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Advanced hepatocellular carcinoma

Ivermectin has the potential to inhibit metastasis and target HCC stem cell functions. Mechanism studies correlated well with cellular phenotypes observed in ivermectin-treated cells, and demonstrated inhibition of mTOR/STAT3 pathway, suppression of epithelial mesenchymal transition (EMT) and reduced expression of stem cell markers.

This study showed that the potent anti-HCC activities of ivermectin and its multiple targets on essential oncogenic pathways. Our findings provide preclinical evidence to initialize clinical trial using ivermectin and sorafenib for treating advanced HCC.[1]

Alzheimers

Ivermectin exhibits a protective effect against oxidative stress-induced Alzheimer's disease. The results showed a significant reduction in activities of SOD and GPx, in addition to significant elevation in carbonyl protein level compared to NC group.[2]

Amyotrophic lateral sclerosis

Ivermectin inhibits AMPA receptor-mediated excitotoxicity in cultured motor neurons and extends the life span of a transgenic mouse model of amyotrophic lateral sclerosis. In vitro data indicate that this protective mechanism is due to the potentiation by ivermectin of an effect of ATP mediated by the P2X4 receptor.[3]

Asthma

Similarly, in mouse asthma model stimulated with ovalbumin, ivermectin significantly suppresses airway inflammation and hyperactivity via the inhibition of cytokines production and immune cells' recruitment.[4]

Bile duct cancer

Anti-parasitic drug ivermectin exhibits potent anticancer activity against gemcitabine-resistant cholangiocarcinoma in vitro.[5]

Breast cancer

Ivermectin kills breast cancer cells through a mixed apoptotic and necrotic mechanism.[6] In vitro data obtained in breast cancer cells also indicated that the P2X4R/P2X7R/Pannexin-1 sensitivity to ATP can be modulated by a Food and Drug Administration (FDA)-approved anti-parasitic agent named ivermectin.[7,8]

Buruli virus

Aka *Mycobacterium ulcerans*. It also inhibits replication of the scary flesh-eating virus that's around Melbourne at the moment.[9]

Cancer cells in general

Ivermectin causes cell death in cancer cell lines by inducing PAK1-mediated cytostatic autophagy, caspase-dependent apoptosis and immunogenic cell death (ICD) through the modulation of some pathways, including the WNT-T cell factor (TCF), Hippo and Akt/mTOR pathways. It can affect the

growth and proliferation of cancer cells. In addition, ivermectin induces the multidrug resistance protein (MDR), has potent anti-mitotic activity, targets angiogenesis and inhibits cancer stem-like cells (CSCs).[10]

Ivermectin reverses the drug resistance in cancer cells, it increased the sensitivity of tumor cells, including the drug-sensitive or resistant cancer cells, solid tumor cells or leukemia cells, to the chemotherapeutic drugs.[11]

Chlamydia

Other studies and experimental evidence demonstrated ivermectins effectiveness against Chlamydia trachomatis.[12]

Colon cancer

Selamectin (topical Ivermectin), a drug widely used in veterinarian medicine (Nolan & Lok, 2012), **is ten times more potent** acting in the nanomolar range. Finally, Ivermectin has in vivo efficacy against **human colon cancer** xenografts sensitive to TCF inhibition with no discernable side effects.[13]

Covid

In the setting of SARS-CoV-2 infection, ivermectin seems to be an inhibitor of both viral entrance and replication. It's a potent suppressor of the inflammatory response prompted by bacterial LPS. This anti-inflammatory effect is imparted by attenuating the production of pro-inflammatory cytokines such as TNF-α, IL-1ß, and IL-6.[14]

Flavivirus

Ivermectin is a potent inhibitor of flavivirus replication specifically targeting NS3 helicase activity.[15]

Quantitative proteomics revealed that ivermectin-related proteins are involved in four statistically significant antiviral pathways, including human cytomegalovirus (HCMV), human papillomavirus (HPV), Epstein–Barr virus (EBV), **human immunodeficiency virus 1 (HIV1)**, and COVID-19 infection pathways.

Foot and Mouth Disease

Viral inhibition assays using the non-cytotoxic concentration of ivermectin were performed to check the antiviral potential of ivermectin on different stages of virus replication. At 2.5 µM and 5 µM concentrations of ivermectin, **the virus titer was reduced significantly** ($p < 0.001$) by two to three log in all three strains of viruses at both non-toxic concentrations.[16]

Glioblastoma

Anthelmintic drug ivermectin **inhibits angiogenesis**, growth and survival of glioblastoma through inducing mitochondrial dysfunction and oxidative stress.[17] Angiogenesis inhibitors are unique cancer-fighting agents because they block the growth of blood vessels that support tumor growth rather than blocking the growth of tumor cells themselves.[18] Glioblastomas are a fast growing type of brain tumour. They are the most common type of cancerous (malignant) brain tumour in adults and are a type of brain tumour that belongs to a group of brain tumours called gliomas.[19]

Hepatitis B

Ivermectin Inhibits HBV Entry into the Nucleus by Suppressing KPNA2. [20]

Hepatitis E

Ivermectin effectively inhibits hepatitis E virus replication, requiring the host nuclear transport protein importin α1.[21]

HIV & Dengue

Ivermectin is a specific inhibitor of importin α/β-mediated nuclear import able to **inhibit replication of HIV-1 and dengue virus**. Biochemical J. 2012;443:851–6.

