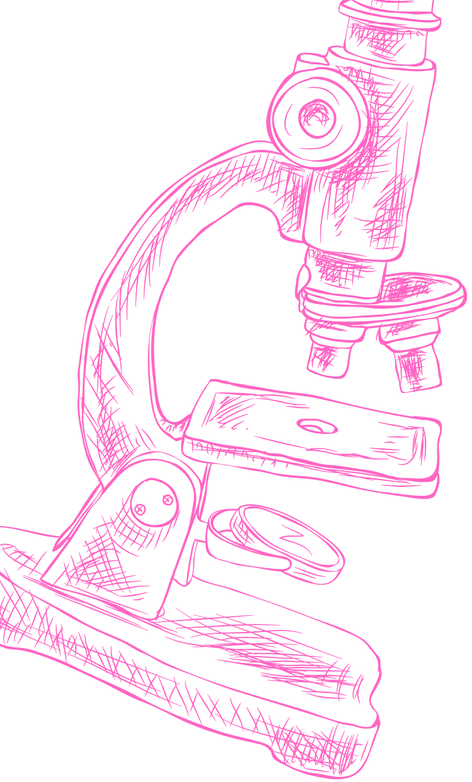
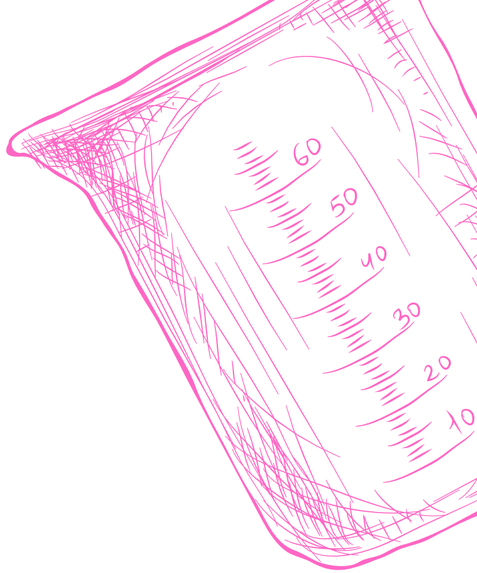
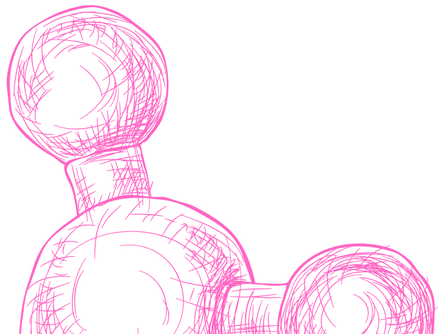
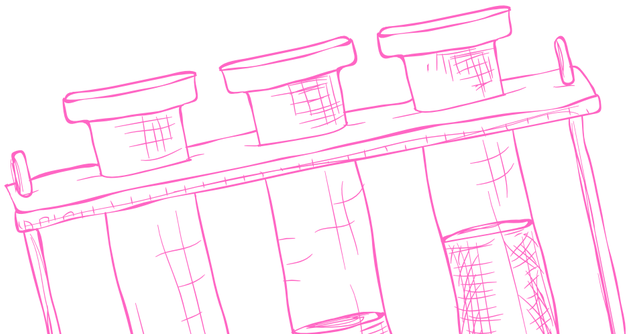


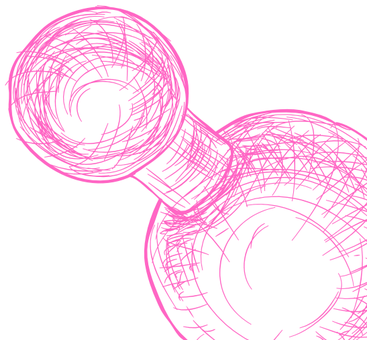


DIGESTION AND ABSORPTION OF CARBOHYDRATES LIPIDS SECOND STAGE

DR.RASHAD AL - TUUAMAH
Medical Biochemistry



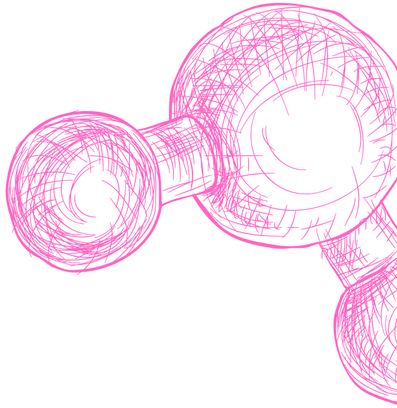




OVERVIEW OF MACRONUTRIENTS

CARBOHYDRATES ARE THE MOST ABUNDANT ORGANIC MOLECULES IN NATURE. THEY HAVE A WIDE RANGE OF FUNCTIONS, INCLUDING PROVIDING A SIGNIFICANT FRACTION OF THE DIETARY CALORIES FOR MOST ORGANISMS.

, ACTING AS A STORAGE FORM OF ENERGY IN THE BODY, AND SERVING AS CELL MEMBRANE COMPONENTS THAT MEDIATE SOME FORMS OF INTERCELLULAR COMMUNICATION.

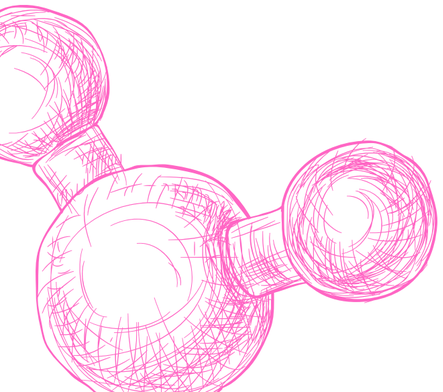


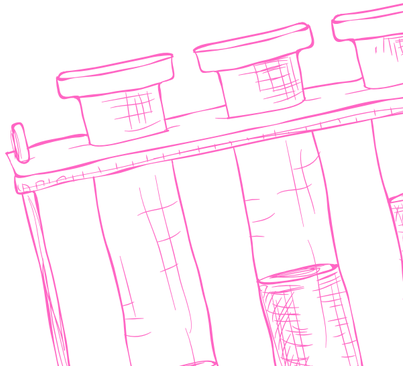
CARBOHYDRATES ALSO SERVE AS A STRUCTURAL COMPONENT OF MANY

ORGANISMS, INCLUDING THE CELL WALLS OF BACTERIA, THE EXOSKELETON OF INSECTS.

AND THE FIBROUS CELLULOSE OF PLANTS. THE EMPIRIC FORMULA FOR MANY OF THE SIMPLER

CARBOHYDRATES IS (CH_{2}O)N, WHERE N \GEQ 3, HENCE THE NAME "HYDRATE OF CARBON".

