

Aspirin and Pancreatic Cancer—Letter

Raffaella Mormile



In a recent publication by Risch and colleagues (1), regular use of aspirin was associated with reduced risk of pancreatic cancer (1). Few options besides the avoidance of smoking and obesity are available to prevent pancreatic cancer (1). It has been highlighted that people who take aspirin for prevention of other diseases, including cardiovascular disease, likely also reduce their risk of developing pancreatic cancer (1). Aspirin has been established more than a quarter century ago as an evidence-based therapy to reduce recurrent cardiovascular events in patients affected by coronary artery disease based on limited data by contemporary standards (2). However, aspirin for secondary prevention in patients suffering from ischemic heart disease has been recently and critically reappraised (2). The anticancer effects of aspirin have been indicated in a variety of organs, although the precise mechanisms remain unknown (3). It has been suggested that the anticancer action of aspirin may be connected with downregulation of the antiapoptotic protein survivin (3). Survivin is a member of the inhibitor of apoptosis protein family that is overexpressed selectively in a wide variety of human malignancies (3). Apoptosis represents a distinct form of cell death (3). Survivin

expression in pancreatic cancer has been widely studied (4). It has been shown that serum survivin concentrations are significantly elevated in patients with pancreatic cancer when compared with a control population of healthy blood donors (4). In addition, the serum survivin levels have been reported to be higher in patients with pancreatic cancer at advanced stages and those with recurrence and poor differentiation by reducing the cancer cell apoptosis (4). Although survivin is considered an unfavorable prognostic factor in cancer cells, it has been demonstrated to represent a mechanism by which myocytes at risk of apoptosis retain their viability (4, 5). Survivin myocardial expression after acute myocardial infarction (AMI) has been associated with the survival of at risk myocardium and favorable remodeling after AMI (5). All these contentions led us to hypothesize that the reduced risk of pancreatic cancer by aspirin as a result of survivin downregulation may represent an indirect evidence to critically reappraise aspirin for secondary prevention for cardiovascular disease, taking into account the protection effect of survivin on AMI. Large randomized controlled trials are needed to define aspirin endpoints in both cardiovascular diseases and pancreatic cancer, outlining the critical threshold dose above which aspirin may be not safe for the heart.

Division of Pediatrics and Neonatology, Moscati Hospital, Aversa, Italy.

Corresponding Author: Raffaella Mormile, Division of Pediatrics and Neonatology, Moscati Hospital, Gramsci Street, Aversa 81031, Italy. Phone: 3933-9204-5468; E-mail: raffaellamormile@alice.it

doi: 10.1158/1055-9965.EPI-17-0059

©2017 American Association for Cancer Research.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

Received January 17, 2017; accepted January 21, 2017; published OnlineFirst May 15, 2017.

References

1. Risch HA, Lu L, Streicher SA, Wang J, Zhang W, Ni Q, et al. Aspirin use and reduced risk of pancreatic cancer. *Cancer Epidemiol Biomarkers Prev* 2017;26:68–74.
2. Welsh RC, Roe MT, Steg PG, James S, Povsic TJ, Bode C, et al. A critical reappraisal of aspirin for secondary prevention in patients with ischemic heart disease. *Am Heart J* 2016;181:92–100.
3. Yang L, Zhu H, Liu D, Liang S, Xu H, Chen J, et al. Aspirin suppresses growth of human gastric carcinoma cell by inhibiting survivin expression. *J Biomed Res* 2011;25:246–53.
4. Dong H, Qian D, Wang Y, Meng L, Chen D, Ji X, et al. Survivin expression and serum levels in pancreatic cancer. *World J Surg Oncol* 2015;13:189.
5. Santini D, Abbate A, Scarpa S, Vasaturo F, Biondi-Zoccai GG, Bussani R, et al. Surviving acute myocardial infarction: survivin expression in viable cardiomyocytes after infarction. *J Clin Pathol* 2004;57:1321–4.