

Abstract

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Niacin and carcinogenesis.

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BACKGROUND: The dietary status of niacin (vitamin B3) has the potential to influence DNA repair, genomic stability, and the immune system, eventually having an impact on cancer risk, as well as the side effects of chemotherapy in the cancer patient.

FINDINGS: In addition to its well-known redox functions in energy metabolism, niacin, in the form of NAD, participates in a wide variety of ADP-ribosylation reactions. Poly(ADP-ribose) is a negatively charged polymer synthesized, predominantly on nuclear proteins, by at least seven different enzymes. Poly(ADP-ribose) polymerase-1 (PARP-1) is responsible for the majority of polymer synthesis and plays important roles in DNA damage responses, including repair, maintenance of genomic stability, and signaling events for stress responses such as apoptosis. NAD is also used in the synthesis of mono(ADP-ribose), often on G proteins, with poorly understood roles in signal transduction. Last, NAD and NADP are required for the synthesis of cyclic ADP-ribose and nicotinic acid adenine dinucleotide (NAADP), two mediators of intracellular calcium signaling pathways.

CONCLUSIONS: Disruption of any of these processes has the potential to impair genomic stability and deregulate cell division, leading to enhanced cancer risk. There are various sources of evidence that niacin status does have an impact on cancer risk, including animal models of leukemogenesis and skin cancer, as well as epidemiological data from human populations.

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