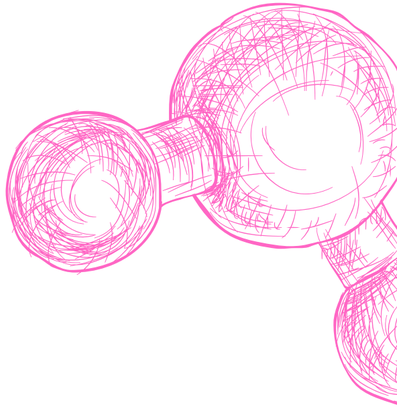




DIETARY CARBOHYDRATE DIGESTION

DIETARY CARBOHYDRATE DIGESTION OCCURS PRIMARILY IN THE MOUTH AND INTESTINAL LUMEN, FACILITATED BY ENZYMES

CALLED GLYCOSIDE HYDROLASES (GLYCOSIDASES). THESE ENZYMES, MAINLY ENDOGLYCOSIDASES AND DISACCHARIDASES,

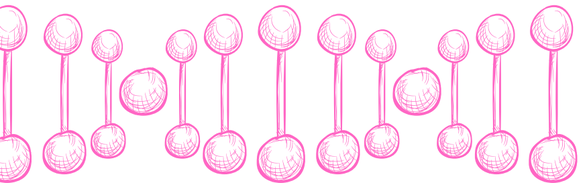


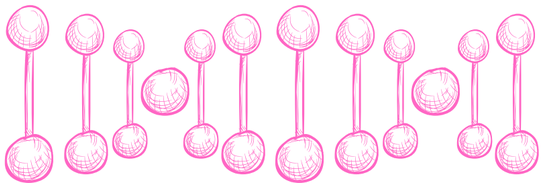
BREAK DOWN POLYSACCHARIDES AND DISACCHARIDES INTO MONOSACCHARIDES

GLYCOSIDASES ARE SPECIFIC FOR THE STRUCTURE OF GLYCOSYL RESIDUES AND THE BONDS THEY

HYDROLYZE. THE END PRODUCTS —GLUCOSE, GALACTOSE, AND FRUCTOSE—ARE ABSORBED BY

ENTEROCYTES IN THE SMALL INTESTINE.



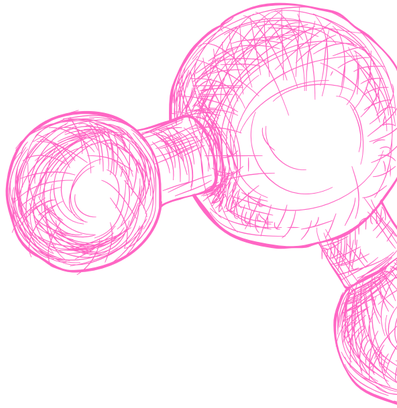


SALIVARY ALPHA-AMYLASE

SALIVARY ALPHA-AMYLASE BEGINS THE DIGESTION OF DIETARY POLYSACCHARIDES, SUCH AS STARCH

AND GLYCOGEN, BY HYDROLYZING RANDOM ALPHA(1$\RIGHTARROW4) BONDS DURING CHEWING. HUMANS

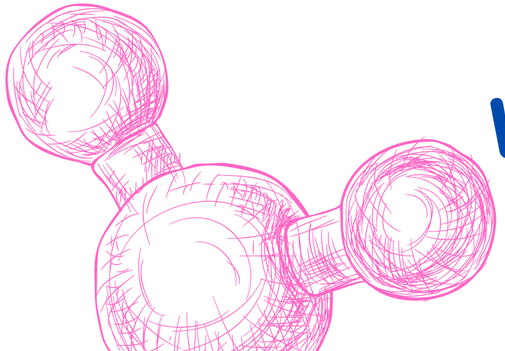
CANNOT DIGEST CELLULOSE DUE TO THE ABSENCE OF BETA(1\RIGHTARROW$4)-ENDOGLYCOSIDASES

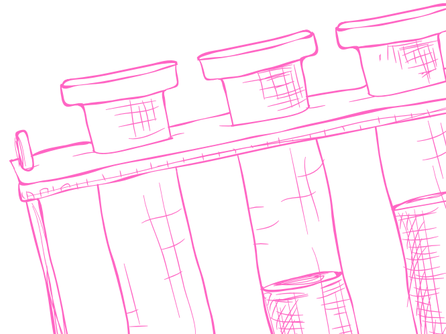


THE RESULTANT MIXTURE INCLUDES DEXTRINS, WHICH ARE SHORT OLIGOSACCHARIDES WITH

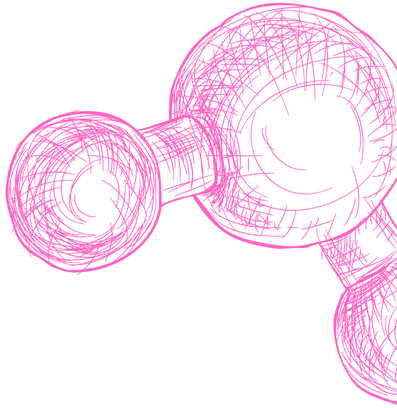
BRANCHED AND UNBRANCHED STRUCTURES, ALONG WITH SOME DISACCHARIDES. CARBOHYDRATE

DIGESTION TEMPORARILY STOPS IN THE STOMACH DUE TO HIGH ACIDITY, WHICH INACTIVATES SALIVARY ALPHA-AMYLASE.

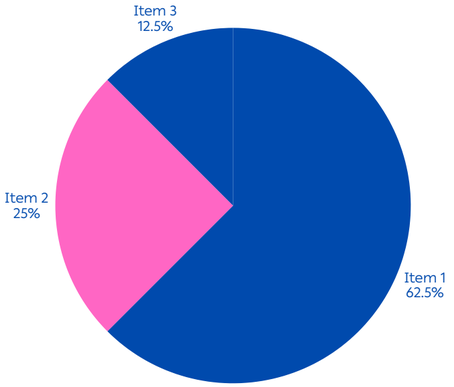




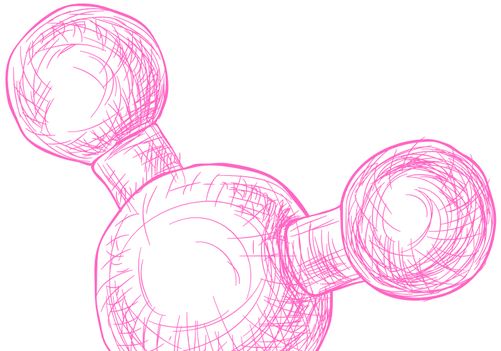
INTESTINAL DISACCHARIDASES IN THE SMALL INTESTINE, BICARBONATE FROM THE PANCREAS NEUTRALIZES

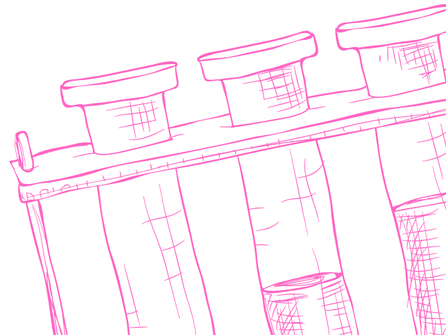


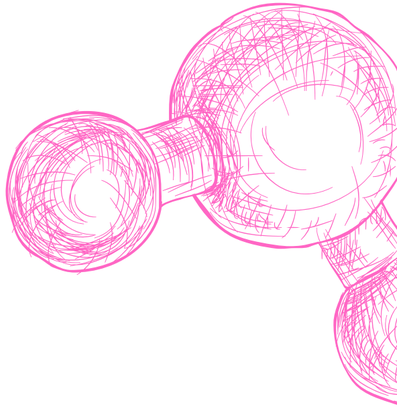
ACIDIC STOMACH CONTENTS, ALLOWING PANCREATIC ALPHA-AMYLASE TO CONTINUE STARCH DIGESTION. FINAL



