis. Interleukin 12 (IL-12) is a pleiotropic cytokine that plays an essential role in Th1-type immune response against cancer, a condition where cells in a particular part of the body grow and reproduce uncontrollably. (X 2017) | Accelerated wound healing phenotype in Interleukin 12/23 deficient miceHigher levels of IL-1β and IL-6 during wound healing (Matias et al 2011) |
| Replicative immortality Cell senescence. |  | Replicative immortality or resistance to the normal senescence of differentiated cells is a hallmark of cancers (Ozturk et al 2006) | Fine tuning and reshaping of senescence mechanisms is observed in wound healing. Mechanisms blocking chronic cell senescence accelerate healing (Wilkinson and Hardman 2020) |
| Human telomerase reverse transcriptase (TERT) | hTERT expression is up-regulated in tumors via multiple genetic and epigenetic mechanisms including hTERT amplifications, hTERT structural variants, hTERT promoter mutations and epigenetic modifications through hTERT promoter methylation.(Leão et al. 2018) | gene delivery of hTERT by adenovirus (Ad-hTERT) dramatically improved ischemic wound healing in an aged rabbit model.  |
| Altered energy metabolism | increased glucose metabolism, glycolysis, fatty acid synthesis and glutamine metabolic rates. | Warburg and reverse Warburg effect‘the Warburg effect’. Subsequently, the ‘reverse Warburg effect’ proposed that tumor-associated fibroblasts can produce large amounts of lactic acid via aerobic glycolysis, which is provided to adjacent cells in a paracrine manner, causing the activation of mitochondria, increasing OXPHOS in adjacent cells and promoting tumoractivity .(Peng et al. 2021) Generally, ‘the Warburg effect’ and ‘reverse Warburg effect’ play an essential role in the development of cancer. Aberrant glucose metabolism regulates cancer proliferation, cell cycle, drug resistance, and DNA repair (Lin et al. 2019) | the master regulator of inflammatory and metabolic responses (e.g., aerobic glycolysis), is essential for physiological wound healing.  NLRP3-mediated inflammation is activated in keloids, (b) whether Glut1 expression is elevated in burn tissue (Vinaik et al. 2020) glycolysis is prominent. Lactic acid in the interstitial fluids of healing wounds amount up to 5–15 mM. Endogenous lactic acid and exogenous lactate can promote reparative angiogenesis with recruitment of endothelial progenitor cells, activate procollagen factors, and enhance extracellular matrix deposition in mice with ischemic wounds. (Sun et al. 2017) |
| Chromosomal Alterations | Polyploidization, Aneuploidy | The vast majority of cancers show aneuploidy, with around 90% of solid tumors and 75% of hematopoietic cancers having abnormal chromosome numbers. Aneuploidy has been shown to precede transformation in a variety of cancers (Coward and Harding 2014). Ploidy reduction frequently occurred during an early phase of tumorigenesis, and enhanced polyploid-derived cancer development.(Matsumoto et al. 2021). | Both JNK and Hippo signaling are upregulated at the wound ite, suggesting that these pathways regulate the wound response. Hippo signaling, in onjunction with the TOR and insulin/IGF athways, plays an important role in organ size control in both mammals and insects. Yki, the major effector of Hippo signaling, is required for the polyploidization response. Hippo signaling in response to wound damage may activate *cycE*, a gene known to be regulated by Yki in other tissues to stimulate S phase reentry.(Losick, Fox, and Spradling 2013). In *Drosophila* wound-induced polyploidy (WIP) is an essential mechanism to replace tissue mass and restore tissue integrity in the absence of cell division. (Losick 2016b).Normal tissue has a low level of aneuploidy (Knouse et al 2014) which may be increased when rapid proliferation is the priority. |
| centrosome amplification | CA is associated with oncogenic phenotypes increased invasiveness and aberrant stem cell divisions. The potential mechanisms leading to CA in cancer are numerous and include centriole over duplication, *de ovo* centrosome formation, fragmentation of overly elongated centrioles and cytokinesis failure(Sabat-Pośpiech et al. 2019).Centrosome amplification is commonly observed in hematologic malignancies and solid tumors, and a clear link exists between centrosome amplification and aneuploidy in a wide variety of cancer cell lines (Vitre et al. 2015) | The reorientation of the microtubule organizing center during cell migration into a wound in the monolayer was observed in living wound-edge cells expressing gamma-tubulin tagged with green fluorescent protein. (Yvon et al. 2002) |
| Genomic instability | Believed to be a unique cancer hallmark (Negrini et al 2010) | Inflammation induced genomic instability mediated by microRNAs in intestinal mucosal injury (Butin-Israeli et al 2019). |
| Neoantigens/ neo-epitopes | RNA splicing leading to novel popypeptides | RNA splicing is involved in carcinogenesis and a potential source of neoantigens in tumors (Anczuków et al 2016, Park and Chung 2019) | Although neoantigens are not known during wound healing the underlying altered splicing mechanisms are present (Jensen et al 2014) |
| indoleamine 2,3-dioxygenase 1 (IDO1) induced ribosomal codon reassignment leading to novel polypeptides | Generation of new antigens through ribosomal codon reassignment induced by the enzymeindoleamine 2,3-dioxygenase 1 (IDO1), which is induced by IFNγ3–5(Bartok et al 2021, Pataskar et al 2022). | Although neoantigns have not been demonstrated directly in healing tissue, the pathways responsible are triggered during healing including indoleamine 2,3-dioxygenase (IDO1) activity. (Ito et al 2015) |

References to table 1:

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Supplementary table 2:

**Wound healing functions of the cancer related genes**: We searched literature for the genes listed by COSMIC catalogue of somatic mutations in cancer (<https://cancer.sanger.ac.uk/cosmic/curation>) which is a list of genes that are somatically mutated and causally implicated in different types of cancers in humans. The genes already covered in supplementary table 1 are omitted from table 2. The reference giving evidence for some role of the gene in normal wound healing process is tabulated. It can be seen that out of the 264 genes listed by COSMOS, only for 21 (i.e. less than 8%), we did not find evidence for a direct role in wound healing. Out of the 21, eight are known to be involved in DNA repair (marked by \*).

