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Editorial

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How did I Come Across into Cancer Field?

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At about one decade ago, my starting in research was related to skeletal muscle, including since its physiology until therapeutics for treating muscle wasting conditions. In my MSc studies, I developed a novel experimental model to studying muscle wasting conditions inherited to skeletal muscle, including screening of drugs for alleviating muscle atrophy. In fact, I have studied the role of Ca^{2+}/cAMP signalling interaction since then, considering we found that drugs which elevate cAMP levels may alleviate muscle wasting [1]. In my PhD studies, I shifted my studies to smooth muscle, herein studying the role of Ca^{2+}/cAMP signalling interaction in its physiology [2]. Herein, we confirmed that elevating cAMP levels may relax smooth muscle, but also enhance the release of transmitter from sympathetic neurons [2]. Here we have coined the term ´calcium paradox´ to explain why in some conditions, as when we use Ca^{2+} channel blockers (CCBs) in low concentrations, these drugs may enhance the contractions of smooth muscle, instead of inhibiting them! We concluded that the Ca^{2+}/cAMP signalling interaction properly explain this phenomenon! And the history has gone ahead! Considering CCBs are widely used in clinics as antihypertensive drugs, now we have the possibility to use such drugs for stimulating neurotransmitter release, thus of course these drugs could be used in medical problems related to neurotransmitter release deficit [3-6]. But, now you may ask: ok, but how is this related to cancer field? Now we have gone into the point: Ca^{2+}/cAMP signalling interaction has been now well accepted as a fundamental cellular process which exists in virtually many cells, governing the release of neurotransmitter and hormones, muscle contraction, and so on… Yes, there is evidence that such interaction also exists in cancer cells! Most importantly, there is a consensus that Ca^{2+} homeostasis is dysregulated in cancer cells, and strategies which aim to alleviate intracellular Ca^{2+} excess have been considered interesting approaches for inhibiting cancer progression [7,8]. Interestingly, increasing cAMP levels has also been demonstrated to reduce cancer progression [9]. Well, through manipulating Ca^{2+}/cAMP signalling interaction, we may alleviate intracellular Ca^{2+} excess, and also increase cAMP levels, a multitarget action, indeed [10-13]! I believe the most interest fact for this approach is the possibility of using CCBs, already approved for other clinical purposes, but now for another medical problem: cancer! In fact, I agree with the phrase: "novel approaches from old pharmaceuticals!"

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