

Adenocarcinoma in Pancreatic Organogenesis

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ABSTRACT

Pathological diseases in the pancreatic organ are resulting in patients' deadly diseases. An omnipotent source of the highly fatal diagnosis of death lies in cancerogenesis within the pancreatic organ. Unravel the potential factors of the initiation of cancerogenesis in the pancreas will help to improve early detection and, thus, to enhance patients' therapy and survival rate.

ABBREVIATIONS

EMT: Epithelial-to-mesenchymal transition; PanIN2: Pancreatic intraepithelial neoplasia 2; PDAC: Pancreatic ductal adenocarcinoma

ADENOCARCINOMA FROM AN ECTOPIC PANCREAS

Lately, Kaneko et al. represented a case study in illustrating that within the ectopic pancreas, the adenocarcinoma appears in 14 reported cases of patients in the scientific literature, as well as in the clinical picture described in the article [1, 6]. The study highlights operative interventions in the field of surgery, highlighting the conclusion, that obstruction in the ectopic pancreas should be considered as an adenocarcinoma. A classification model is introduced for particular criteria related to the diagnosis of carcinoma from a heterotopic pancreas. A heterotopic pancreas also called the ectopic pancreas, defines pancreatic tissue outside the boundaries of the pancreas and without anatomic and vascular connections to the pancreas. Interestingly, in the case study presented in the paper, no apparent cancer was seen in other organs. As summarized, the establishment of cancerogenesis within the pancreas and in the ectopic pancreas might suggest an identical mechanism regarding the initiation and progression. Thus, a previously published model by Willmann et al. might shed light on mechanism of cancerogenesis, respective the endocrine lineage allocation.

ADENOCARCINOMA FROM PANCREATIC ORGANOGENESIS

Kijima et al. already suggest the mechanism of epithelial-to-mesenchymal transition (EMT) involved in human prostate cancer, lung squamous cell carcinoma, and human hepatocellular carcinoma cells [2-4]. Furthermore, a complete EMT mechanism, as well as an incomplete EMT mechanism, illustrates poor prognosis for the patient's survival rate. In line with previously suggested mechanism,

Willmann et al. provide further hypotheses on the origin of cancerogenesis within the mechanism of EMT [5]. In the recently published paper, a single cell delamination model illustrates epithelial, respective endocrine primed cells, which leave the ductal cord during mice embryogenesis of the budding pancreatic gland. The single-cell delamination model suggests a migration of the "cancer cell of origin" in the surrounding tissue of the former ductal inherited cells.

Thereby, in the so-called "cancer cell of origin" a shift in the polarity complex suggest to either break down or relocate slightly polarity complex components within the cell, later supports the hypothesis of fine-tuned maturation steps in the development of the Islets of Langerhans. Factors are introduced for supporting the model of the "cancer cell of origin" with the gene *Synaptotagmin 13*. In fact, the misregulation of the gene *Synaptotagmin 13* might introduce pancreatic lesions as pancreatic intraepithelial neoplasia 2 (PanIN2), which can progress to a pancreatic ductal adenocarcinoma (PDAC). Further experiments will unravel the role of the gene *Synaptotagmin 13* in the process of cancerogenesis, respective in the process of EMT. This may help the community for improved understanding of disease emergence and thus, for patients improved treatment.

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From the ductal structure framed by the black lines (**Figure 1**), immature Islets of Langerhans emerge into the surrounding tissue. Next to the ductal structure, hypothetical

PanIN are illustrated by black lines, which derive in later stages to PDAC.

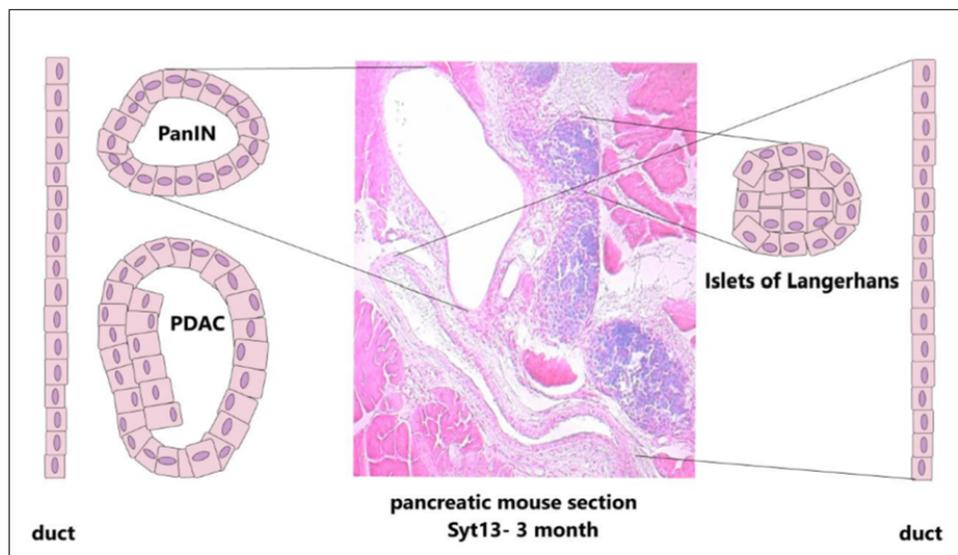


Figure 1. Hypothetical model of PanIN2, respective PDAC in the pancreatic region of a *Synaptotagmin 13* mouse model.

CONCLUSION

Cancerogenesis, in general, is a topic that is at the forefront of worldwide human health needs. Thus, further scientific approaches will accelerate the understanding of this disease, as well as mechanisms to treat exceptionally better the fatal illness such as adenocarcinoma in the pancreatic region. Incorporating previous work from Kijima et al. of different cancer-affected tissues in line with novel indication markers will support the scientific community and help to improve patients' health.

DECLARATION

The material used for this short communication is available to the public under the following link: <http://dnb.info/1110015216>.

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