Tabulated below are the genes from the catalogue, not covered by table 1 and at least 1 reference to literature for evidence of its involvement in wound healing.

|  |  |
| --- | --- |
| 1. ABL1
 | Zulueta-Coarasa et al 2014 |
| 1. ACVR1
 | Sorkin et al 2017 |
| 1. ACVR1B
 | Qiu et al 2011 |
| 1. ACVR2A
 | Munz et al 1999 |
| 1. AKT1
 | Somnath et al 2008 |
| 1. ALK
 | Wallace et al 2013 |
| 1. AMER1/WTX
 | Li et al 2020 |
| 1. AR
 | Ashcroft 2002, Lai 2012 |
| 1. ARHGAP26
 | Yao et al 2015 |
| 1. ARID1A
 | Sun et al 2016 |
| 1. ARID1B
 | Sun et al 2016 |
| 1. ARID2
 | Sun et al 2016 |
| 1. AFXL1
 | Wang et al 2021 |
| 1. ATM
 | Eickmeier et al 2014 |
| 1. ATP1A1
 | Wu et al 2016 |
| 1. ATP2B3
 | Talarico and Mangini 2007 |
| 1. ATR
 | Sharma et al 2023 |
| 1. ATRX
 | Danussi et al 2022 |
| 1. AXIN1
 | Qin et al 2020, Peng et al 2021 |
| 1. AXIN2
 | Whyte et al 2013 |
| 1. B2M
 | Sun et al 2016, Wang et al 2020, Molenaar et al 2021 |
| 1. BAP1
 | Liu et al 2022, Yeu et al 2019. |
| 1. BARD1\*
 |  |
| 1. BCL9L
 | Sannino et al 2016, Zhang et al 2020 |
| 1. BCOR
 | Manjur et al 2021 |
| 1. BCORL1
 | Xu et al 2018 |
| 1. BIRC3
 | Riwaldt et al 2017 |
| 1. BLM
 | Chen 2016 |
| 1. BRAF
 | Escuin-Ordinas et al 2021 |
| 1. BRCA1
 | Wei et al 2020, Portier et al 2021 |
| 1. BRCA2\*
 |  |
| 1. BTK
 | Yang et al 2019 |
| 1. CACNA1D
 |  |
| 1. CALR
 | Kepp 2020,  |
| 1. CARD11
 |  |
| 1. CASP8
 | Lee et al 2009 |
| 1. CBL
 | Rush et al 2014 |
| 1. CBLB
 | Li et al 2022 |
| 1. CBLC
 | Li et al 2022 |
| 1. CD79A
 |  |
| 1. CD79B
 |  |
| 1. CDC73
 | Yu et al 2022 |
| 1. CDH1
 | Hwang et al 2012 |
| 1. CDK12
 | DNA repair |
| 1. CDKN1B
 | Bencivenga et aL 2021 |
| 1. CDKN2A
 | liu w et.al 2020 |
| 1. CDKN2C
 |  |
| 1. CEBPA
 | Guo et al 2018 |
| 1. CHD4
 | Shao et al 2020 |
| 1. CHEK2
 |  |
| 1. CIC
 | Da Via 2020 |
| 1. CNOT3
 |  |
| 1. COL2A1
 | Almeida et al 2015 |
| 1. CREBBP
 | Holmstrom et al 2019 |
| 1. CRLF2
 | Jiang et al 2019 |
| 1. CSF1R
 | Mac Donald et al 2010 |
| 1. CSF3R
 | Meier et al 2022 |
| 1. CTCF
 | Deonarine et al 2007 |
| 1. CTNNA1
 | Chin et al 2022 |
| 1. CTNNB1
 | Zhou et al 2021, Liu et al 2020 |
| 1. CUX1
 | Latreille et al 2017 |
| 1. CXCR4
 | Deonarine et al 2007, Chen et al 2021 |
| 1. CYLD
 | Lim et al 2012 |
| 1. DAXX
 | Deonarine et al 2007 |
| 1. DDB2
 | Dardare et al 2022 |
| 1. DDR2
 | Olaso et al 2011 |
| 1. DDX3X
 | Chen et al 2015 |
| 1. DGCR8
 | Yi et al 2009 |
| 1. DICER1
 | Ghatak et al 2015, Braun 2021 |
| 1. DNM2
 | Willis et al 2021 |
| 1. DNMT3A
 | Bhatt et al 2022 |
| 1. DROSHA
 | Kuehbacher 2007 |
| 1. ELF3
 | Oliver et al 2011 |
| 1. EP300
 | Ring et al 2020 |
| 1. EPAS1
 | Takeda 2004 |
| 1. ERBB3
 | Okwueze et al 2007 |
| 1. ERBB4
 | Frey et al 2009 |
| 1. ERCC2\*
 |  |
| 1. ERCC3\*
 |  |
| 1. ERCC5\*
 |  |
| 1. ESR1
 | Qi et al 2020 |
| 1. ETNK1
 | Li et al 2019 |
| 1. EZH2
 | Osokine et al 2022, Jin et al 2022 |
| 1. FAS
 | Kou et al 2018, Guan et al 2000 |
| 1. FAT1
 | Peng et al 2021 |
| 1. FAT4
 | Jiang et al 2017 |
| 1. FBXO11
 | Jin et al 2015 |
| 1. FBXW7
 | Zhong et al 2021 |
| 1. FEN1
 | Zhang et al 2020 |
| 1. FES
 |  |
| 1. FGFR1
 | Prudovsky 2021 |
| 1. FGFR2
 | Prudovsky 2021 |
| 1. FGFR3
 | Prudovsky 2021 |
| 1. FGFR4
 | Prudovsky 2021 |
| 1. FLT3
 | Aydin 2016 |
| 1. FLT4
 | Godfraind 2013 |
| 1. FOXA1
 | Liu et al 2022 |
| 1. FOXL2
 | Marongiu 2016 |
| 1. FUBP1
 | Zhang et al 2021 |
| 1. GATA1
 | Zhang et al 2016 |
| 1. GATA2
 | Chiang 2014 |
| 1. GNA11
 | Doçi et al 2017 |
| 1. GNAQ
 | Doçi et al 2017 |
| 1. GNAS
 | Jin et al 2019 |
| 1. GRIN2A
 |  |
| 1. H3F3A
