*Recipe for cancer education: a novel integrated cooking and nutrition education curriculum for medical students and physicians in dietary preventive and supplemental treatment for pancreatic cancer*

## Dominique J. Monlezun, Patricia Tsai, Leah Sarris & Timothy S. Harlan

Submit manuscripts:
www.editorialmanager.com/jmap

🙋 Springer

🙋 Springer

Springer

REVIEW

# Recipe for cancer education: a novel integrated cooking and nutrition education curriculum for medical students and physicians in dietary preventive and supplemental treatment for pancreatic cancer

**Dominique J. Monlezun · Patricia Tsai ·
Leah Sarris · Timothy S. Harlan**

**Abstract** The poor outcomes surrounding pancreatic cancer (5 % 5-year survival) are rooted in the advanced nature of the cancer at diagnosis, aggressiveness of the tumor, technical challenges for surgical treatment, and the limitations of systemic therapies. Research on pancreatic cancer and diet has focused on the effects of bioactive food compounds upon major pathways in cancerous cell development, particularly with the active components of flavonoids, proteins, and vitamins. Such bioactive components, particularly found in fruits and vegetables, have been associated with increased sensitivity to chemotherapeutic drugs for treating pancreatic cancer and inhibiting angiogenesis and cancerous cell growth. Diet-based approaches to prevention and treatment of pancreatic cancer may thus offer clinical efficacious and cost-effective opportunities to optimize existing modes of treatment (surgery, systemic therapies, radiotherapies, and palliative care), offset rising medical costs for patients, and improve quality of life for patients undergoing treatment. Integration of these findings at the clinical level may require a national, scalable model such as that of Tulane University School of Medicine's Goldring Center for Culinary Medicine (GCCM) to meaningfully improve the care of health care providers for pancreatic cancer patients.

D. J. Monlezun (✉) · P. Tsai · L. Sarris · T. S. Harlan
The Goldring Center for Culinary Medicine, Tulane University School of Medicine, 1430 Tulane Avenue #8541, New Orleans, LA 70112, USA
e-mail: djmonl37@gmail.com

D. J. Monlezun
Department of Global Health Systems and Development, Tulane University School of Public Health and Tropical Medicine, New Orleans, USA

## Introduction

Pancreatic cancer is the second most common digestive cancer in the US, following colorectal cancer, and the fourth leading cause of cancer-related death in both males and females [1]. The poor outcomes surrounding pancreatic cancer (5 % 5-year survival) are rooted in the advanced nature of the cancer at diagnosis, aggressiveness of the tumor, technical challenges for surgical treatment, and the limitations of systemic therapies.

This paper highlights major findings on nutrition and pancreatic cancer treatment and prevention.

Diet-based approaches to prevention and treatment of pancreatic cancer offer opportunities to enhance the effectiveness of existing modes of treatment (surgery, systemic therapies, radiotherapies, and palliative care), offset rising medical costs for patients, and improve quality of life for patients undergoing treatment.

Despite significant attention from researchers on nutrition and pancreatic cancer, there has not been a concomitant effort to integrate these findings at the clinical level. Tulane University School of Medicine's Goldring Center for Culinary Medicine (GCCM), as the world's first medical school-based teaching kitchen, provides a promising [2], scalable model for bridging the gap between nutrition research and clinical aspects of patient care.

Tulane University School of Medicine launched GCCM in November of 2012 in response to dire challenges in the global obesity epidemic and disparities in health care. GCCM was designed to establish the world's first evidence-based nutrition education curriculum for medical

students, medical professionals, and patients with the object of improving health equity through low-cost disease management, specifically low-cost nutrition counseling. At GCCM, a licensed physician and professional chef, in collaboration with chefs at the Johnson & Wales University Department of Culinary Nutrition, have developed a comprehensive curriculum for medical schools, residency programs, and continuing medical education (CME) modules for practicing physicians. The curriculum serves as a novel synthesis of validated nutrition education and hands-on cooking skills through a simulation-based medical education with deliberate practice (SBME-DP) model. The classes seek to empower medical students, residents, and practicing physicians to improve patients' health through effective nutrition counseling. The designed curriculum fits well within the emerging complex care management (CCM) paradigm as a dominant approach within implementation of the Affordable Care Act. [3].