DIGESTION OCCURS AT THE MUCOSAL LINING OF THE DUODENUM AND UPPER JEJUNUM



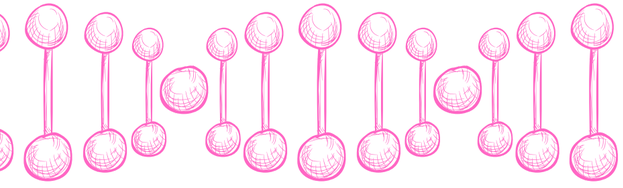


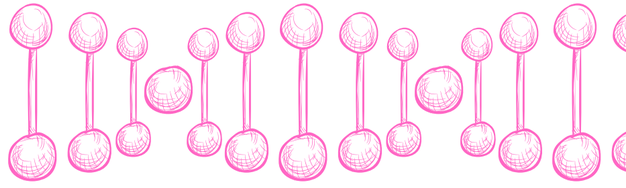


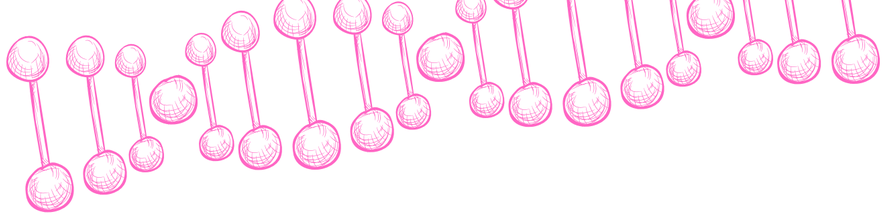
INVOLVING DISACCHARIDASES LIKE ISOMALTASE, MALTASE, SUCRASE, LACTASE, AND TREHALASE. THESE ENZYMES

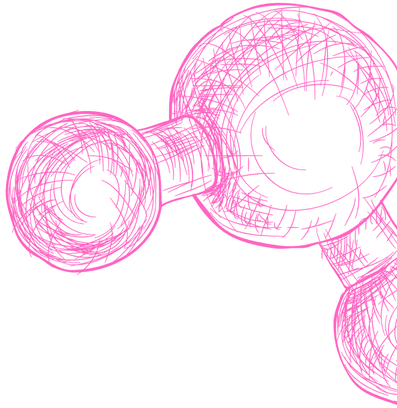
CLEAVE SPECIFIC GLYCOSIDIC BONDS IN DISACCHARIDES, PRODUCING MONOSACCHARIDES SUCH AS GLUCOSE

FRUCTOSE, AND GALACTOSE. THEY ARE TRANSMEMBRANE PROTEINS LOCATED ON THE BRUSH BORDER OF ENTEROCYTES.









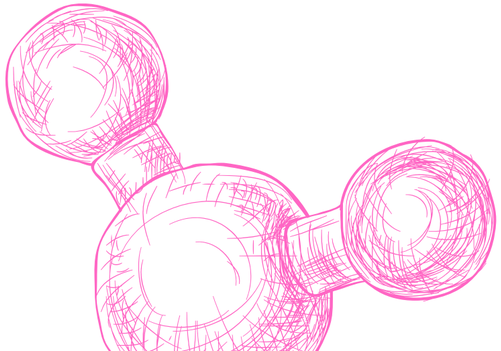
ABNORMAL DEGRADATION OF DISACCHARIDES :

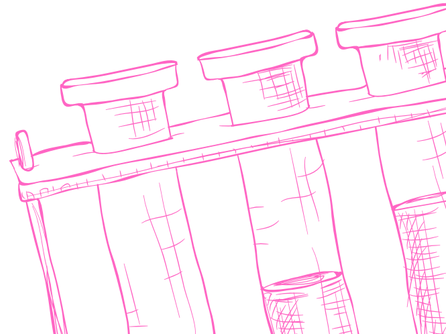
DIGESTIVE ENZYME DEFICIENCIES :

GENETIC DEFICIENCIES OF DISACCHARIDASES LEAD TO DISACCHARIDE INTOLERANCE, WHILE INTESTINAL DISEASES, MALNUTRITION, AND CERTAIN

DRUGS CAN ALSO IMPAIR DISACCHARIDE DEGRADATION. SEVERE DIARRHEA CAN CAUSE A TEMPORARY LOSS OF BRUSH BORDER ENZYMES, MAKING IT DIFFICULT FOR PATIENTS TO CONSUME DAIRY OR SUCROSE WITHOUT

WORSENING THEIR CONDITION.





LACTOSE INTOLERANCE:

LACTOSE INTOLERANCE: OVER 60% OF ADULTS WORLDWIDE ARE LACTOSE INTOLERANT DUE TO LACTASE DEFICIENCY

WITH HIGHER PREVALENCE IN INDIVIDUALS OF AFRICAN OR ASIAN DESCENT COMPARED TO THOSE OF NORTHERN EUROPEAN HERITAGE. THIS AGE-DEPENDENT LOSS OF

LACTASE TYPICALLY BEGINS AROUND AGE 2 AND IS LINKED TO GENETIC VARIATIONS ON CHROMOSOME 2. MANAGEMENT INCLUDES REDUCING MILK INTAKE

CONSUMING YOGURT AND CERTAIN CHEESES, AND USING LACTASE SUPPLEMENTS.

SUCRASE-ISOMALTASE DEFICIENCY:

SUCRASE-ISOMALTASE (SI) DEFICIENCY LEADS TO SUCROSE INTOLERANCE, PREVIOUSLY CONSIDERED RARE BUT NOW AFFECTING UP TO 9% OF AMERICANS OF EUROPEAN

DESCENT. INITIALLY THOUGHT TO BE AN AUTOSOMAL-RECESSIVE DISORDER, 25 MUTATIONS IN THE SUCROSE GENE HAVE BEEN IDENTIFIED, WITH HOMOZYGOUS

INDIVIDUALS EXPERIENCING SEVERE SYMPTOMS LIKE OSMOTIC DIARRHEA AND VOMITING. HETEROZYGOUS CARRIERS MAY HAVE MILDER SYMPTOMS, INCLUDING

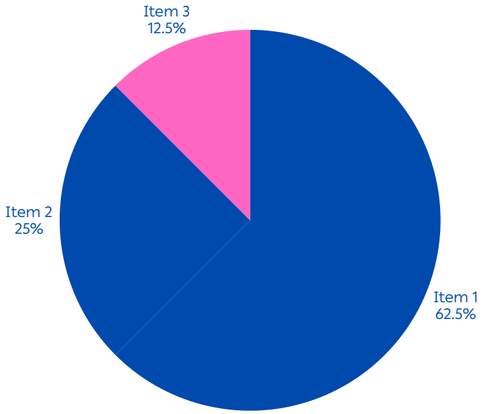
CHRONIC DIARRHEA AND BLOATING. TREATMENT INVOLVES RESTRICTING SUCROSE INTAKE AND ENZYME REPLACEMENT THERAPY

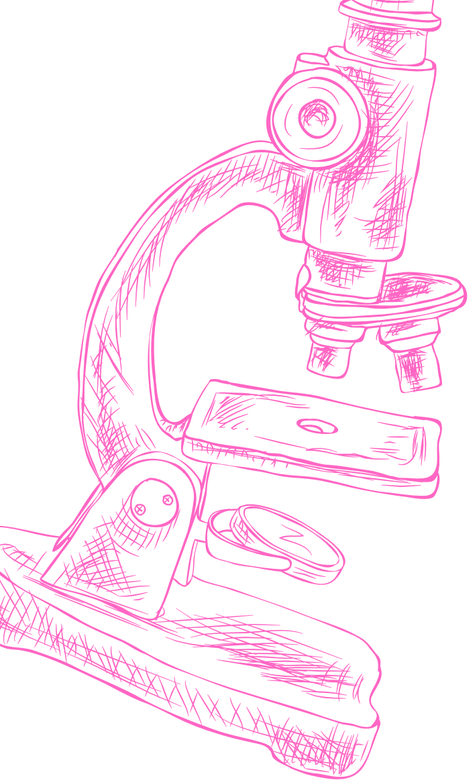
DIAGNOSIS OF ENZYME DEFICIENCIES :

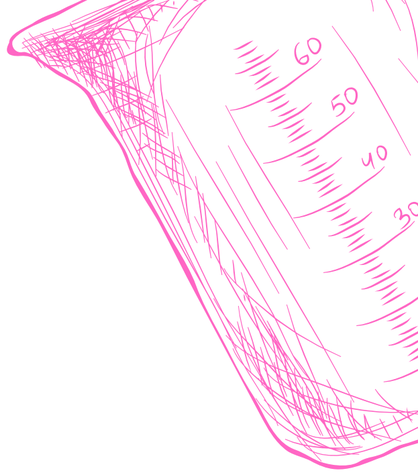
ENZYME DEFICIENCIES CAN BE DIAGNOSED THROUGH ORAL TOLERANCE TESTS WITH

SPECIFIC DISACCHARIDES, WHILE MEASURING H_{2} IN BREATH EFFECTIVELY INDICATES

UNABSORBED CARBOHYDRATES METABOLIZED BY INTESTINAL FLORA.