 | Sun et al 2022 |
| 1. H3F3B
 | Chen et al 2021 |
| 1. HIF1A
 | Botusan et al 2008, Deonarine et al 2007 |
| 1. HIST1H3B
 | Gomes et al 2019 |
| 1. HNF1A
 | Zhu et al 2017 |
| 1. HRAS
 | Sugita et al 2018 |
| 1. IDH1
 | Shen et al.2020 |
| 1. IDH2
 | kim et al 2019 |
| 1. IKBKB
 | Ramirez et al 2018 |
| 1. IK2F1
 |  |
| 1. IL6ST
 | hunter et al 2015 |
| 1. IL7R
 | Kim et al 2018 , Wee et al 2022 |
| 1. IRS4
 | Kuai et al 2022 |
| 1. JAK2
 | Wang et al 2022, Takahash et al 2021 |
| 1. JAK3
 | Mishra et al 2013 |
| 1. KCNJ5
 | Rezania et al 2016 |
| 1. KDM5C
 | Xiao et al 2022 |
| 1. KDM6A
 | Ni et al 2019 |
| 1. KDR
 | Santos et al 2007 |
| 1. KEAP-1
 | Rabbani et al 2017 |
| 1. KIT
 | Huttunen et al 2002 |
| 1. KMT2A
 | Zhang et al 2017 |
| 1. KMT2C
 | Chiappetta et al 2019 |
| 1. KMT2D
 | Lv et al 2018 |
| 1. LEF1
 | Phan et al 2020 |
| 1. LRP1B
 | Ni et al 2013 |
| 1. LZTRI
 | Ye et al 2023 |
| 1. MAP2K1
 | Deonarine et al 2007 |
| 1. MAP2K2
 | Halbrook et al 2017 |
| 1. MAP2K4
 | Liu et al 2021 |
| 1. MAP3K1
 | Wang et al 2022 |
| 1. MAP3K13
 | Chen et al 2018 |
| 1. MAPK1
 | Jin et al 2022 |
| 1. MAX
 |  |
| 1. MED12
 | Li et al 2020 |
| 1. MEN1
 | Luo et al 2021 |
| 1. MLH1
 | Fukuhara etal 2014 |
| 1. MPL
 |  |
| 1. MSH2\*
 |  |
| 1. MSH6
 | Chen et al 2019 |
| 1. MTOR
 | Squarize et al 2010 |
| 1. MUC6
 | Buisine et al 2001 |
| 1. MYC
 |  |
| 1. MYCN
 | Hasan et al 2013 , Sradhanjali et al 2021 |
| 1. MYD88
 | Macedo et al 2007 |
| 1. MYOD1
 | Wu et al 2020 |
| 1. NCOA2
 | Mullany et al 2021 |
| 1. NCOR1
 | Geiger et al 2020 |
| 1. NCOR2
 | Wu et al 2018 |
| 1. NF1
 | Kolvunen et al 2005 |
| 1. NF2
 | Jia et al 2022 |
| 1. NFE2L2
 | Hiebert P. and Werner S. 2019 |
| 1. NFKBIE
 | Qian et al 2022 |
| 1. NOTCH1
 | Shao et al 2020 |
| 1. NOTCH2
 | Kimtall et al 2017 |
| 1. NPM1
 | Loubeau et al 2014 |
| 1. NRAS
 | Liu et al 2017 |
| 1. NTSC2
 | Li et al 2022 |
| 1. NTRK3
 | Xiong et al 2016 |
| 1. PAX5
 | Hosokawa K et al 2021 |
| 1. PBRM1
 | Mota et al 2019 |
| 1. PDGFRA
 | Yao et al 2022 |
| 1. PHF6
 | Zhuang et al 2021 |
| 1. PHOX3B
 |  |
| 1. PIK3CA
 | Kim et al 2020 |
| 1. PIK3CB
 | Wang et al 2022 |
| 1. PIK3R1
 | Ai et al 2018 |
| 1. PLCG1
 | Seo et al 2022 |
| 1. POLD1
 | Shen et al 2017 |
| 1. POLE\*
 |  |
| 1. POLQ
 | Pan et al 2021 |
| 1. POTI\*
 |  |
| 1. PPM1D
 | Lu et al 2020 |
| 1. PPP2R1A
 | Shi et al 2022 |
| 1. PPP6C
 | Wang et al 2020 |
| 1. PRDM1
 | Ranoni et al 2021 |
| 1. PREX2
 | Yang et al 2019 |
| 1. PRKACA
 | Simpson et al 2008 |
| 1. PRKAR1A
 |  |
| 1. PRKD1
 | Luef et al 2016 |
| 1. PTCH1
 | Lisovsky et al 2016 |
| 1. PTK6
 | Ito et al 2016, Qiu et al 2018 |
| 1. PTPN11
 | Xu et al 2022 |
| 1. PTPN13
 | Hamyeh et al 2020 |
| 1. PTPRB
 | Weng et al 2019 |
| 1. PTPRT
 | Wang et al 2019 |
| 1. OKI
 | Wang et al 2021 |
| 1. RAC1
 | Tang et al 2020 ,Dipersio C.M 2007 |
| 1. RAD21
 | Gou et al 2022 |
| 1. RBM10
 | Cao et al 2023 , Cao et al 2022 |
| 1. RECQL4
 | Guo et al 2020 |
| 1. RET
 | Lisse et al 2020 |
| 1. RHOA
 | Desai et al 2004 |
| 1. RNF43
 | Radaszkiewicz et al 2021 |
| 1. RPL10
 | Heter et al 2019 |
| 1. RPL5
 | Zhang et al 2022 |
| 1. RUNX1
 | Li et al 2019 |
| 1. SALL4
 | Erickson et al 2016 |
| 1. SDHA
 |  |
| 1. SETBP1
 | Kimball et al 2019 |
| 1. SETD2
 | Li et al 2021 |
| 1. SETDB1
 | Han et al 2020, Zakharova et al 2022 |
| 1. SF3B1
 | Guo et al 2022, Randazzo et al 2021 |
| 1. SH2B3
 | Toma et al 2022 |
| 1. SIX2
 | Noizet et al 2016 |
| 1. SMAD2
 | Tomikawa et al 2012 |
| 1. SMAD3
 | Ashcroft et al 1999 |
| 1. SMARCA4
 | Kim et al 2021 |
| 1. SMARCB1
 | Hong et al 2021 |