GCCM's accompanying research arm, the Cooking for Health Optimization with Patients (CHOP) model, is a longitudinal comparative effectiveness study to investigate two long-term and short-term hypotheses: (1) (direct, short-term) GCCM curriculum enhances the attitudes, dietary habits, and competencies (ADCs) of medical students and professionals in providing effective patient nutrition education, and (2) (indirect, long-term) this system capacity, development-based intervention will result in improved patient outcomes in chronic disease management or lower disease incidence. Both hypotheses are designed to operate in this nationally scalable and sustainable GCCM education model so that the intervention can be efficiently disseminated. The CHOP model has three large longitudinal prospective observational cohort studies—medical students (CHOP-Medical Students), residents (CHOP-Residents), and practicing physicians (CHOP-CME)—in addition to a fourth cohort study with patients' families (CHOP-Patients: Community). Randomized controlled trials are additionally conducted through CHOP-Patients: RCTs for diabetic patients and eventually for patients with hypertension, heart disease, and cancer. Qualitative studies using a community participatory research design are being launched in conjuncture with the above quantitative study to ensure that community and patient input is utilized to guide GCCM class programming along with CHOP study design, analysis, and result reporting. This community partnership is meant to support optimal patient acceptance of GCCM and its accompanying research arm to ensure this site serves as an effectively scalable model for other medical centers nationally and, eventually, globally. Although the CHOP-CME module on cancer prevention and treatment is still in its infancy, it holds great potential for strengthening patient nutrition counseling by targeting nutrition education for practicing physicians.

## Methods

Diet-based and nutrition studies on pancreatic cancer prevention and treatment was searched using PubMed. Search terms included but were not limited to: "pancreatic cancer", "gastrointestinal cancer", "nutrition", "diet", "dietary", "prevention", "treatment", "therapy", "protein", "risk", and "metabolic disorder". English language research published between January 1999 and December 2013 were considered for this review.

## Results

Review of relevant literature revealed that existing research on pancreatic cancer and diet has focused on the effects of bioactive food compounds upon major pathways in cancerous cell development. Results were categorized according to bioactive food component with a corresponding summary of its correlation to pancreatic cancer growth and proliferation.

Flavonoids

Flavonoids, bioactive food components from fruits and vegetables have been associated with reducing the risk of developing pancreatic cancer, inhibiting cancerous cell growth (by arresting cell cycle, inhibiting angiogenesis, or inducing apoptosis), and improving the effectiveness of chemotherapeutic drugs [4]. Flavonoids like apigenin and luteolin can be found in parsley, spices (oregano, rosemary, and basil), artichokes, spinach, and chamomile. Apigenin may inhibit cancer cell growth and proliferation via down-regulation of geminin protein expression [5] or by inhibiting Il-6 induced EC migration and proliferation [6]. Apigenin may also down-regulate glucose uptake by inhibition of the GLUT-1 transporter [7] and arrest cell growth at the G2/M phase of the cell cycle [8]. Similarly, baicalein, a flavone extracted from the roots of the skullcap flower, has been known to induce apoptosis in cancer cells [9, 10]. Another flavonoid, quercetin, which can be found in kale, sweet potatoes, and dill, has been associated with antioxidant properties, inducing apoptosis in cancer cells, and arresting cell growth by down-regulation of Hsp70 protein [11]. Furthermore, studies on the isoflavone genistein, which can be found in soybeans, has been linked to increased effectiveness of chemotherapeutic drugs used in pancreatic cancer treatment: gemcitabine, erlotinib, and cisplatin [12–15].

Protein

A plant-based, low-protein diet has been associated with inhibiting tumor growth in other cancer types, specifically

prostate and breast cancers, by down-regulation of mTOR activity and decreased PSA and IGF-1 levels [16]. By contrast, a high-fat, high-protein diet was associated with high risk of pancreatic carcinogenesis, possibly due to the natural regression of early lesions and interference with repair mechanisms [17]. Risk of pancreatic cancer was inversely related to estimated initial levels of methionine, an essential amino acid found in meats like beef or fish (tuna and salmon) as well as soybeans and Brazilian nuts.

## Vitamins

Vitamins have also been linked to inhibiting pancreatic cell growth. Increased intake of vitamin D, which can be found in fatty fish, eggs, liver, mushrooms, and ultraviolet rays from sunlight, has been inversely related to risk of pancreatic cancer [18, 19]. Vitamin E, found in nuts like almonds or sunflower seeds and leafy greens like spinach, has also been linked to stimulated cancer cell death and increased responsiveness to the chemotherapeutic drug, gemcitabine, by facilitating the inactivation of kinase Akt via phosphatase PHLPP1 [20, 21]. Deficiency in folate, which is found in cereal grains, vegetables, and liver, has been associated with an increase in the risk of pancreatic cancer due to hypomethylation of DNA [22, 23].