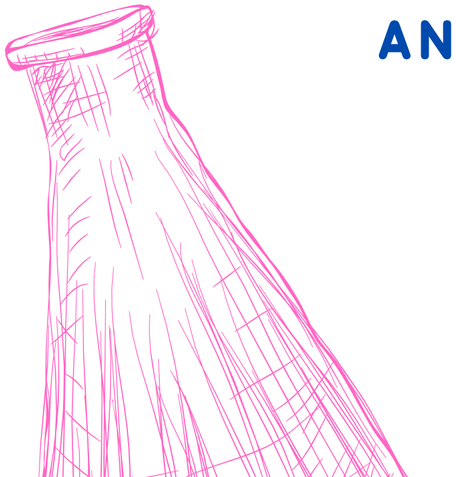
IV. LIPIDS :

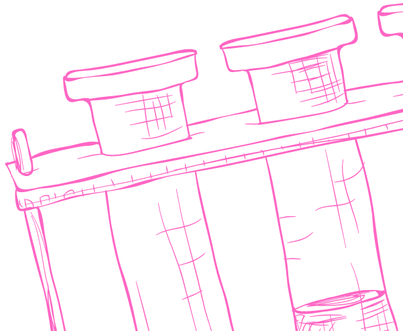
LIPIDS ARE A DIVERSE GROUP OF HYDROPHOBIC ORGANIC MOLECULES THAT ARE OFTEN COMPARTMENTALIZED IN MEMBRANES OR

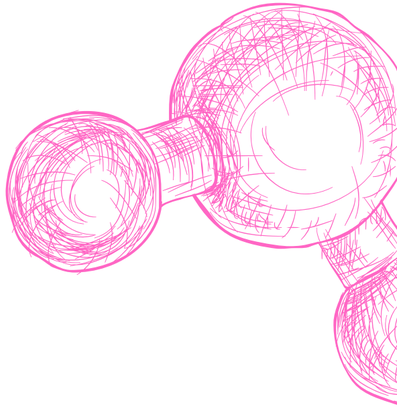
STORED AS TRIACYLGLYCEROL DROPLETS IN ADIPOCYTES. THEY SERVE AS A PRIMARY ENERGY SOURCE, CREATE HYDROPHOBIC BARRIERS IN CELL

, AND PLAY ROLES IN TRANSPORTING FAT-SOLUBLE VITAMINS AND HORMONES. IMBALANCES IN LIPID METABOLISM CAN LEAD TO SIGNIFICANT HEALTH

ISSUES, INCLUDING ATHEROSCLEROSIS, DIABETES, AND OBESITY.







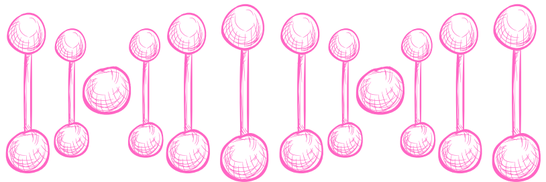
DIGESTION IN THE STOMACH :

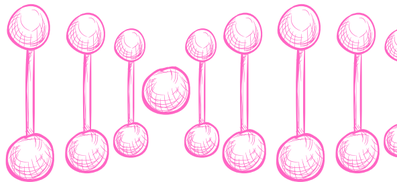
LIPID DIGESTION IN THE STOMACH IS LIMITED AND PRIMARILY INVOLVES LINGUAL LIPASE FROM THE TONGUE AND GASTRIC LIPASE FROM THE GASTRIC

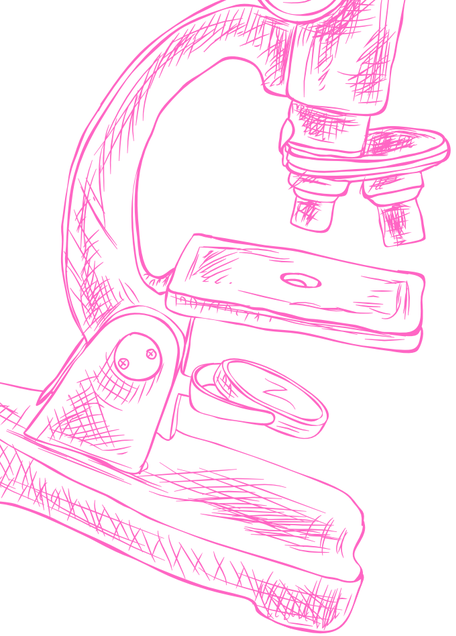
MUCOSA, BOTH ACTIVE AT A PH OF 4 TO 6. THESE ENZYMES HYDROLYZE FATTY ACIDS FROM TRIACYLGLYCEROL (TAG), ESPECIALLY THOSE

WITH SHORT- OR MEDIUM-CHAIN LENGTHS, MAKING THEM CRUCIAL FOR INFANTS RELIANT ON MILK FAT FOR CALORIES. THEY ALSO ASSIST

INDIVIDUALS WITH PANCREATIC INSUFFICIENCY, SUCH AS CYSTIC FIBROSIS, IN DIGESTING TAG DESPITE LOW PANCREATIC LIPASE LEVELS.







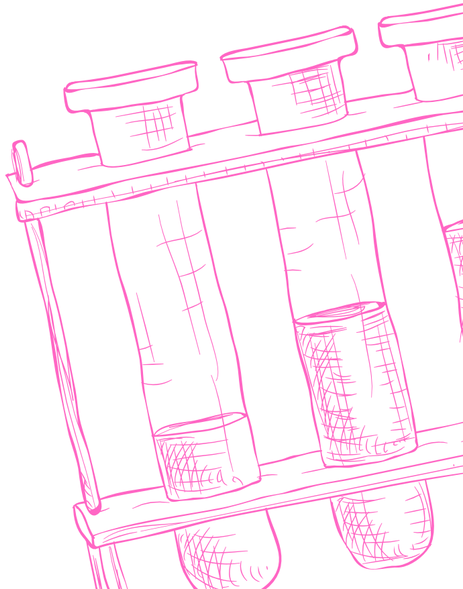
CYSTIC FIBROSIS:

CYSTIC FIBROSIS (CF) IS AN AUTOSOMAL-RECESSIVE DISORDER CAUSED BY MUTATIONS IN THE CFTR GENE, LEADING TO DEFECTIVE CHLORIDE CHANNELS IN

VARIOUS EPITHELIAL TISSUES. THIS RESULTS IN DECREASED CHLORIDE SECRETION AND INCREASED SODIUM AND WATER UPTAKE, CAUSING THICK

MUCUS THAT OBSTRUCTS PANCREATIC DUCTS AND LEADS TO PANCREATIC INSUFFICIENCY. TREATMENT INVOLVES ENZYME REPLACEMENT THERAPY AND

SUPPLEMENTATION WITH FAT-SOLUBLE VITAMINS. CF ALSO CAUSES CHRONIC LUNG INFECTIONS AND CAN LEAD TO MALE INFERTILITY.

