using multiplex flow-cytometry. Total RNA was then isolated from the CD8T cells and were subjected to lncRNA and mRNA gene expression analysis.

Results The isolated exosomes were 30–180nm in diameter and appeared as cup-shaped vesicles. The exosomes were positive for TSG101, CTLA4 and FAS expression. Interestingly, results from the apoptosis assay indicated that leanCRC exosomes were able to induce a higher percentage of CD8T cell death, in comparison to obeseCRC exosomes ($p < 0.01$). Similarly, this observation was also seen in the nonCRC exosomes. Furthermore, in the cytokine analysis, leanCRC exosomes induced a higher release of Perforin and IFNG than obeseCRC exosomes ($p < 0.05$). Notably, this observation was inverted in the nonCRC exosomes. Interestingly, obeseCRC exosomes induced higher levels of sFas and sFasL than leanCRC. Moreover, the microarray analysis showed that most of the dysregulated lncRNAs and genes were involved in the oxidative stress pathways.

Conclusions Contrary to our hypothesis, leanCRC exosomes were more immunosuppressive than obeseCRC exosomes, particularly in relation to CD8T cells. However, effects on other immune markers should be investigated as well.

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CLINICAL PROFILE AND OUTCOME OF ACUTE PANCREATITIS IN CHILDREN ADMITTED IN PHILIPPINE CHILDREN'S MEDICAL CENTER

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Background Acute pancreatitis, once considered rare in children, has been reported to show an increasing incidence worldwide. Determining Philippine data on clinical profile, factors, severity and outcome would help improve the recognition, diagnosis and management of this emerging disease.

To determine the clinical profile of children with Acute Pancreatitis and the factors associated with their outcome and severity. **Methods** A retrospective cohort study was done on children 0–18 years old with a diagnosis of Acute Pancreatitis. Demographic, clinical and diagnostic data gathered were compared among severity classification and outcome.

Results Thirty-five cases were identified in a period of 18 years, but only 28 cases were reviewed. Mean age was 11.5 years old $\pm$ 4.1 SD (range 4 - 18) with slight male predominance. Ninety-three percent presented with abdominal pain. Most common etiology was idiopathic (44%), followed by biliary disease and infection (21% each). Gallstones and choledochal cyst post excision were the most common of the comorbidities seen. Most common imaging findings was that of an edematous or enlarged pancreas. Five cases of acute recurrent pancreatitis noted. Most common local complications seen were pseudocyst formation and fluid collection with 11% each. One death was due to hemorrhagic pancreatitis seen intraoperatively. Of the factors reviewed, gallstones and the 3–10 years old age group were found have a statistically significant difference in terms of having a mild vs non-mild severity classification and type of clinical outcome on discharge, respectively.

Conclusions In this study, gallstones and age group of 3–10 years are found to affect severity and outcome and should be considered closely during management of pediatric acute pancreatitis.

A multi-center study on the incidence and clinical profile and outcome is recommended to obtain a better picture of the acute pancreatitis in Filipino children to help clinicians in recognizing and decreasing the morbidity and mortality of this disease.

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FGF14 IS A FUNCTIONAL TUMOR SUPPRESSOR THROUGH INHIBITING PI3K/AKT/MTOR PATHWAY IN COLORECTAL CANCER

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Background Colorectal cancer (CRC) is one of the most common malignancies worldwide. Emerging evidence indicates that promoter methylation of genes associated with gene silencing, plays an important role in the development and progression of CRC. In this study, we aimed to investigate the epigenetic regulation, biological function and molecular mechanism of FGF14 in CRC.

Methods The expression of FGF14 in CRC cell lines, CRC tissues and paired adjacent normal tissues was detected by PCR and Western blot. The biological function of FGF14 in CRC was interrogated by cell viability assay, colony formation, flow cytometry, cell invasion and migration assay, as well as *in vivo* study.

Results We found FGF14 was downregulated or silenced in all (10/10) CRC cell lines, while it was expressed in normal colonic tissues. The expression of FGF14 was lower in primary CRCs as compared to their adjacent normal tissues. Significant higher methylation of FGF14 was observed in CRCs than that in normal tissues based on the data from TCGA database. The loss of FGF14 gene expression was restored by treatment with DNA methyltransferase inhibitor 5-Aza. Re-expression of FGF14 in CRC cell lines inhibited cell viability and colony formation, and induced cell apoptosis. In xenograft mouse model, overexpression of FGF14 significantly reduced tumor growth ($P < 0.001$). FGF14 induced mitochondrial apoptosis and inhibited PI3K/AKT/mTOR pathway.

Conclusions In conclusion, FGF14 is a novel tumor suppressor, which suppresses cell proliferation and induces cell apoptosis via mediating PI3K/AKT/mTOR pathway.

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PAN-CANCER STUDY OF PROGRESSIVE PROTEIN SIGNATURES FOR DIGESTIVE CANCERS

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Background As functional executors, protein signatures will be more sensitive than gene signatures in characterizing biological systems. The direct study of the functional proteome is capable to complement and extend omics analysis. Especially, combining cancer research with proteome analysis can identify protein biomarkers to distinguish disease types or subtypes.

Methods The progressive signatures during dynamical biological processes are attracting more and more attention. As an

Abstract IDDF2019-ABS-0120 Figure 1 The analysis results of TCGA pan-cancer proteomics data on multiple digestive cancer sites

intuitive and interpretable signature, the trend test would disclose the continuous change during the dynamical process. Thus, the linear trend of protein expression during disease development and progression (e.g. across consecutive disease stages) would indicate the important behavior of disease-relevant proteins corresponding to worsening diseases, particularly in the pan-cancer context.

Results We re-analyzed the TCGA pan-cancer proteomics data on multiple digestive cancer sites, i.e. COAD, COADREAD, LIHC, READ, STAD and STES. The proteins with significant expression trend along cancer stages can be obtained as (figure 1A.) shown. The COAD, COADREAD tend to be similar on protein signatures, and READ, STAD and STES would be grouped together, and LIHC is an outlier due to its more observed progressive protein signatures. Then, by statistics of observation frequency of protein signatures in each cancer (figure 1B), and those of cancer sites for each protein (figure 1C), we found: (i) LIHC indeed have many protein signatures than others; (ii) some protein signatures of LIHC are same in COAD, COADREAD, meanwhile many protein signatures are exclusive for LIHC, i.e. specific protein signatures for liver cancer; (iii) protein signatures would have great differences in their expression trends in different digestive cancer sites, e.g. 4E-BP1 seems to be suppressor-protein except in LIHC (figure 1D), while YB-1 tends to be onco-protein.

Conclusions Totally, this work revisited the progressive markers of digestive cancers on the proteome and pan-cancer manner, and revealed new insights on understanding dynamic heterogeneity of digestive disease progression by digestive disease signatures.

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UPREGULATION OF THE LONG NON-CODING RNA LINC00460 PROMOTES GASTRIC CANCER PROGRESSION BY EPIGENETICALLY SILENCING P21 VIA EZH2 AND INDICATES POOR OUTCOME

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Background Multiple studies have revealed that long non-coding RNAs (lncRNAs) extensively participate in human cancer progression. Recently, we found that LINC00460 was an oncogene in promoting colorectal cancer cell proliferation. However, its role in gastric cancer (GC) remains unclear.

Methods LINC00460 expression was detected in 80 GC and matched non-cancerous tissues by quantitative reverse