Ivermectin inhibits LPS-induced production of inflammatory cytokines and improves LPS-induced survival in mice. Inflamm Res. 2008;57:524–9.[22,23]

Hypoxia and Hypoglycemia

Ivermectin protects against hypoxia/hypoglycemia.[24] Ivermectin inhibits the hypertrophic response of human induced pluripotent stem cell-derived cardiomyocytes, as well as exhibiting protection against mitochondrial ATP decline and cardiomyocyte hypertrophy,[25] and maintains mitochondrial ATP levels under hypoxia in cardiomyocytes.[26]

Improves motor deficits in electric nerve conduction

Ivermectin improves motor deficits in EAE and electric nerve conduction. Ivermectin enhances the degradation of myelin debris. Finally, ivermectin favours remyelination in organotypic cerebellar slices in the lysolecithin (LPC)-induced model of demyelination.[27,28]

Remyelination is the phenomenon by which new myelin sheaths are generated around axons in the adult central nervous system (CNS).[29]

Leukaemia

Antibiotic ivermectin selectively **induces apoptosis in chronic myeloid leukemia** through inducing mitochondrial dysfunction and oxidative stress.[30]

Liver fibrosis

Ivermectin attenuates CCl4-Induced liver fibrosis in mice by suppressing hepatic stellate cell activation.[31]

[It] **Ivermectin attenuates liver injury,** reduces plasma levels of transaminase, suppressed hepatic accumulation of macrophages, **inhibits the production of proinflammatory factors**, and alleviates the expression of fibrotic genes. All of the above data demonstrates the **beneficial effects of ivermectin on liver fibrosis**.[32]

Lung cancer

The ability of systemic Ivermectin to also block lung cancer growth in vivo supports the possible use of Ivermectin in particular, and other macrocyclic lactones such as Selamectin (topical Ivermectin) in

general, as WNT-TCF pathway response blockers to combat WNT-TCF-dependent human diseases including **cancers of the intestine, breast, skin, and lung.**[33]

Melanoma

Ivermectin can suppress almost completely the growth of **human melanoma** and a few other cancer xenografts in mice.[34]

Multiple Sclerosis

Ivermectin administration reduced the clinical symptoms of EAE mice by **preventing the infiltration of inflammatory cells into the CNS**. Ivermectin attenuates the pathogenesis of EAE, indicating that it may be a promising option for T-cell-mediated autoimmune diseases such as MS.[35]

Ivermectin induces autophagy and release of ATP and HMGB1, key mediators of inflammation.[36]

There is **reversible silencing of neuronal excitability** in behaving mice by a genetically targeted, ivermectin-gated Cl- channel.[37]

In both animal models of MS and in patients, cortical neurons show abnormal excitability levels.[38]

Myeloma

Combinations of ivermectin with proteasome inhibitors **induce synergistic lethality in multiple myeloma**.[39] Myeloma is a type of blood cancer that develops from plasma cells in the bone marrow.[40]

Neuropathic pain

Established mechanical and cold pain-related hypersensitivity generated by the spared nerve injury model of **neuropathic pain was reversed by ivermectin treatment**.[41]

Nerve pain

Peripheral (axonal) targeting of the ivermectin completely silenced the cell body response to depolarization of the neurites. A low dose of IVM acting on the modified GluCl channel can silence mammalian CNS neurons in vivo for days. Because AAV-directed channel expression can be targeted to neurons of interest (ie those critical to the transmission of the 'pain' message), the therapeutic window can be greatly increased, even after systemic IVM administration, compared to traditional systemic analgesic drug approaches. (Lerchner et al., 2007).[41]

Peripheral (axonal) targeting of the ivermectin completely silenced the cell body response to depolarization of the neurites, as it did when applied directly to the cell body.[42]

Ovarian cancer

Ivermectin suppresses the growth of a variety of human ovarian cancer cell lines in vitro by inactivating the oncogenic kinase PAK1 somehow (Hashimoto H, et al. Drug Discov Ther. 2009;3:243-246). This kinase is now known to be essential for the growth of more than 70% of all human cancers including breast, prostate, pancreatic, colon, gastric, lung, cervical, thyroid cancers as well as hepatoma, glioma, melanoma, MM (multiple myeloma) and NF (neurofibromatosis) tumors.[43]