| 1. SMARCD1
 | Tamai et al 2022 |
| 1. SMO
 | Frech et al 2022 |
| 1. SOCS1
 | Feng et al 2016 |
| 1. SPEN
 | Li et al 2020 |
| 1. SPOP
 | Tan et al 2019 , Chen et al 2022 |
| 1. SRSF2
 | Yu et al 2020 |
| 1. STAG2
 | Surdez et al 2021 |
| 1. STAT5B
 | Bernaciak et al 2009 |
| 1. STK11
 | Malhotra et al 2022 |
| 1. SUFU
 | Yang et al 2020 |
| 1. TBL1XR1
 | Liu et al 2015 |
| 1. TBX3
 | Ichijo et al 2017, Ichijo et al 2021 |
| 1. TENT5C
 | Kazazian et al 2020 |
| 1. TET2
 | Tan et al 2016 |
| 1. TGFBR2
 | Tauriello et al |
| 1. TNFAIP3
 | Shamilov et al 2020 |
| 1. TNFRSF14
 | Li et al 2021 |
| 1. TP63
 | Harazono et al 2022 |
| 1. TRAF7
 | Zhang et al 2021 |
| 1. TRRAP
 | Huang et al 2021 |
| 1. TSC1
 | Squarize et al 2010 |
| 1. TSC2
 | Larson et al 2010 |
| 1. TSHR
 | Feng et al 2021 |
| 1. U2AF1
 |  |
| 1. UBR5
 | Shin et al 2020 |
| 1. USP8
 | Shin et al 2020 |
| 1. WT1
 | Lopez-Baez 2018 |
| 1. XPO1
 | Wang and Liu 2019 |
| 1. ZFHX3
 | Dayoub et al 2022 |
| 1. ZRSR2
 |  |

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Supplementary material table 3a:

Genes differentially expressed during various stages of wound healing (Cooper et al 2005) namely activation phase, early effecter, late effecter, stop signal, early inflammatory, late inflammatory and related inflammatory and their involvement in cancer: References giving evidence for involvement in cancer (at least one each) are tabulated.

Cooper, L., Johnson, C., Burslem, F., & Martin, P. (2005). Wound healing and inflammation genes revealed by array analysis of 'macrophageless' PU.1 null mice. *Genome biology*, *6*(1), R5. https://doi.org/10.1186/gb-2004-6-1-r5

|  |
| --- |
| Activation |
| 2510002C21Rik |  |
| Actc1 | Suresh, R., & Diaz, R. J. (2021). |
| Adh1 | Li et al 2007 |
| Adrb | Fjæstad et al 2022 |
| Agxt | Sun et al 2019 |
| Ak1 | Jan et al 2019 |
| Alox12b | Jiang et al 2020 |
| Atf3 | Thompson et al 2009 |
| Bcl10 | Kuo et al 2021 |
| Bhlhb2 |  |
| Bpgm | Li et al 2022 |
| Capn6 | Chen et al 2020 |
| Casp7 | Chaudhary et al 2016 |
| Ccne1 | Xu et al 2021 |
| Ccr4 | Li et al 2012 |
| Cirbp | Lu et al 2018 |
| Cish2 |  |
| Col5a2 | Wang et al 2021 |
| Col6a2 | Li et al 2022 |
| Col6a3 | Li et al 2018 |
| Csf1 | Richardsen et al 2015 |
| Csf3 | Saunders, et al 2021 |
| Csrp2 | Chen et al 2020 |
| Csrp3 | Zhuo et al 2022 |
| Cyr61 | Shi et al 2019 |
| Dusp1 | Liu et al 2014 |
| Egr1 | Saha et al 2021 |
| Fbln1 | Xiao et al 2014 |
| Figf | Marconcini et al 1999 |
| Fkbp5 | Li et al 2011 |
| Fosb | Tang et al 2016 |
| Fosl1 | Sobolev et al 2022 |
| Gatm | Zhang et al 2021 |
| Gem |  |
| Glns | Yu et al 2021 |
| Gsto1 | Manupati et al 2019 |
| H1f2 |  |
| Has1 | Liu et al 2015 |
| Hegfl |  |
| Hexa |  |
| Hspb2 | Yu et al 2020 |
| Icam1 | Reina et al 2017 |
| Ier2 | Kyjacova et al 2021 |
| Irf1 | Karki et al 2020 |
| Irs2 | Jeong et al 2018 |
| Junb | Konish et al 2008. |
| Klf9 | Zhong et al 2018 |
| Mail |  |
| Mest | Wang et al 2021 |
| Mfap2 | Xu et al 2022 |
| Mkrn1 | Ko et al 2012 |
| Myc | Duffy et al 2021 |
| Nfkbia | Bredel et al 2013 |
| Nr4a1 | Hedrick et al 2015 |
| Per1 | Liu et al 2021 |
| Per2 | Xiong et al 2018 |
| Prom | Saha et al 2020 |
| Pscd3 | Nagahama et al 2010 |
| Ptgs2 | Saindane et al 2020 |
| Sele | Li et al 2021 |
| Serpinh1 | Wang et al 2022 |
| Snca | Wu et al 2022 |
| Tnni1 | Cong et al 2022 |
| Zfp36 | Chen et al 2020 |
| EARLY EFFECTOR |
| Acadl | Zhao et al 2020 |
| Adar | Xu et al 2018 |
| Adcy4 | Fan et al 2019 |
| Aldh1a3 | Yamashita et al 2020 |
| Ankrd2 | Bean et al 2014 |
| Areg | Xu et al 2016 |
| Car2 | Jie et al 2021 |