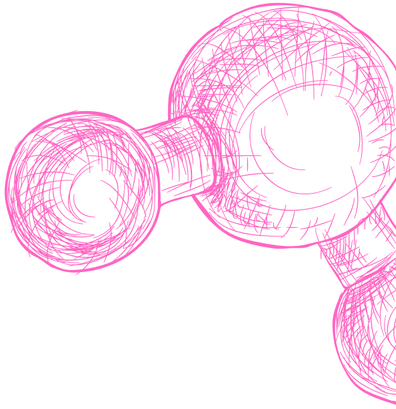


EMULSIFICATION IN THE SMALL INTESTINE:

DIETARY LIPID EMULSIFICATION OCCURS IN THE DUODENUM, INCREASING THE SURFACE AREA OF LIPID DROPLETS FOR EFFECTIVE ENZYME ACTION.

THIS PROCESS RELIES ON THE DETERGENT PROPERTIES OF CONJUGATED BILE SALTS AND MECHANICAL MIXING FROM PERISTALSIS. BILE

SALTS, DERIVED FROM CHOLESTEROL AND MODIFIED WITH GLYCINE OR TAURINE, STABILIZE THE LIPID DROPLETS, PREVENTING COALESCENCE AS THEY ARE BROKEN DOWN.

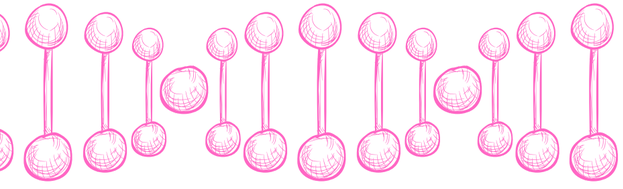


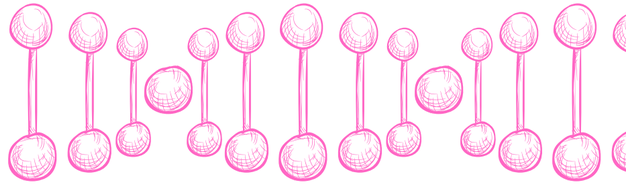
DEGRADATION BY PANCREATIC ENZYMES:

THE DIETARY TAG, CHOLESTERYL ESTERS, AND PHOSPHOLIPIDS ARE ENZYMATICALLY

DEGRADED (DIGESTED) IN THE SMALL INTESTINE BY PANCREATIC ENZYMES, WHOSE SECRETION

IS HORMONALLY CONTROLLED.



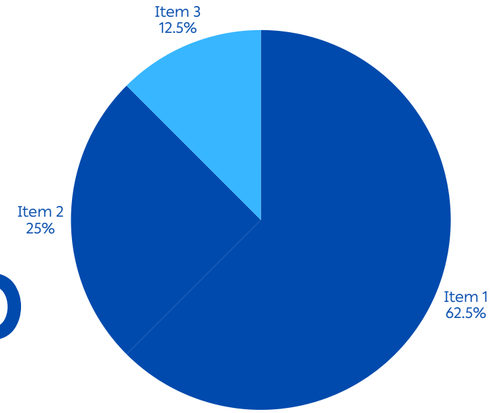


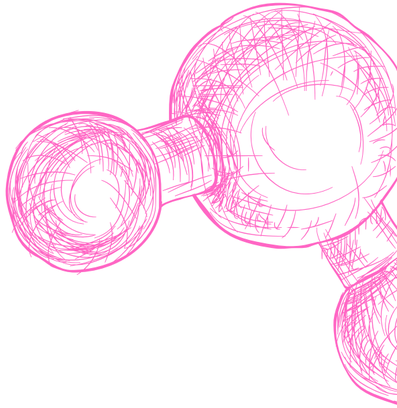
TRIACYLGLYCEROL DEGRADATION:

TAG MOLECULES ARE TOO LARGE FOR EFFICIENT UPTAKE BY

INTESTINAL ENTEROCYTES, SO THEY ARE HYDROLYZED BY

PANCREATIC LIPASE, PRODUCING



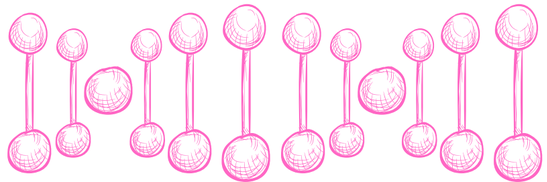


MONOACYLGLYCEROL (2-MAG) AND FREE FATTY ACIDS (FFA) :

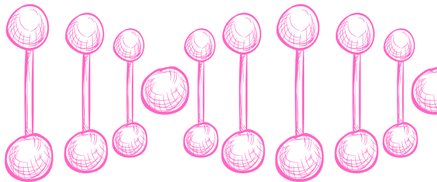
COLIPASE, WHICH IS ACTIVATED IN THE INTESTINE, BINDS TO LIPASE AND

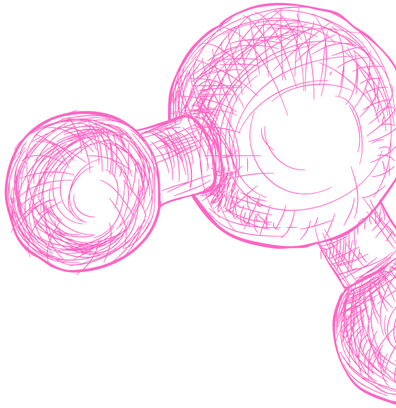
ENHANCES ITS ACTIVITY DESPITE THE PRESENCE OF BILE SALTS. ORLISTAT

AN ANTIOBESITY DRUG, INHIBITS THESE LIPASES TO REDUCE FAT ABSORPTION



AND PROMOTE WEIGHT LOSS.



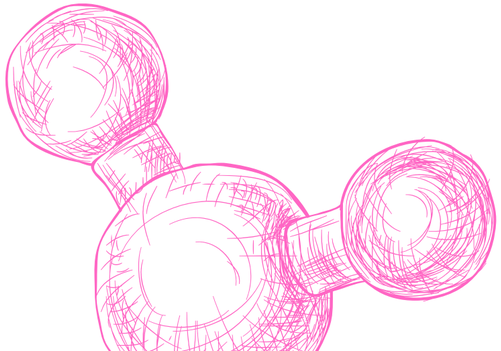


CHOLESTERYL ESTER DEGRADATION:

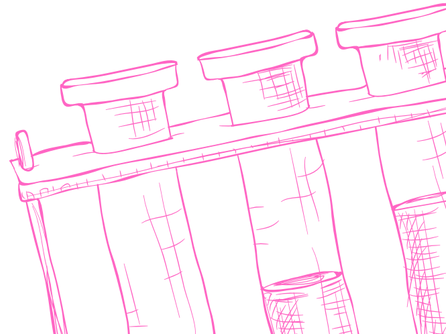
MOST DIETARY CHOLESTEROL IS IN THE FREE FORM, WITH 10% TO 15% AS CHOLESTERYL

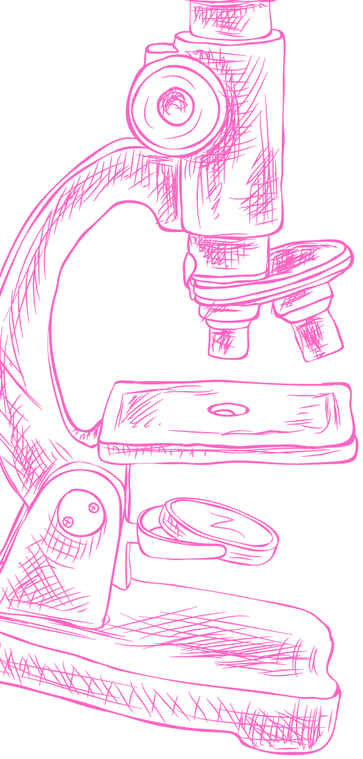
ESTERS, WHICH ARE HYDROLYZED BY PANCREATIC CHOLESTERYL ESTER

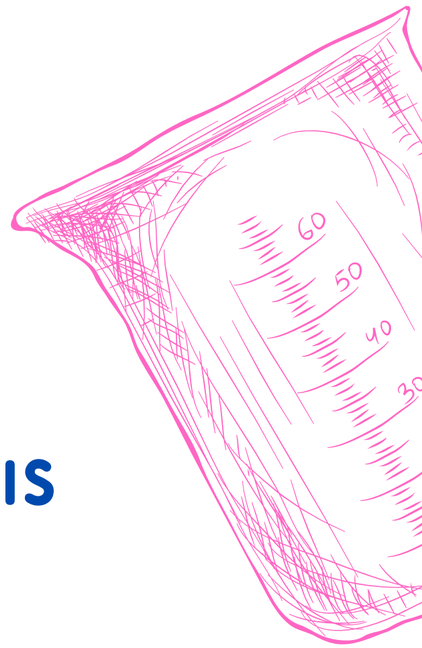
HYDROLASE TO PRODUCE CHOLESTEROL AND FREE FATTY ACIDS, WITH ENZYME



ACTIVITY ENHANCED BY BILE SALTS.