Oesophageal squamous cell carcinoma

Ivermectin suppresses tumour growth and metastasis through degradation of PAK1 in oesophageal squamous cell carcinoma.[44]

Prostate cancer

Ivermectin binds to HSP27 phosphorylation pocket and prevents its interaction with misfolded proteins. It potently inhibits HSP27 phosphorylation in lung and prostate cancer (Figure 2A) as well as bladder and breast cancer. [45]

Rabies

Ivermectin impedes the replication of DNA viruses, like pseudorabies, through the disruption of the nuclear localization of the protein UL42, a key mediator of DNA synthesis.[46]

In addition to its well-known antiparasitic activity, ivermectin exhibits antineoplastic and antiviral properties.[47,48]

Renal Cancer

Antibiotic ivermectin preferentially targets renal cancer through inducing mitochondrial dysfunction and oxidative damage.[49,50] Further, Ivermectin significantly inhibits proliferation and induces apoptosis in multiple RCC cell lines that represent different histological subtypes and various mutation status. Importantly, ivermectin is significantly less or ineffective in normal kidney cells compared with RCC cells, demonstrating the preferential toxicity of ivermectin to RCC. Ivermectin also significantly inhibits RCC tumor growth in vivo.[51]

RNA Viruses

Several studies reported antiviral effects of ivermectin on RNA viruses such as Zika, dengue, yellow fever, West Nile, Hendra, Newcastle, Venezuelan equine encephalitis, chikungunya, Semliki Forest, Sindbis, Avian influenza A, Porcine Reproductive and Respiratory Syndrome, Human immunodeficiency virus type 1, and severe acute respiratory syndrome coronavirus 2.[52]

Schizophrenia

Experimental cancer (PAK inhibitor) drug reverses schizophrenia in adolescent mice.[53] Ivermectin is a PAK inhibitor.[54]

Skin inflammation

IVM is endowed with topical anti-inflammatory properties that could have important applications for the treatment of T-cell-mediated skin inflammatory diseases.[55]

Spinal cord injury

Ivermectin (IVM-MWCNT (multi-walled carbon nanotube)) might be a novel treatment in spinal cord injury, which could act through decreasing the oxidative stress and increase the polarization of M1 in comparison to M2 macrophages.[56]

Another study showed that both IVM (Ivermectin) and IVM-MWCNT (0.1 mg/kg per day up to 3 days) after induction of spinal cord injury significantly enhanced the spinal cord injury outcomes, with the evidence of significant improvement in all experimental task including locomotion and neuropathic tests.[57]

Staph infections

Ivermectin has an anti-bacterial effect against certain S. aureus isolates.[58]

Stroke and transient cerebral ischemia-reperfusion

Research results indicated that three days of treatment with **Ivermectin reduced brain infarct size (**$P < 0.001$) and histopathological changes such as cerebral leukocyte accumulation and edema ($P < 0.05$).[59] Treatment with ivermectin also decreased myeloperoxidase activity ($P < 0.01$), lipid peroxidation, and malondialdehyde levels ($P < 0.05$) while increasing AMPK activity ($P < 0.001$), and improved memory and learning compared to the untreated IR group. These effects likely occurred via AMPK-dependent mechanisms. [60]

Strychnine toxicity

Ivermectin is an effective antidote of strychnine toxicity.[61]

Tuberculosis

It showed a bacterial killing effect against various species of Mycobacterium, including Mycobacterium tuberculosis. [12,62]

In Summary

Ivermectin is an antiviral, antibacterial, anti-inflammatory, antitumorigenic, anticoagulant, antibiotic and an antioxidant. It's effective against dengue, yellow fever, chikungunya, Zika, Avian flu A, West Nile, Hendra, Newcastle, Venezuelan equine encephalitis, Semliki Forest, Sindbis, Porcine Reproductive and Respiratory Syndrome and HIV1. It kills specific prostate cancer cells, lung cancer, colon cancer, ovarian cancer, throat cancer, breast cancers, leukaemia and Weil's disease. It's neuroprotective work to prevent oxidative stress, and other cell damage related neurological diseases including Alzheimer's, ALS, Motor Neuron Disease and Multiple Sclerosis.

Melatonin

Melatonin is uncommonly effective in reducing oxidative stress under a remarkably large number of circumstances. It achieves this action via a variety of means: direct detoxification of reactive oxygen and reactive nitrogen species and indirectly by stimulating antioxidant enzymes while suppressing the activity of pro-oxidant enzymes. In addition to these well-described actions, melatonin also reportedly chelates transition metals, which are involved in the Fenton/Haber–Weiss reactions; in doing so, melatonin reduces the formation of the devastatingly toxic hydroxyl radical resulting in the reduction of oxidative stress. Melatonin's ubiquitous but unequal intracellular distribution, including its high concentrations in mitochondria, likely aid in its capacity to resist oxidative stress and cellular apoptosis. There is credible evidence to suggest that melatonin should be classified as a mitochondria-targeted antioxidant.