| Car4 |  |
| Cenpb | McGovern et al 2012 |
| Crabp2 | Feng et al 2019 |
| Crap |  |
| Ctla2a | Feng et al 2019 |
| Ddef1 | Justis P et al 2005 |
| Dnajc3 |  |
| Eps8 | Li et al 2013 |
| Ereg | cheng et al 2021 |
| Ets1 | Kim et al 2018 |
| Fgfrp |  |
| Fgl2 | Feng et al 2020 |
| Fin14 |  |
| Fth | Hu et al 2021 |
| Fv1 | Geisen et al 2015 |
| Fxyd5 | Raman et al 2015 |
| Galgt1 | Singhal et al 2015 |
| Gch |  |
| Gjb6 | Son et al 2020 |
| Gp38 | Del et al 2014 |
| Gpx3 | Nirgude et al 2021 |
| H2-T22 | Mei et al 2021 |
| Hck | Musumeci et al 2015 |
| Hif1a | Wu et al 2019 |
| Hmga2 | Mansoori et al 2021 |
| Hmgcr | Göbel et al 2019 |
| Hnrpdl | Zhang et al 2018 |
| Igh-8 | Waugh et al 2008 |
| Il1r2 | Zhang et al 2019 |
| Il4ra | Bednarz-Misa et al 2020 |
| Il8rb | Sun et al 2022 |
| Itga6 | Brooks et al 2016 |
| Klc1 | Moamer et al 2019 |
| Lama3 | Feng et al 2021 |
| Lamb3 | Zhu et al 2020 |
| Lamc2 | Liu et al 2021 |
| Lbp | Meng et al 2021 |
| Lgals9 | Armenta-Castro et al 2020 |
| Ltbr | Lau et al 2014 |
| Ly6a | Upadhyay et al 2019 |
| Ly6e | Yeom et al 2016 |
| Mal | Lara-Lemus R.2019 |
| Map3k1 | Pham et al 2013 |
| Map4k4 | Gao et al 2017 |
| Mapk6 | Cai et al 2021 |
| Mmp11 | Gobin et al 2019 |
| Mt1 | Si et al 2018 |
| Myd88 | Zhu et al 2020 |
| Myf6 | Arons et al 2020 |
| Nat2 | Zhu et 2021 |
| Nfh |  |
| Osp94 |  |
| Pdgfra | Wu et al 2022 |
| Pex5 | Dahabieh et al 2018 |
| Pfkp | Shen et al 2020 |
| Plaur | Liu et al 2021 |
| Pld1 | Zhang et al 2014 |
| Pp11r |  |
| Pros1 | Al Kafri, N., &Hafizi, S.2019 |
| Ptges | Wang et al 2019 |
| Ptgis | Sadler et al 2016 |
| Rbp1 | Gao et al 2020 |
| Rnase4 | Vanli et al 2022 |
| Rock2 | Dourado et al 2018 |
| S100a6 | Wang et al 2021 |
| S3-12 |  |
| Saa1 | Takehara et al 2020 |
| Samhd1 | Schott et al 2022 |
| Sdcbp | Das et al 2020 |
| Serpina3c |  |
| Serpinb2 | Chen et al 2022 |
| Serpinb4 | Izuhara et al 2018 |
| Serpine1 | Wang et al 2022 |
| Serping1 | Peng et al 2018 |
| Sgk | Bruhn et al 2010 |
| Sh3yl1 | Blessing et al 2015 |
| Slc11a1 | Zhu et al 2022 |
| Sod2 | Kim et al 2017 |
| Sod3 | Carmona et al 2020 |
| Spi12 |  |
| Sprr2a | Specht et al 2013 |
| Sprr2b | Yao et al 2021 |
| Stat1 | Zhang et al 2017 |
| Stat3 | Zou et al 2020 |
| Stk2 | Tsugeno et al 2014 |
| Tac1 | Reddy et al 2009 |
| Tapbp |  |
| Tgm2 | Malkomes et al 2021 |
| Tgm3 | Feng et al 2020 |
| Thbd | Mohamed et al 2018 |
| Thy1 | Hu et al 2020 |
| Trim25 | Takayama et al 2018 |
| Tubb5 |  |
| Vcam1 | Zhang et al 2020 |
| Vdr | Thorne et al 2008 |
| Xdh | Takayama et al 2018, Chen et al 2017 |
| LATE EFFECTOR |
| Alox15b | The human protein atlas |
| Anxa8 | The human protein atlas |
| Chi3l1 | Kawada et al 2012 |
| Ctsb | Peng S et al 2021 |
| Cxcl5 | Deng et al 2022 |
| Cyp1b1 | Mckay et al 1995 |
| Defb1 | Lee et al 2016 |
| Gsta4 | Zhang et al 2022 |
| Gyk |  |
| Hp | Tai et al 2017 |
| Ifi204 | Dauffy et al 2006 |
| Iigp |  |
| Il1rl1 | Wang et al 2020 |
| Isg15 | Han et al 2018 |
| Isgf3g | Weihua et al 1997 |
| Krt1-16 | Han et al 2021 |
| Krt2-6a |  |
| Krt2-6b |  |
| Lrg | Takemoto et al 2015 |
| Mmp13 | Huang et al 2010. |
| Mmp3 | Suhaimi et al 2020 |
| Mmp9 | Joseph et al 2020 |
| Osmr | Lee et al 2021 |
| Procr | Wang et al 2018 |
| Prss18 | Bao et al 2019 |
| Psmb8 | Chen et al 2021 |
| S100a8 | Huang et al 2020 |
| Saa1 | Takehara et al 2020 |
| Saa3 | Djurec et al 2018 |
| Serpina3n |  |
| Slc2a1 | Liu et al 2022 |
| Slpi | Wei et al 2020 |
| Sprr1b | Sasahira et al 2021 |
| Sprr2a | Specht et al 2013 |
| Sprr2d | The human protein atlas |
| Sprr2f | Contreras et al 2010 |
