high-quality bowel preparation is often difficult. The main aim was to evaluate the impact of the intensive patient educational programme on the quality of bowel preparation. The secondary endpoint was to assess the patient's compliance, acceptability, and tolerability towards the preparation regimen apart from identifying factors associated with good quality bowel preparation.

Methods We performed an assessor-blinded, randomised, controlled trial in an outpatient surgical clinic of a tertiary referral center. Patients were randomly assigned to a control group with standard written and verbal instructions or to an intervention group, where patients received an intensive, structured and comprehensive education programme. The randomisation allocation was concealed in an opaque envelope. All subjects were required to complete a questionnaire as to assess their compliance, acceptability and tolerability towards bowel preparation. Pearson chi-square and multivariable logistics regression were used in statistical analysis.

secondary endpoint

Results Three hundred and twelve patients were randomised, but only 300 patients were included in the analysis. Patients' characteristics of both groups were similar. The proportion of good bowel preparation quality in the interventional group was significantly higher than the control group (98.7% versus 52.3%, $p < 0.001$). The median total Boston Bowel Preparation Scale (BBPS) score for the interventional group was significantly greater than the control group (8 versus 5, $p < 0.001$). As compared to the control group, subjects in the interventional group demonstrated higher compliance (96.6% vs 85.4%, $p < 0.001$), acceptability (89.3% vs 33.1%, $p < 0.001$) and tolerability (85.9% vs 19.2%, $p < 0.001$) to bowel preparation regimen. Variables associated to good quality of bowel preparation identified from this study include intensive, structured and comprehensive education programme (OR: 22.5, $p < 0.001$, 95% CI: 4.05, 124.67), compliance to bowel cleansing agent (OR: 30.3, $p < 0.05$, 95% CI: 4.22, 217.01), acceptability of preparation (OR: 2.5, $p < 0.05$, 95% CI 1.04, 6.05), tolerability towards bowel preparation (OR: 5.3, $p < 0.05$, 95% CI: 1.55, 18.39) and non-hypomotility drugs (OR: 3.2, $p < 0.05$, 95% CI 1.25, 8.43).

Conclusions Patient educational programme is effective in optimizing the quality of bowel preparation for colonoscopy.

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PAN-TISSUE GENE DYS-REGULATION STUDY OF TYPE II DIABETES BY INTEGRATIVE PATHWAY ANALYSIS

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Background Different complications and stages often co-occur in the development of Type II Diabetes (T2D). The potential molecular mechanism between complications and related tissues is still not clear, thus it is crucial to characterize the common and specialty of dynamical biological processes in different responsive tissues of Type II Diabetes. Pan-tissue analyses that examine dys-regulation genes and pathways among various tissue types have emerged as a powerful way to obtain novel insights into Type II Diabetes. Considering the definite function modules (e.g. pathways) will be easier to interpret than chaotic genes, we propose a novel computational framework, Tissue Pathway Cross-talk (TPC), to recognize the functional relationship between tissue-specific and tissue-common pathways from omics data.

Methods Here we present TPC investigation on pathway alterations in a pan-tissue cohort including 6 tissues (adipose, liver, Mitochondria, muscle, pancreatic beta-cells, pancreatic islets) from 17 GEO datasets, comprising pathways from KEGG, reactome, Biocarta, and wikiPathway. Using our standardized workflow, we identified betweenness-induced hub-genes from the high-order network connecting common and specific pathways in different tissues.

Results After the comprehensive comparison with the previously reported milestone molecules of T2D, we found: (1) the critical function changes were identified by the tissue-specific pathways, which would be considered as a consensus phenotype-change event during T2D development and progression (table 1); (2) 28 tissue-common pathways related to T2D were significantly enriched with prior-known T2D associated genes, which would be pathway signatures of T2D regulation patterns (table 2); (3) Betweenness-induced hub-genes on pathway-network are selected as the key candidates to study the tissue diversity mechanism under the same disease/T2D condition.

Abstract IDDF2019-ABS-0209 Table 1 Analysis results: The Jaccard Index of pathway intersection between any two tissues.

Muscle	856					
Liver	348 (0.342)	510				
Mitochondria	177 (0.186)	120 (0.181)	273			
Pancreatic beta-cells	122 (0.132)	99 (0.165)	44 (0.106)	188		
Adipose	431 (0.42)	282 (0.34)	149 (0.20)	90 (0.13)	607	
Pancreatic islets	377 (0.365)	275 (0.349)	103 (0.142)	82 (0.124)	297 (0.344)	554
	Muscle	Liver	Mitochondria	Pancreatic beta-cells	Adipose	Pancreatic islets

Abstract IDDF2019-ABS-0209 Table 2 Analysis results: 28 tissue-common pathways enriched in six tissues.