PHOSPHOLIPID DEGRADATION:

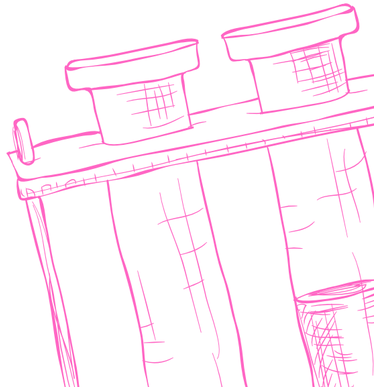
PANCREATIC JUICE CONTAINS PROENZYME PHOSPHOLIPASE A2, ACTIVATED BY TRYPSIN

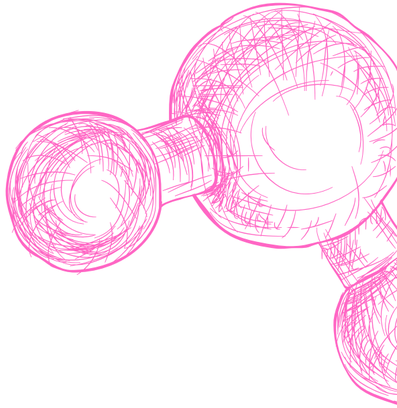
AND REQUIRING BILE SALTS FOR OPTIMAL ACTIVITY. THIS ENZYME REMOVES A FATTY ACID

FROM CARBON 2 OF PHOSPHOLIPIDS, PRODUCING LYSOPHOSPHOLIPIDS, SUCH AS

LYSOPHOSPHATIDYLCHOLINE, WHICH CAN BE FURTHER DEGRADED OR ABSORBED. THE REMAINING FATTY ACID AT CARBON 1 CAN BE

REMOVED BY LYSOPHOSPHOLIPASE, YIELDING GLYCERYLPHOSPHORYL BASES.





CONTROL:

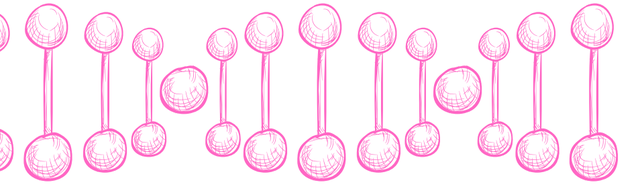
PANCREATIC SECRETION OF HYDROLYTIC ENZYMES FOR LIPID DIGESTION IN THE SMALL INTESTINE IS HORMONALLY

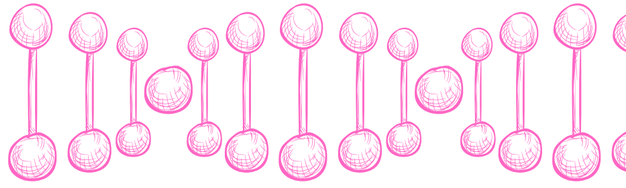
REGULATED BY ENTEROENDOCRINE CELLS. CHOLECYSTOKININ (CCK) IS RELEASED IN RESPONSE TO

LIPIDS AND PARTIALLY DIGESTED PROTEINS, STIMULATING GALLBLADDER CONTRACTION AND PANCREATIC ENZYME

RELEASE WHILE REDUCING GASTRIC MOTILITY. SECRETIN IS PRODUCED IN RESPONSE TO LOW PH, PROMPTING THE

PANCREAS TO RELEASE BICARBONATE-RICH FLUID TO NEUTRALIZE INTESTINAL CONTENTS FOR OPTIMAL ENZYME ACTIVITY.





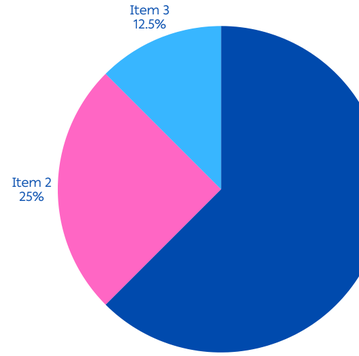
ABSORPTION BY ENTEROCYTES:

FFA, FREE CHOLESTEROL, AND 2-MAG FORM MIXED MICELLES WITH BILE SALTS AND FAT-SOLUBLE VITAMINS

WHICH FACILITATE LIPID ABSORPTION AT THE BRUSH BORDER OF ENTEROCYTES IN THE JEJUNUM. THE

HYDROPHILIC SURFACE OF MICELLES HELPS TRANSPORT HYDROPHOBIC LIPIDS THROUGH THE UNSTIRRED WATER

LAYER FOR ABSORPTION.



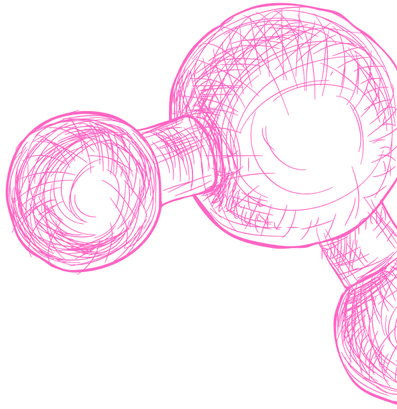
CHOLESTEROL AND PLANT :

STEROLS ENTER ENTEROCYTES VIA THE NPC1L1 PROTEIN, AND EZETIMIBE INHIBITS

THIS PROCESS TO REDUCE CHOLESTEROL ABSORPTION. SHORT- AND MEDIUM-CHAIN

FATTY ACIDS ARE WATER-SOLUBLE AND DO NOT REQUIRE MIXED MICELLES FOR

ABSORPTION.



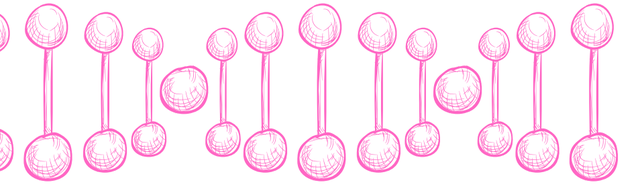
SECRETION FROM ENTEROCYTES:

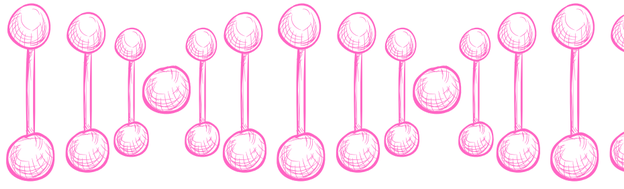
NEWLY RESYNTHESIZED TAG AND CHOLESTERYL ESTERS ARE PACKAGED

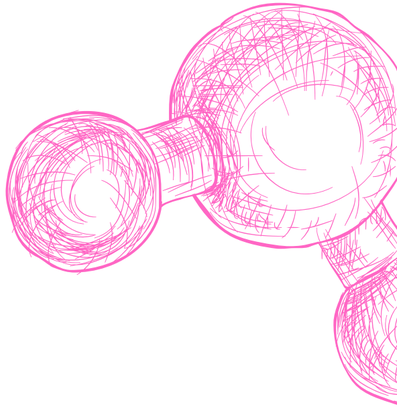
INTO LIPID DROPLETS SURROUNDED BY A LAYER OF PHOSPHOLIPIDS, NONESTERIFIED

CHOLESTEROL, AND APOLIPOPROTEIN B-48 TO ENHANCE SOLUBILITY AND PREVENT

AGGREGATION.