| Sprr2h | Hudlikar et al 2020 |
| Timp1 | Schoeps et al 2021 |
| Tnc | Yoshida et al 2015 |
| Usp18 | Pinto-Fernandez et al 2021 |
| STOP |
| Actc1 | Suresh, R., & Diaz, R. J.2021 |
| Acvr2 | Jung et al 2007 |
| Alcam | Darvishi et al 2020 |
| Amd2 | Hollander et al 2015 |
| Apoe | Kemp et al 2021 |
| Atp7a | Li et al 2016 |
| Bmi1 | Cao et al 2011 |
| Bmpr1a | Fernandez-Rozadilla et al 2013 |
| Calm4 |  |
| Cbx3 | Niu et al 2022 |
| Cbx5 | He et al 2021 |
| Ccne2 | Taghavi, et al 2016 |
| Cd2ap | Xie et al 2022 |
| Cdh6 | Meng et al 2022 |
| Chgb | Weisbrod et al 2013 |
| Cldn1 | Bhat et al 2020 |
| Crem | Yu et al 2021 |
| Ctse | Ye et al 2021 |
| Cyp51 |  |
| Diap3 |  |
| Dlx3 | Bajpai et al 2021 |
| Dmd | Jones et al 2021 |
| Dsc3 | Cui et al 2019 |
| Efnb1 | Shi et al 2021 |
| Egfr | Uribe et al 2021 |
| Emp2 | Chung et al 2017 |
| Gata3 | Takaku et al 2015 |
| Grpel2 | Lai et al 2021 |
| Hba-a1 |  |
| Hbb-b1 | Zheng et al 2017 |
| Hmgcr | Göbel, et al 2019 |
| Hsp105 | Nosaka et al 2021 |
| Hsp70-2 | Jagadish et al 2016 |
| Itga4 | Mo et al 2022 |
| Itpr5 |  |
| Kif1b | Ando et al 2019 |
| Klf3 | Wang et al 2019 |
| Krt1-1 |  |
| Krt1-2 |  |
| Krt1-24 |  |
| Krt1-24 |  |
| Krt1-3 |  |
| Krt1-c29 |  |
| Krt2-1 |  |
| Krt2-10 |  |
| Krt2-18 |  |
| Krt2-19 |  |
| Krt2-6g |  |
| Krtap6-1 |  |
| L1Md-Tf29 |  |
| Matr3 | Yang et al 2020 |
| Mbd4 | Tanakaya et al 2019 |
| Mrps15 | The human protein atlas |
| Msr1 | Wang et al 2021 |
| Msx3 |  |
| Mt4 | Pai et al 2015 |
| Mtap6 | Mamoor, S. 2021 |
| Narg1 | Bae et al 2013 |
| Ncl | Berger et al 2015 |
| Nfia | The human protein atlas |
| Nfic | The human protein atlas |
| Nfyb | Fang et al 2018 |
| Nol5 |  |
| Npy6r | Mei et al 2022 |
| Nr3c1 | Wang et al 2023 |
| Nrg3 | Li et al 2021 |
| Pcdh7 | Zhang et al 2021 |
| Pdi3 |  |
| Plxna3 | Gabrovska et al 2011 |
| Pnn | Zhang et al 2022 |
| Ppp1r3c | Lee et al 2015 |
| Prss12 | Liu et al 2019 |
| Ptgfrn | Aguila et al 2019 |
| Ptprr | Wang et al 2019 |
| Pura | The human protein atlas |
| Pvrl3 | Sun et al 2018 |
| Rad50 | Li et al 2021 |
| Rev1I | Chatterjee et al 2020 |
| Rpo1-1 |  |
| Rps2 | Wang et al 2009 |
| Rps24 | Wang et al 2015 |
| Rps27a | Wang et al 2014 |
| S100a3 | Zhang et al 2021 |
| Scd1 | Katoch et al 2022 |
| Sdfr2 |  |
| Sh3d19 | The human protein atlas |
| Sox2 | Mamum et al 2020 |
| Spr | Wu et al 2020 |
| Sprr1b | Sasahira et al 2021 |
| Stag1 | van der Lelij et al 2017 |
| Top2b | Uusküla-Reimand, L., & Wilson, M. D. 2022 |
| Tpt1 | Chen et al 2013, National library of medicine |
| Tyrp1 | Jha et al 2021 |
| Ube3a | Zheng et al 2021 |
| Utx | Wang et al 2019 |
| Wnt5a | Asem et al 2016 |
| Zfa |  |
| Zfp26 |  |
| Zfp292 | Takeda et al 2015 |
| Zfp46 |  |
| Zfp62 |  |
| Zfp97 |  |
| EARLY INFLAMMATORY |
| C3 | Lin et al 2014 |
| C3ar1 | Zou et al 2021 |
| Casp1 | Liu et al 2021 |
| Ccl9 | Yan et al 2015 |
| Ccr1 | Zilio et al 2022 |
| Cd14 | Cheah et al 2015 |
| Cd53 | Dunlock et al 2022 |
| Clecsf8 | Raskov et al 2021 |
| Cxcl10 | Reschke et al 2021 |
| Cxcl2 | Zhang et al 2021 |
| Dab2 | Adamson et al 2016 |
| Fpr-rs2 |  |
| Gbp2 | Yu et al 2020 |
| Gp49a |  |
| Ifi203 |  |
| Ifi204 | Dauffy et al 2006 |
| IL-17 | Vitiello et al 2020 |
| Il1b | Rébé, C., &Ghiringhelli, F. (2020) |
| Irg1 | Papathanassiu et al 2021 |
| Lcp1 | Zeng et al 2021 |
| Mcpt7 |  |
| Mpeg1 | Bayly et al 2020 |
| Mrc1 | Huang et al 2022 |
| Mx1 | Aljohani et al 2020 |
| Pfc | Hingorani, et al 2020 |
| Pglyrp | Li et al 2021 |
| Prg | Liu et al 2022 |
| S100a9 | Gebhardt et al 2006 |
| Slfn2 | Mavrommatis et al 2013 |