## Dietary risk factors

In contrast to research on the role that bioactive food components may play in prevention of pancreatic cancer, existing studies have also highlighted the correlation between risk of pancreatic carcinogenesis and certain modifiable habits, including excessive alcohol consumption, caffeine intake, artificial sweeteners, and tobacco usage. Review of literature on alcohol consumption has indicated that heavy alcohol usage, defined as three or more drinks per day, may have a 1.22- to 1.36-fold increase in the risk of developing pancreatic cancer [24, 25]. Preliminary findings indicate that ethanol itself and metabolites of alcohol, such as acetaldehyde and fatty acid ethyl esters, may be involved in pathways to pancreatitis and carcinogenesis [25]. Results of pooled data analyses have demonstrated little to no effect of caffeine or artificial sweeteners on the risk of developing cancer [26, 27, 29]. However, a threefold increase in risk of carcinogenesis has been associated with tobacco usage and smoking at least one pack of cigarettes per day [26–28].

## Discussion

A wealth of research has indicated that numerous bioactive components (including flavonoids, protein, and vitamins from fruits and vegetables) have been associated with increased sensitivity to chemotherapeutic drugs and inhibition of angiogenesis and cancerous cell growth. Excessive alcohol consumption or heavy tobacco usage, in contrast, has been shown to increase the risk of pancreatic carcinogenesis.

The aggressive nature of pancreatic cancer and its subsequent toll on quality of life for pancreatic cancer patients further highlight the need for more intentional and targeted nutritional therapies for patients. The Cooking for Health Optimization (CHOP) model as a multi-center trial with over 10 medical schools and universities pioneered by Tulane University School of Medicine's Goldring Center for Culinary Medicine (GCCM) addresses the need for more robust nutrition education for health care providers and patients, an essential step in implementing key findings of research at the intersection between diet and disease. Through CHOP, GCCM seeks to demonstrate cost-efficient improved patient outcomes by strengthening nutrition education for medical students through electives and physicians through continuing medical education (CME) and also by providing hands-on cooking and nutrition education for lower income patients in its teaching kitchen, which is housed in a former food desert and now adjacent to a national grocery store.

Given that pancreatic cancer costs patients approximately $65,000 in surgery and over $3,000 in systemic therapies [30], while only offering a 5 % chance or less of surviving beyond 5 years, GCCM's CHOP model of engaging nutrition education offers a potentially cost-effective strategy for optimizing current modes of treatment for pancreatic cancer, saving patients, insurers, and health care providers valuable time and resources while improving patient quality of life.

**Conflict of interest**    None.

## References

1. Jemal A, Siegel R, Xu J, Ward E (2010) Cancer statistics. CA Cancer J Clin 60:277–300

2. Birkhead A, Foote S, Monlezun DJ, Loyd J, Joo E, Leong B, Sarris L, Harlan T (2013) Medical student-led community cooking classes: a novel preventative medicine model easy to swallow. Am J Prev Med 46:e41–e42

3. Hong C, Abrams M, Ferris T (2014) Toward increased adoption of complex care management. N Engl J Med 371:491–493

4. Gil-Izquierdo A, Gil M, Ferreres F, Tomas-Barberan F (2001) In vitro availability of flavonoids and other phenolics in orange juice. J Agric Food Chem 49:1035–1041

5. Melstrom L, Salabat M, Ding X, Milam B et al (2008) Apigenin inhibits the GLUT-1 glucose transporter and the phosphoinositide 3-kinase/Akt pathway in human pancreatic cells. Pancreas 37:426–431

6. Johnson J, Gonzalez de Mejia E (2011) Dietary factors and pancreatic cancer: the role of food bioactive compounds. Mol Nutr Food Res 55:58–73

7. Salabat M, Melstrom L, Strouch M, Ding X et al (2008) Geminin is overexpressed in human pancreatic cancer and downregulated by the bioflavonoid apigenin in pancreatic cancer cell lines. Mol Carcinog 47:835–844

8. Ujiki M, Ding X, Salabat M, Bentrem D et al (2006) Apigenin inhibits pancreatic cancer cell proliferation through G2/M cell cycle arrest. Mol Cancer 5:76

9. Pu P, Wang X, Salim M, Zhu L, Wang L, Chen K, Xiao J, Deng W, Shi H, Jiang H, Li H (2012) Baicalein, a natural product, selectively activating AMPKa$_2$ and ameliorates metabolic disorder in diet-induced mice. Mol Cell Endocrinol 362:1128–1138

10. Takashi H, Chen M, Pham H, Angst E, King J, Park J, Brovman E, Ishiguor H, Harris D, Reber H, Hines O, Gukovskaya A, Go V, Eibl G (2011) Baicalein, a component of *Scutellaria baicalensis*, induces apoptosis by Mcl-1 down-regulation in human pancreatic cancer cells. Biochim Biophys Acta 1813:1465–1474

11. Gibellini L, Pinti M, Nasi M, Montagna J, De Biasi S, Roat E, Bertoncelli L, Cooper EL, Cossarizza A (2010) Quercetin and cancer chemoprevention. Evid Based Complementary Altern Med 2011:1–15