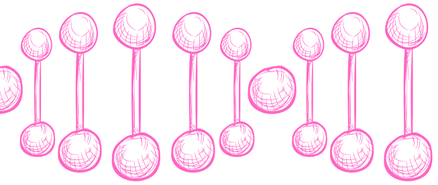


THESE LIPOPROTEIN PARTICLES, CALLED CHYLOMICRONS, ARE RELEASED FROM

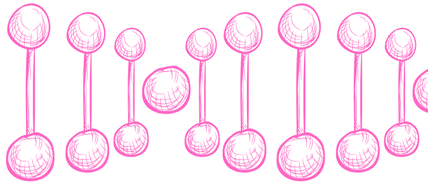
ENTEROCYTES INTO THE LYMPHATIC SYSTEM, GIVING IT A MILKY APPEARANCE KNOWN AS

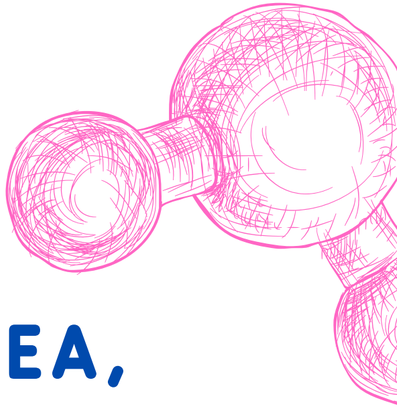
CHYLE. CHYLOMICRONS TRAVEL THROUGH THE LYMPHATIC SYSTEM TO THE THORACIC

DUCT AND ENTER THE BLOODSTREAM, WHERE THEY MATURE BY ACQUIRING



APOLIPOPROTEINS E AND C-II FROM HDL.





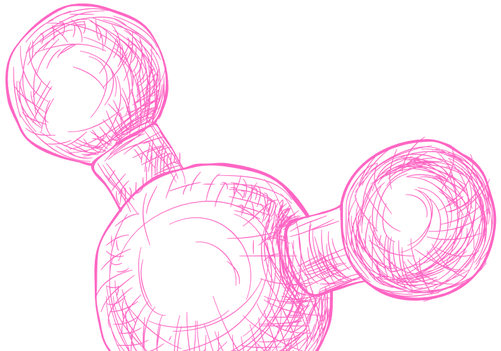
LIPID MALABSORPTION:

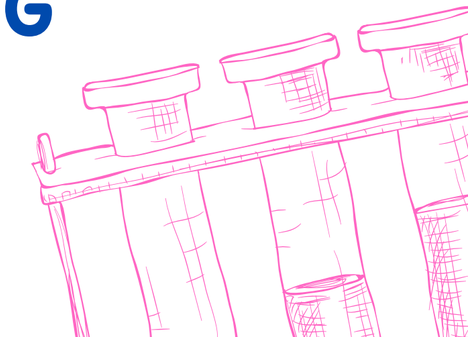
LIPID MALABSORPTION LEADS TO STEATORRHEA, CHARACTERIZED BY EXCESS LIPIDS IN FECES, DUE

TO DISTURBANCES IN DIGESTION OR ABSORPTION. CONDITIONS SUCH AS CYSTIC FIBROSIS, SHORT

BOWEL SYNDROME, AND BARIATRIC SURGERY CAN CONTRIBUTE TO THIS ISSUE BY IMPAIRING

DIGESTION OR ENZYME SECRETION.





DIETARY PROTEIN DIGESTION :

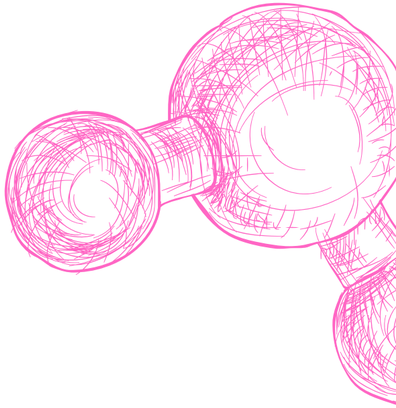
PROTEINS ARE THE MOST ABUNDANT AND FUNCTIONALLY DIVERSE MOLECULES IN LIVING

SYSTEMS, ESSENTIAL FOR PROCESSES LIKE METABOLISM, MOVEMENT, AND STRUCTURAL

SUPPORT. THEY INCLUDE ENZYMES, HORMONES, CONTRACTILE PROTEINS, AND TRANSPORT

PROTEINS SUCH AS HEMOGLOBIN AND ALBUMIN.

DESPITE THEIR DIVERSITY, ALL PROTEINS ARE LINEAR POLYMERS OF AMINO ACIDS, WITH

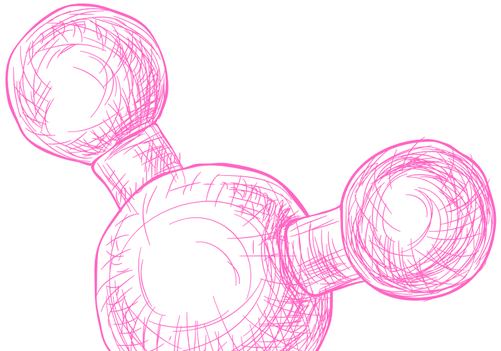


UNIQUE STRUCTURES THAT ENABLE SPECIFIC BIOLOGICAL FUNCTIONS. MOST DIETARY

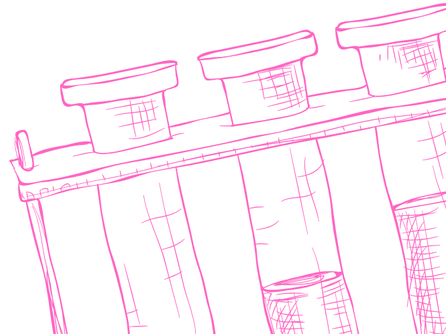
NITROGEN COMES FROM PROTEIN. SINCE PROTEINS ARE TOO LARGE FOR INTESTINAL

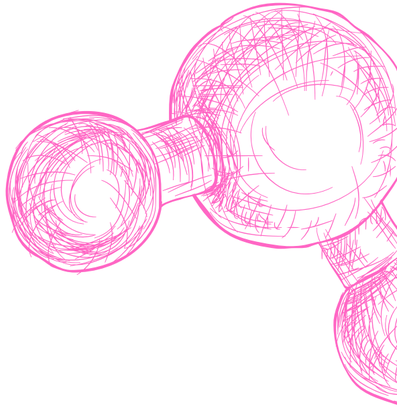
ABSORPTION, THEY MUST BE HYDROLYZED INTO DI- AND TRIPEPTIDES AND INDIVIDUAL AMINO

ACIDS. PROTEOLYTIC ENZYMES FROM THE STOMACH, PANCREAS, AND SMALL INTESTINE



FACILITATE THIS DEGRADATION.



