

How VGCC Activation by Wireless Technologies Leads to Cancer

There are four types of evidence that EMFs, acting via increased Voltage Gated Calcium Channel Activity (VGCC), can cause cancer.

1. EMFs can act to attack the DNA of cells in ways that can cause cancer. EMFs act to produce single strand breaks in cellular DNA, to produce double strand breaks in cellular DNA and also oxidized bases in cellular DNA, of which 8-OH-guanine is the most commonly reported but there are also reports of several others. Each of these can have roles in causing cancer. The double strand breaks cause chromosome breaks and rearrangements. Single strand breaks produce copy number mutations. Oxidized bases lead to point mutations, where there is a specific change in a DNA base pair. Each of these have roles in causing cancer. Each of these DNA changes are caused by free radical breakdown products of peroxynitrite and are, therefore what are known as downstream effects of VGCC activation.
2. EMFs have been repeatedly reported to cause tumor promotion. There are two mechanisms I am familiar with for this to happen and there may be others. Tumor promotion can occur through tight junction disruption, something that also occurs in EMF disruption of the blood-brain barrier. EMFs probably do this via oxidants activating a transcription factor known as AP-1, which activates the synthesis of two matrix metalloproteinases, MMP-2 and MMP-9. The MMP's act, in turn to degrade the tight junction proteins -- this explains both EMF-mediated breakdown of the blood-brain barrier and also of tumor promotion. And the MMP's also have roles in metastasis, adding another possible type of action in carcinogenesis and progression. Another tumor promotion mechanism can be via protein kinase C activation -- this is the way that the tumor promoters phorbol esters are known to act. Intracellular calcium [Ca²⁺]_i can act via protein kinase C as can a number of other regulatory molecules including some hormones that are released via VGCC activation. These act, in turn, to increase the release of diacylglycerol which stimulate protein kinase C.
3. These mechanisms described in 1 & 2 are probably involved in inflammatory carcinogenesis, providing a third type of evidence.
4. There is also evidence showing that mutations causing excess activity of the T-type VGCCs can cause cancer, providing a 4th type of evidence.

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