

# Amyloid Deposits: a Possible Link between Type 2 Diabetes Mellitus and Cancer

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## Editorial

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## Editorial

In a previous editorial we've demonstrated that increasing evidence points to the cytotoxicity of islet amyloid polypeptide aggregates showing them not just an insignificant phenomenon derived from the disease progression but as agents which directly induce processes that impair the functionality and the viability of beta-cells eventually leading to the loss of beta-cell mass in type 2 diabetes (T2D) patients [1].

Recently, Serum amyloid A protein was found to be expressed in tumor cells in 44.2% cases and in tumor associated macrophages in 62.5% cases. Serum amyloid A positivity in tumor associated macrophages in breast cancer was associated with larger tumor-size, higher histological-grade, negative estrogen-receptor and progesterone-receptor statuses, and HER-2 overexpression. It was also linked to worse recurrence-free survival in a multivariable regression model [2]. Serum amyloid-A was also shown to be increased with disease stage in squamous cell cervical cancer patients [3].

Further, Amyloid-beta precursor protein was causally linked to tumorigenicity as well as invasion of aggressive breast cancer [4]. Amyloid precursor-like protein 2 (APLP2) was also found to be overexpressed in cancer cells; together with amyloid precursor protein were linked to increased tumor cell proliferation, migration, and invasion [5]. Additionally, depletion of Amyloid Precursor Protein causes G0 arrest in non-small cell lung cancer cells [6]. Moreover, stable knock-down of APLP2 expression (with inducible shRNA) in pancreatic cancer cells reduced the ability of these cells to migrate and invade. Down-regulation of APLP2 decreased the weight

and metastasis of orthotopically transplanted pancreatic tumors in nude mice [7]. I repeat my strong wish and call to focus on the full of potentials amyloid research as well as amyloid antagonists.

## References

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