Melatonin's capacity to prevent oxidative damage and the associated physiological debilitation is well documented in numerous experimental ischemia/reperfusion (hypoxia/reoxygenation) studies especially in the brain (stroke) and in the heart (heart attack). Melatonin, via its antiradical mechanisms, also reduces the toxicity of noxious prescription drugs and of methamphetamine, a drug of abuse.

Experimental findings also indicate that **melatonin renders treatment-resistant cancers sensitive** to various therapeutic agents and may be useful, due to its multiple antioxidant actions, in especially delaying and perhaps treating a variety of age-related diseases and dehumanizing conditions.

Melatonin has been effectively used to combat oxidative stress, inflammation and cellular apoptosis and to restore tissue function in a number of human trials; its efficacy supports its more extensive use in a wider variety of human studies.

The **uncommonly high-safety profile of melatonin** also bolsters this conclusion. It is the current feeling of the authors that, in view of the widely diverse beneficial functions that have been reported for melatonin, these may be merely epiphenomena of the more fundamental, yet-to-be identified basic action(s) of this ancient molecule.

Melatonin was found to reduce the formation of higher order oligomeric structures without affecting the overall aggregation kinetics of Tau.[63]

Melatonin has been shown to prevent tau hyperphosphorylation in cellular and animal models.[64]

Overall, melatonin administration shows mild anti-aggregation and cytoprotective effects.[65]

Melatonin and cardiac conditions including myocarditis

Inhibitory effect of melatonin on Mst1 ameliorates myocarditis through attenuating ER stress and mitochondrial dysfunction. [66]

Melatonin attenuates ER stress and mitochondrial damage in septic cardiomyopathy: A new mechanism involving BAP31 upregulation and MAPK-ERK pathway.[67]

Melatonin Alleviates Cardiac Function in Sepsis-Caused Myocarditis via Maintenance of Mitochondrial Function. [68]

Androgen receptor inhibition alleviated inflammation in experimental autoimmune.[69]

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The role of melatonin in chronic kidney disease.[70]

Kidney protective effects of melatonin.[71]

- Melatonin in chronic kidney disease: a promising chronotherapy targeting the intrarenal renin–angiotensin system.[72]
- Melatonin prevents kidney injury in a high salt diet-induced hypertension model by decreasing oxidative stress.[73]
- Melatonin Attenuates Cisplatin-Induced Acute Kidney Injury through Dual Suppression of Apoptosis and Necroptosis [2019]. Consistent with in vivo findings, melatonin dose-dependently decreased apoptosis and necroptosis in cisplatin-treated mouse renal tubular epithelial cells.[74]
- Effects of melatonin on liver and lung tissues of animals with bile duct ligation-induced hepatopulmonary syndrome. Many studies have shown that melatonin has anti-fibrosis effect, which can inhibit the development of pulmonary fibrosis by reducing oxidative stress. [75]
- Melatonin Inhibits Endoplasmic Reticulum Stress and Epithelial-Mesenchymal Transition ...
- Twenty-one days after BLM injection, lung fibrosis was evaluated. As expected, melatonin significantly alleviated BLM-induced pulmonary fibrosis.[76]
- Melatonin prevents kidney damage caused by metabolic complications of obesity.[77]
- Melatonin Prevents Chronic Kidney Disease-Induced Hypertension in Young Rat Treated with Adenine. [78]
- Inhibitory effect of melatonin on Mst1 ameliorates myocarditis through attenuating ER stress and mitochondrial dysfunction. [79]
- A Comparative Study on the Use of Alprazolam and Melatonin for Sleep Disturbances in Hemodialysis Patients found melatonin to be best choice, yet many researchers and scientists are still blind to the other amazing benefits of melatonin for CKD and ESRD.[80]

The Research Folders

1 - Melatonin - https://is.gd/tU2qf1
2 - N-acetylcysteine - https://is.gd/tNVOiw
3 - GABA - https://is.gd/OoCHIK
4 - Cinchona Bark - https://is.gd/OOz1vg
5 - Parasites and CNS issues - https://is.gd/LfPfWs
6 - Ivermectin - https://is.gd/KEJrTv
7 - Facemasks - https://is.gd/XtdJnl
8 - General Health - https://is.gd/mqsTGF - (all the other research in folders not listed above)
9 - MND and ALS and other neurological diseases - https://is.gd/hd7vOx
The end

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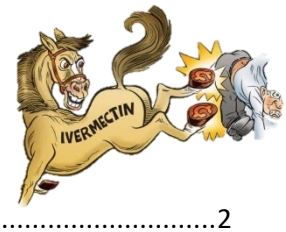

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Endnotes

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