| Spp1 | Gao et al 2022 |
| Sqrdl | Liu et al 2020 |
| LATE INFLAMMATORY |
| Acadm | Ma et al 2021 |
| Adam9 | Lin et al 2014 |
| Agtr2 | The human protein atlas |
| Ars2 | Chen et al 2018 |
| B2m | Zhao et al 2021 |
| C4 |  |
| Cacybp | Li et al 2022 |
| Casp8ap2 | Myacheva et al 2023 |
| Ccr2 | Fein et 2020 |
| Cidea | Laurencikene et al 2008 |
| Clecsf6 |  |
| Clk | Tam et 2020 |
| Clk4 | Kang et al 2022 |
| Cops2 | Alves et al 2020 |
| Coq7 | Brea- Calvo et al 2006 |
| Ctss | Wilkinson et al 2019 |
| Cyp2b19 | Heslby et al 2008 |
| Dbt | Miao et al 2023 |
| Eif3 | Yin et al 2018 |
| Eif4a2 | Chen et al 2019 |
| Fin16 |  |
| Fmr1 | Hu et al 2022 |
| Fnbp4 | Zhong et al 2018 |
| Gas5 | Yu, Y., &Hann, S. S. 2019 |
| Gbp3 | He et al 2021 |
| Gtf2h1 | Geng et al 2022 |
| Hells | Robinson et al 2019 |
| Hnrph1 | Liu et al 2021 |
| Ifi1 |  |
| Ifi202a |  |
| Ifit1 | Pidugu et al 2019 |
| Ifit2 | Lai et al 2022 |
| Ifit3 | Pidugu et al 2019 |
| Lzp-s |  |
| Mup3 |  |
| Nmyc1 | Beltran et al 2014 |
| Np220 |  |
| Nssr |  |
| Nucb2 | Suzuki et al 2012 |
| Pcee |  |
| Ppic | The human protein atlas |
| Ppicap |  |
| Prpk | Zykova et al 2018 |
| Ptbp2 | Chen et al 2022 |
| Rbmx | Yan et al 2021 |
| Rptn | The human protein atlas |
| Septin7 | Wang et al 2018 |
| Sfrs5 |  |
| Sh3bgrl | Zhang et al 2022 |
| Slfn3 | Mavrommatis et al 2013 |
| Sucla2 | Kohno et al 2020 |
| Tank | Revach et al 2020 |
| Tgtp |  |
| Thra | Kim et al 2013 |
| Ttrap | The human protein atlas |
| Uchl5 | Liu et al 2020 |
| Ucp1 | Huang et al 2022 |
| Wsb1 | Kim et al 2015 |
| Zac1 | Su et al 2020 |
| Zfp101 |  |
| Zfp118 |  |
| Zfp265 |  |
| Zfp97 |  |
| REALTED INFLAMMATORY |
| C1qa | Liang et al 2022 |
| C1qc | Chen et al 2021 |
| Ccl2 | O'Connor, T., &Heikenwalder, M. 2021 |
| Ccl7 | Liu et al 2018 |
| Cish3 |  |
| Cma2 |  |
| Cpa3 | Huang et al 1999 |
| Hdc | Nicoud et al 2020 |
| Il6 | Wang et al 2017 |
| Mcpt5 |  |
| Mup1 |  |
| Mup4 | Singh et al 2007 |
| MUPV |  |
| Ptx3 | Giacomini et al 2018 |
| Slc6a4 |  |
| Tnfip6 | Zhang et al 2021 |

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 Supplementary table 3b:

Gene expression in wound healing according to Deonarine et al 2007, along with evidence for the involvement of the genes in cancer. Studies giving evidence for the role in cancer are tabulated.

Deonarine, K., Panelli, M.C., Stashower, M.E. *et al.* Gene expression profiling of cutaneous wound healing. *J Transl Med* **5**, 11 (2007). https://doi.org/10.1186/1479-5876-5-11

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| --- | --- |
| 1. ACP5
 | Ren et al 2018 |
| 1. ACVRL1
 | Hanna et al 2018 |
| 1. ADAM10
 | Smith et al 2020 |
| 1. ADAM15
 | Xu et al 2021 |
| 1. ADAMTS3
 | Wu et al 2021 |
| 1. AP1S2
 | The Human Protein Atlas |
| 1. AQP3
 | Zhu et al 2018 |
| 1. ARG1
 | Arlauckas et al 2018 |
| 1. ARG2
 | Grzywa et al 2020 |
| 1. BCAP31
 | Han et al 2022 |
| 1. BCL7B
 | Yang et al 2021 |
| 1. BST2
 | Liu et al 2018 |
| 1. BTG1
 | Zhao et al 2020 |
| 1. CASP10
 | Oh et al 2010 |
| 1. CASP6
 | Hong et al 2020 |
| 1. CCL18
 | Korbecki et al 2020 |
| 1. CCL2
 | O’Connnor et al 2021 |
| 1. CCL20
 | Chen et al 2021 |
| 1. CCL22
 | Rohrle et al 2020 |
| 1. CCR7
 | Salem et al 2021 |
| 1. CD163
 | Matsubara et al 2021 |
| 1. CD3Z
 |  |
| 1. CD44
 | Xu et al 2020 |
| 1. CD6
 | Ruth et al 2021 |
| 1. CD68
 | Zhang et al 2022 |
| 1. CD81
 | Vences –Catalan et al 2022 |
| 1. CD86
 | Wennhold et al 2021 |
| 1. CD99
 | Yu et al 2022 |
| 1. CDW52
 | Salisbury et al 1994 |
| 1. CEBPB
 | Okazaki et al 2022 |
| 1. CLL-L3B
 |  |
| 1. CLSTN1