NAME	DATABASE
Activation of NF-kappaB in B cells;	reactome
Oxygen-dependent proline hydroxylation of Hypoxia-inducible Factor Alpha;	reactome
ER-Phagosome pathway;	reactome
Cross-presentation of soluble exogenous antigens (endosomes);	reactome
Autodegradation of Cdh1 by Cdh1:APC/C;	reactome
APC/C:Cdc20 mediated degradation of Securin;	reactome
APC/C:Cdh1 mediated degradation of Cdc20 and other APC/C:Cdh1 targeted proteins in late mitosis/early G1;	reactome
Cdc20:Phospho-APC/C mediated degradation of Cyclin A;	reactome
Vpu mediated degradation of CD4;	reactome
SCF(Skp2)-mediated degradation of p27/p21;	reactome
Formation of the beta-catenin:TCFtransactivating complex;	reactome
Downstream TCR signaling;	reactome
DNA Damage/Telomere Stress Induced Senescence;	reactome
FCERI mediated NF-kB activation;	reactome
Regulation of ornithine decarboxylase (ODC);	reactome
AUF1 (hnRNP D0) binds and destabilizes mRNA;	reactome
Asymmetric localization of PCP proteins;	reactome
Degradation of DVL;	reactome
Defective CFTR causes cystic fibrosis;	reactome
Retrograde transport at the Trans-Golgi-Network;	reactome
CDK-mediated phosphorylation and removal of Cdc6;	reactome
Ubiquitin-dependent degradation of Cyclin D1;	reactome
G2/M Checkpoints;	reactome
The role of GTSE1 in G2/M progression after G2 checkpoint;	reactome
FBXL7 down-regulates AURKA during mitotic entry and in early mitosis;	reactome
Regulation of PTEN stability and activity;	reactome
TGF beta receptor I degradation signaling (TGF-beta_super_family_signaling_pathway(canonical))	inh
Integrin signaling pathway (Integrin signaling pathway)	inh

Conclusions Totally, we proposed a new viewpoint as pan-tissue analysis and implemented a pathway-based computational approach to detect the critical difference and common pathways in the responsive tissues during T2D progression, and also shown that the tissue-common pathways can especially serve as genetic warning signals for the T2D sub-states (e.g.complications).

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SECRETOME MODULATION OF CACO2 CELL LINE INDUCED BY A MULTI-STRAIN PROBIOTIC

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Background Probiotics are defined as live, non-pathogenic bacteria that confer health benefits beyond their nutritional value. Particularly VSL#3, a probiotic mix containing 4 strains of Lactobacilli (*L. paracasei*, *L. plantarum*, *L. acidophilus* and *L. delbrueckii* subsp. *bulgaricus*), 3 strains of Bifidobacteria (*B. longum*, *B. infantis*, *B. breve*) and *Streptococcus thermophilus*, has demonstrated efficacy in the management of diseases characterized by increased intestinal permeability. The aim of the present study was to study secreted bioactive factors in order

to evaluate the mechanisms of action to enhance intestinal epithelia.

Methods Two different lots of VSL#3 (Manufacturer: Nutrilinea Srl, Gallarate (VA) - Italy, lot #802097 and lot #802100) were used. Caco2 cell line were treated with a conditioning media (CM) prepared using 1g of probiotic formula grown in D-MEM cell culture medium (free of serum and antibiotics) at 37°C for 48 hours without shaking and in anaerobic conditions. Caco2 were treated with diluted CM at 1:10 and 1:25 for 24 and 48 hours. Media culture for each condition has been collected and analyzed by a deeper proteomics approach. Differential protein expression was evaluated by shotgun proteomics analysis based on nLC-HDMS^E and carried out on Synapt G2-Si mass spectrometer.

Results The analysis of supernatants from Caco2, treated with CM, showed the presence of bacteria strain-specific proteins. Human proteins synthesized from CaCo2 were also identified, such as caspase 1, IL8, HSP70, HSP70b, HSP90, HSP105. The productions were time- and dose-dependent. In CM diluted 1:10, probiotic-derived proteins have been shown to be more expressed at 24 hours. Human caspase 1, IL8, HSP 70, HSP 70b, HSP 90, HSP 105 were also found upregulated in Caco2 treated for 24 hours with CM diluted 1:10.

Conclusions This is the first time where a probiotic secretome was explored. Analysis of secretome from Caco2 treated with CM helped us to understand the mechanism by probiotics can enhance intestinal barrier: by strengthen the autophagy process, an arm of innate immunity, by overexpression of caspase 1, IL8 and HSP 70, and by HSPs dependent modulation of inflammation.