

Colorectal Cancer 2018 & World Cancer 2018: Impact of deploying a genetic approach to stem cells opens-up new facets in the blank slates of our body - Jyoti Bhojwani - Devi Ahilya Vishwavidyalaya-Indore University, India

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Since the dawn of the post-genomic era (25 years back), applying a genetic approach to solving various intricate problems/ issues in research has taken-off even more swiftly than ever before. Spatio-temporal cues defined for certain critical components in a developmental pathway (involved in causing/progression of certain disease) provide a firm basis for detecting the order, hierarchy and “switching-off or on” of genes that regulate it. The various time-points, at which genes are switched on/off, clearly determines the fate of what a cell does in terms of being functional or non-functional, due to disruption of that specific pathway. Recent research-work in this area provides convincing evidence, towards identifying such components (associated with Wnt-signaling involved in colorectal cancer-CRC disease). These crucial elements indeed determined the genetic transformation of a “blank-slate” (“cells of origin” and/or putative “cancer stem cells”) or “primitive-state” epithelial cells to an intermediate adenoma/polyp (dysplastic), and later to a proliferative (hyperplastic) or cancerous (neoplastic) state. The idea is to re-iterate the power of genetics, in solving and filling the missing links of any developmental pathway involved in progression of a disease (in this case, CRC). A critical

temporal requirement of certain molecules [Caesin-Kinase I (CKI) and Human-Discs-large (hDlg)] was finally established and these proteins were identified as “early” and “late” acting molecules respectively, in a very crucial developmental event, that basically transforms “polyps” to full-fledged “carcinomas” (epithelial cancers) in colorectal tumors. The detection of these genetic and developmental parameters, served as a focal-point and a prominent diagnostic feature, for detection of effects, i.e. gain/loss of other components involved during progression of CRC disease. Coincidentally, the chromosomes on which these genes reside have been found to be dense and rich in SNPs (hotspots), the details of which were published in a separate report. This work harnessed the potential of genetics, developmental biology and bio-informatics tools to solve a long-standing puzzle in pinpointing some genetic factors that were critically involved in the progression of CRC disease. The report has created enough impact, in terms of authentically suggesting, that it is only when we deploy a combinatorial approach towards certain complicated biological problems, we can successfully unveil the underlying mechanisms in greater details.