Das Tumorsubpressorprotein P53 („Wächter des Genoms“) und NF-kappa B

Das Tumorsubpressorprotein P53 sorgt dafür, dass sich eine Zelle nur dann teilt, wenn ihr Erbgut auch intakt ist. Dies ist bei einer Tumorzelle nicht der Fall. Dann zeigt p53 seine zwei Hauptwirkungen: bei reparablen Schäden Zellzyklus-Arrest (Anhalten der Zellteilung), bei irreparablen Schäden Einleitung der Apoptose (Zelltod)“.

„Das Tumorsubpressor-Gen p53 ist in mehr als 60% aller bösartigen Tumoren **mutiert oder deletiert**. **Gegen mutiertes p53-Protein** kann **P53 Autoantikörper** zur Folge haben. Alle Patienten mit p53-Autoantikörpern, haben durch andere Verfahren gesicherte bösartige Tumoren. Ein negatives Testergebnis schließt einen bösartigen Tumor nicht aus“. Quelle: http://de.wikipedia.org/wiki/P53

- p53 ist ein Tumorsuppressor -System, das aktiviert wird, wenn eine Überschussaktivität an DNA-Replikation in Zellen vorkommt.
- davon lässt sich ableiten, dass je höher die replikative DNA-Aktivität in Zellen ist, desto höher ist die Aktivität von p53, wenn Zellen zunehmend " beschleunigt" werden, dann brauchen sie eine stärkere " Ausbremsung". Die überschießende Aktivität von nicht- mutierten, funktionsfähigen p53 ist ein Marker für eine schlechte Prognose, das heißt je bösartiger der Tumor ist, umso mehr p53 ist aktiviert.
- wenn p53 mutiert und infolgedessen funktionsunfähig ist, und wenn dann die Signale einer verstärkten Aktivität der DNA-Replikation weiter bestehen, aber p53 funktionslos ist, dann wird innerhalb der Rückkopplungsschleife p53 verstärkt gebildet werden. Da die „Bremse“ nicht funktioniert, wird sich der Druck auf das Bremspedal verstärken, um die erhöhte replikative DNA-Aktivität unter Kontrolle zu bekommen. Quelle: : **Jose Gros** · 17.88 · Sociedad Española de Oncología Médica

The Tumorsubpressorprotein P53 ensures that a cell divides only when their genetic material is intact. This is not the case of a tumor cell. Then p53 shows its two main effects: for repairable damage cell cycle arrest (stopping cell division) in irreparable damage induction of apoptosis (cell death). "

"The Tumorsubpressor gene p53 is **mutated or deleted** in more than 60% of all malignant tumors. **Mutant p53 protein may also cause P53 autoantibodies**. All patients with p53 autoantibodies have - secured by other methods - malignant tumors. A negative test result does not exclude a malignant tumor as well ". Source: http://de.wikipedia.org/wiki/P53

-p53 is a tumor suppressant system that is activated when an excess DNA replicative activity appears in the cell.
-from this, it can be deducted that the higher the replicative activity in the cell is, the higher the activity of p53 will be, as Cells more 'speeded up' are the ones needing a stronger 'brake activation', thus, the activity of non-mutated and functioning p53 is in itself a marker of poor prognosis, as the more malignant the tumor is, the more p53 will be activated
-if p53 is mutated and non-functional, as the signals of enhanced DNA replication activity continue, but p53 has no function, the feedback loop will enhance p53 expression in a continued way, as the brake not working, the foot will increase pressure on the brake pedal to have replication under control
Source: **Jose Gros** · 17.88 · Sociedad Española de Oncología Médica

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„Last, it is relevant to note that independent of aging and age-related diseases, FOXO4-DRI may be of use against the progression, stemness, and migration of malignant cancer. Given that SASP factors influence these (Campisi, 2013), it will be particularly interesting to determine whether FOXO4-DRI affects those p53-wt cancer cells that have adopted a more migratory and stem-like state due to reprogramming by chronic SASP exposure. In any case, the here reported beneficial effects of FOXO4-DRI provide a wide range of possibilities for studying the potential of therapeutic removal of senescence against diseases for which few options are available.“

- ➔ **Krebsstammzelltherapie** http://www.xerlebnishaft.de/krebsstammzelltherapie.pdf
- ➔ **Zytoskelett** http://www.xerlebnishaft.de/zytoskelett.pdf
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Natürliche Inhibitoren von NF-κB sind[1] z.B.: Tocotrienole, Allicin, Genistein, Quercetin, Ginkgo, Curcumin, Epigallocatechingallat. Diese Stoffe sind die wirksamen Bestandteile von Knoblauch, Rotklee, Ginko biloba, Gelbwurz (Kurkuma), grünem Tee, Traubenkernöl und rotem Palmöl.
Natural inhibitors of NF-κB are, for example: tocotrienols, allicin, genistein, quercetin, ginkgo, curcumin, epigallocatechin gallate. These substances are the active ingredients of garlic, red clover, ginkgo biloba, yellow curd (turmeric), green tea, grape seed oil and red palm oil.

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