Pancreatic cancer is a major cause of cancer related mortalities accounting for approximately 6% of all cancer-related deaths in men and women. Of the estimated 43,920 (22,090 male and 21,830 female) new cases and 37,390 (18,850 male and 18,540 female) deaths related to pancreatic cancer will occur in the United States in 2012 [1]. Chemotherapy and surgical resection are considered effective treatment for this overwhelming disease. However, chemotherapy resistance and late stage diagnosis in patients decreased the eligibility for chemotherapy and surgery [2]. Addressing these problems, there is an urgent need for adjuvant molecular therapy in pancreatic cancer by utilizing some chemical molecules or inhibitors. The challenge of meeting the expectations of this desired target product profile, a key question arises: how to combine the available treatment regimens and will these treatments are effective against pancreatic cancer? A variety of new investigational efforts have been used mono-targeted therapy, chemotherapy, and surgical resection for the treatment and prevention of pancreatic cancer [2,3].

Chemotherapy is the only option in late stage metastatic pancreatic cancer, where surgical resection is not possible. A growing understanding of the chemotherapeutic avails has opened research doors to reach the effect with combinational front-line chemotherapy for the treatment of metastasis. However, the emergence of chemoresistant pancreatic cancer and reduced drug delivery make these chemotherapeutic agents less effective in the prevention of disease. Several peculiar cellular pathways are also responsible for causing resistance in the induction of apoptosis through chemotherapeutics such as gemcitabine and 5-fluorouracil [2,3].

To achieve global control of pancreatic cancer, there must be chemical scaffolds, which disrupt chemo resistance-associated pathways. Additionally, the detection of treatment-resistant pancreatic cancer at earlier stages by measuring circulating stem-cell markers CD24, CD44, and esterase A would be very useful. The recent success with the whole-cell-screening approach is particularly exemplified by the identification of new diagnostic and prognostic markers and new gene therapy strategies [4]. Therefore, the identification of new targets such as tumor-associated antigens, enzymes and hormones, which harbor targeted molecular treatment but also proposed for use in diagnosing pancreatic cancer, would further improve the patient outcome. This momentum is further fuelled by mRNAs, which closely related to pancreatic cancer, resulting in longer disease-free survival compared with standard surgery in xenograft and orthotopic mouse models. Surgical resection also increased the survival rate from 5% to over 25% in pancreatic cancer, but progression to late stage metastasis greatly minimized the impact of surgery, and thus need to be replaced with adjuvant therapies [5,6,7].

The concomitant strategies include chemotherapy, surgery; mono-targeted molecular therapy show better promises with improved patient outcome than single therapy. These combined future treatment efforts will allow clinicians to adjust treatment, based on the specific markers and progression of the individual patient’s disease as well as on economic perspectives. The synergistic effect of adjuvant management achieves effective control with greater patient’s survival rate and also prevented disease recurrence.

The journal of pancreatic disorders and therapy articulate the importance of managing different treatment methods and engage the different strategies in health care. The precedence is not to dump the basic characteristics of chemotheraphy, surgery and mono-targeted molecular therapy, but rather to implement them with greater dynamism.

References