 | Chu et al 2017 |
| 1. COL5A3
 | Wang et al 2021 |
| 1. COX15
 | Zhang et al 2021 |
| 1. COX5A
 | Zheng et al 2020 |
| 1. COX5B
 | Gao et al 2015 |
| 1. CXCL16
 | Korbecki et al 2021 |
| 1. CXCL2
 | Zhang et al 2021 |
| 1. CXCL9
 | Neo, S. Y., & Lundqvist, A. (2020 |
| 1. DEDD
 | Ni et al 2019 |
| 1. DUSP22
 | Sasaki et al 2019 |
| 1. EBI2
 |  |
| 1. EDF1
 | The human protein atlas |
| 1. EGR3
 | Shin et al 2020 |
| 1. EVER1
 | Kalińska-Bienias et al 2016 |
| 1. F3
 | Zhu et al 2019 |
| 1. FCGR1A
 | Xu et al 2020 |
| 1. CD64
 |  |
| 1. FCGR2A
 | Michelakos et al 2022 |
| 1. FCGR3A
 | Li et al 2022 |
| 1. G1P2
 | Yu et al 2020 |
| 1. G1P3
 | Cheriyath et al 2018 |
| 1. HLA-B
 | Michelakos et al 2022 |
| 1. HLA-C
 | Michelakos et al 2022 |
| 1. HLA-DOA
 | Yu et al 2020 |
| 1. HLA-DQB2 --
 | Wu et al 2022 |
| 1. HLA-E -
 | Marin et al 2003, Borst et al 2020 |
| 1. HLA-F
 | Wuerfel et al 2020 |
| 1. HSF2
 | Chen et al 2022 |
| 1. HSPA1B
 | Wang et al 2021 |
| 1. ICSBPI
 |  |
| 1. IFIT1
 | Pidugu et al 2019 |
| 1. IFITM1
 | Yu et al 2015 |
| 1. IFITM2
 | Liu et al 2022 |
| 1. IFITM3
 | Chu et al 2022 |
| 1. IFNG
 | Zaidi M.R 2019 |
| 1. IFNGR1
 | Du et al 2022 |
| 1. IFRD2
 |  |
| 1. IK
 | Gao et al 2021 |
| 1. IL10RB
 | Yoo et al 2011 |
| 1. IL13
 | Terebe et al 2004 |
| 1. IL15RA
 | Xu et al 2021 |
| 1. IL1R1 -
 | Zhang et al 2020 |
| 1. IL24 --
 | Qiu et al 2020 |
| 1. IL4R --
 | Bankaitis et al 2015 |
| 1. ITGAV
 | Cheuk et al 2020 |
| 1. ITGAX
 | Wang et al 2019 |
| 1. KLF4 -
 | Yu et al 2011 |
| 1. KLRD1
 |  |
| 1. LDHA --
 | Feng et al 2018 |
| 1. LNK --
 | Lv et al 2020 |
| 1. LTBP4
 | Su et al 2021 |
| 1. LTF
 | Zhao et al 2021 |
| 1. LY64 -
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| 1. LY75 --
 | Mehdi et al 2020 |
| 1. MAP2K3 --
 | J et al 2014 |
| 1. MAP4K2 --
 | Li et al 2022 |
| 1. MAPBPIP --
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| 1. MAPKAPK2
 | Soni et al 2019 |
| 1. MAPKAPK3
 | Ren et al 2021 |
| 1. MIF
 | O’Reilly et al 2016 |
| 1. MMP11
 | Ma et al 2021 |
| 1. MMP2
 | Wang et al 2018 |
| 1. MRC2
 | Zhao et al 2022 |
| 1. MT1G
 | Wang et al 2019 |
| 1. MT2A
 | Shimizu et al 2021 |
| 1. MT3
 | Koh JY& Lee SJ. 2020 |
| 1. NMI
 | Feng et al 2017 |
| 1. NUP153
 | Wu et al 2019 |
| 1. P4HB
 | Wu et al 2021 |
| 1. PECAM1 --
 | Cao et al 2021 |
| 1. PLAU --
 | Fang et al 2021 |
| 1. PSMB2 --
 | Liu et al 2022 |
| 1. PSMB9 --
 | Li et al 2020 |
| 1. PSMC4 --
 | Kao et al 2021 |
| 1. PSMD13 --
 | Kexuan et al 2022 |
| 1. QSCN6 --
 |  |
| 1. RAB14 --
 | Guo et al 2017 |
| 1. RAB31 --
 | Chua et al 2015 |
| 1. RAB35 --
 | Shaughnessy et al 2018 |
| 1. S100A4 --
 | Fei et al 2017 |
| 1. SCARB1 --
 |  |
| 1. SCARB2 --
 | Feng et al 2022 |
| 1. SOCS3 --
 | Dai et al 2022 |
| 1. SRCRB4D --
 |  |
| 1. STAT1 -
 | Zhang et al 2017 |
| 1. STAT5A -
 | Rani, A and Murphy J 2016 |
| 1. TGF-
 | Massague J 2008 |
| 1. TGFB1 --
 | Sun et al 2021 |
| 1. TIA1
 | Dolicka et al 2022 |
| 1. TIMP1 --
 | Schoeps et al 2021 |
| 1. TIMP2 --
 | Wang et al 2022 |
| 1. TLR 6
 | Babu Prasad ,S & Kumar R 2021 |
| 1. TLR1 --
 | Babu Prasad ,S & Kumar R 2021 |
| 1. TLR2 --
 | Di Lorenzo et al 2020 |
| 1. TLR3
 | Muresan et al 2020 |
| 1. TLR5
 | Cai et al 2011 |
| 1. TLR7
 | Babu Prasad ,S & Kumar R 2021 |
| 1. TNFRSF12A --
 | Yang et al 2018 |
| 1. TNFSF13 --
 | Lin et al 2020 |
| 1. TU3A --
 | Awakura et al 2008 |
| 1. VEGF --
 | Carmeliet et al 2005 |
| 1. VEGFB --
 | Yang et al 2015 |
| 1. VEGFC --
 | Kong et al 2021 |
|  135. VIM --  | Mohebi et al 2020 |
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