12. Banerjee S, Zhang Y, Ali S, Bhuiyan M et al (2005) Molecular evidence for increased antitumor activity of gemcitabine by genistein in vitro and in vivo using an orthotopic model of pancreatic cancer. Cancer Res 65:9064–9072

13. Mohammad R, Banerjee S, Li Y, Aboukameel A et al (2006) Cisplatin-induced antitumor activity is potentiated by the soy isoflavone genistein in BxPC-3 pancreatic tumor xenografts. Cancer 106:1260–1268

14. Johnson J, Gonzalez de Mejia E (2011) Dietary factors and pancreatic cancer: the role of food bioactive compounds. Mol Nutr Food Res 55:58–73

15. Banerjee S, Zhang Y, Ali S, Bhuiyan M et al (2005) Molecular evidence for increased antitumor activity of gemcitabine by genistein in vitro and in vivo using an orthotopic model of pancreatic cancer. Cancer Res 65:9064–9072

16. Fontana L, Adelaiye R, Rastelli A, Miles K, Ciamporcero E, Longo V, Nguyen H, Vessella R, Pili R (2013) Dietary protein restriction inhibits tumor growth in human xenograft models of prostate and breast cancer. Oncotarget 4:2451–2461

17. Z'graggen K, Warshaw A, Werner J, Graeme-Cook F, Jimenez R, Fernandez-del Castillo C (2001) Promoting effect of a high-fat/ high-protein diet in DMBA-induced ductal pancreatic cancer in rats. Ann Surg 233(5):688–695

18. Bao Y, Ng K, Wolpin B, Michaud D et al (2010) Predicted vitamin D status and pancreatic cancer risk in two prospective cohort studies. Br J Cancer 102:1422–1427

19. Skinner H, Michaud D, Giovannucci E, Willett W et al (2006) Vitamin D intake and the risk for pancreatic cancer in two cohort studies. Cancer Epidemiol Biomark Prev 15:1688–1695

20. Huang P, Chuang H, Chou C, Wang H, Lee S, Yang H, Chiu H, Kapuriya N, Wang D, Kulp S, Chen C (2013) Vitamin E facilitations the inactivation of kinase Akt by the phosphatase PHLPP1. Sci Signal 6(267):1–13

21. Nitsche C, Edderkaoui M, Moore R, Eibl G, Kasahara N, Treger J, Grippo P, Mayerle J, Lerch M, Gukovskaya A (2012) Phosphatase PHLPP1 regulations Akt2, promotes pancreatic cancer cell death, and inhibits tumor formation. Gastroenterology 142:377–387

22. Shane B, Alexopolous Y, Hebberd K, Vales S (2006) Folic acid, vitamin B12, and vitamin B6—biochemical, physiological, and molecular aspects of human nutrition. Saunders Elsevier, St. Louis, pp 694–712

23. Mason J, Choi S (2000) Folate and carcinogenesis: developing a unifying hypothesis. Adv Enzyme Regul 40:127–141

24. Becker A, Hernandez Y, Frucht H, Lucas A (2014) Pancreatic ductal adenocarcinoma: risk factors, screening, and early detection. World J Gastroenterol 20(32):11182–11198

25. Genkinger JM, Spiegelman D, Anderson KE, Bergkvist L, Bernstein L, van den Brandt PA, English DR, Freudenheim JL, Fuchs CS, Giles GG (2009) Alcohol intake and pancreatic cancer risk: a pooled analysis of fourteen cohort studies. Cancer Epidemiol Biomarkers Prev 18:765–776

26. Tramacere I, Scotti L, Jenab M, Bagnardi V, Bellocco R, Rota M, Corrao G, Bravi F, Boffetta P, La Vecchia C (2010) Alcohol drinking and pancreatic cancer risk: a meta-analysis of the dose-risk relation. Int J Cancer 126:1474–1486

27. Duell EJ (2012) Epidemiology and potential mechanisms of tobacco smoking and heavy alcohol consumption in pancreatic cancer. Mol Carcinog 51:40–52

28. Noreel SE, Ahlbom A, Erwald R, Jacobson G, Lindberg-Navier I, Olin R, Tornberg B, Wiechel KL (1986) Diet and pancreatic cancer: a case–control study. Am J Epidemiol 124(6):894–902

29. Michaud DS, Giovannucci E, Willett WC, Colditz GA, Fuchs CS (2001) Coffee and alcohol consumption and the risk of pancreatic cancer in two prospective United States cohorts. Cancer Epidemiol Biomark Prev 10(5):429–437

30. Bardou M, Le Ray I (2013) Treatment of pancreatic cancer: a narrative review of cost-effectiveness studies. Best Pract Res Clin Gastroenterol 27:881–892