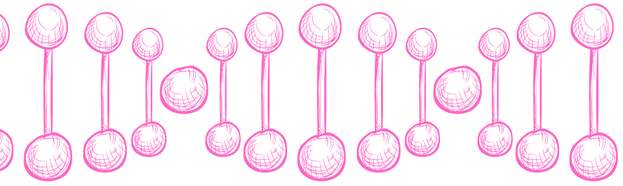


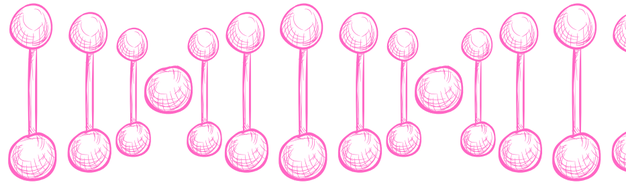
DIGESTION BY GASTRIC SECRETION:

THE DIGESTION OF PROTEINS BEGINS IN THE STOMACH, WHICH SECRETES

GASTRIC JUICE, A UNIQUE SOLUTION CONTAINING HYDROCHLORIC ACI

D (HCL) AND THE PROENZYME .PEPSINOGEN



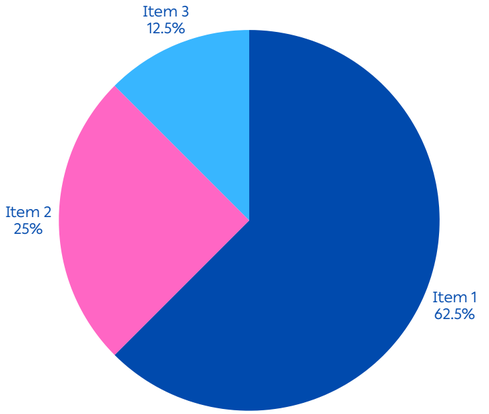


HYDROCHLORIC ACID: STOMACH HCL, WITH A PH OF 2 TO 3, DOES

NOT HYDROLYZE PROTEINS BUT DENATURES THEM AND KILLS SOME

BACTERIA, ENHANCING THEIR SUSCEPTIBILITY TO HYDROLYSIS BY

PROTEASES.



PEPSIN:

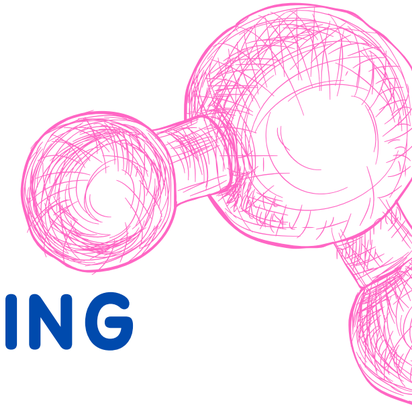
PEPSINOGEN, AN ACID-STABLE ENDOPEPTIDASE SECRETED BY STOMACH CHIEF CELLS AS AN

INACTIVE ZYMOGEN, UNDERGOES AUTOCATALYSIS IN THE PRESENCE OF HCL TO

BECOME ACTIVE PEPIN. THIS ACTIVE ENZYME CLEAVES DIETARY PROTEINS INTO POLYPEPTIDES

AND SOME FREE AMINO ACIDS. ZYMOGENS GENERALLY CONTAIN EXTRA AMINO ACIDS THAT

PREVENT CATALYTIC ACTIVITY UNTIL REMOVED FOR PROPER ENZYME FOLDING.



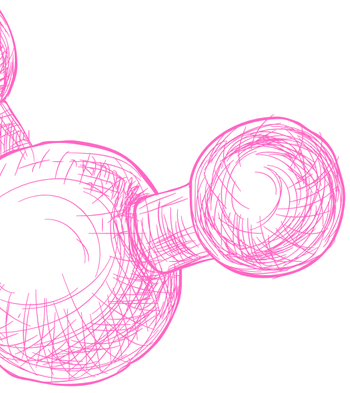
DIGESTION BY PANCREATIC ENZYMES :

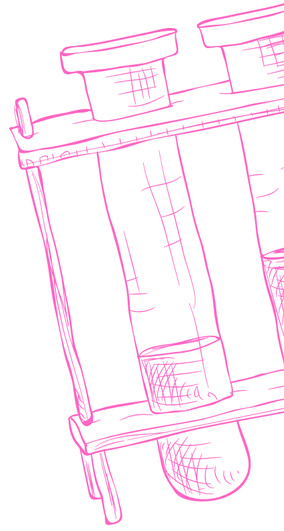
SPECIFICITY: PANCREATIC PROTEASES, INCLUDING ENDOPEPTIDASES AND

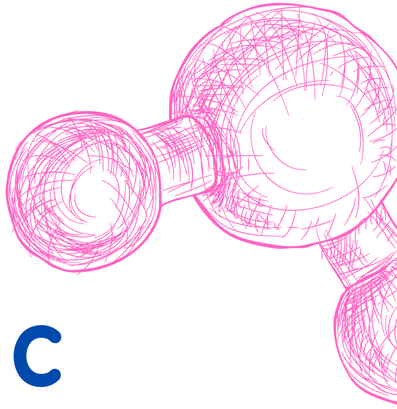
EXOPEPTIDASES, CLEAVE POLYPEPTIDES INTO OLIGOPEPTIDES AND AMINO ACIDS BASED ON

THE SPECIFICITY FOR ADJACENT R-GROUPS. FOR INSTANCE, TRYPSIN SPECIFICALLY ACTS ON

PEPTIDE BONDS WITH ARGININE OR LYSINE.





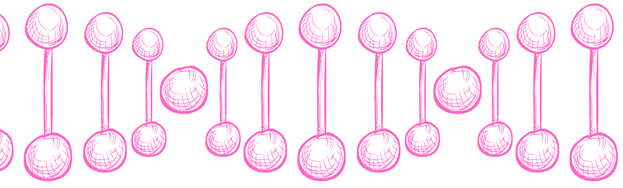


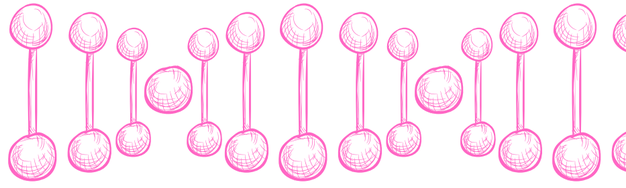
ZYMOGEN RELEASE:

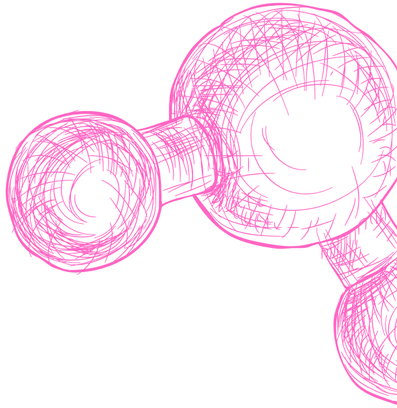
THE RELEASE OF INACTIVE PANCREATIC ZYMOGENS IS

TRIGGERED BY CHOLECYSTOKININ, A HORMONE SECRETED IN THE

SMALL INTESTINE, WHICH STIMULATES THE PANCREAS.





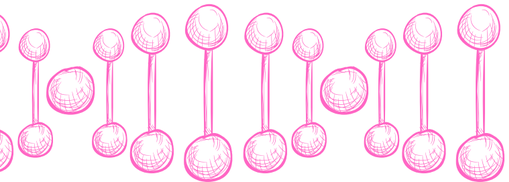


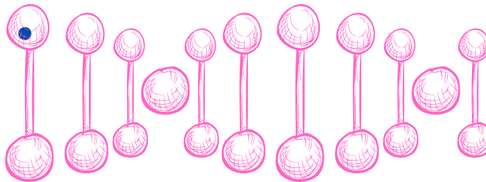
ZYMOGEN ACTIVATION:

ENTEROPEPTIDASE, FOUND ON THE INTESTINAL MUCOSAL SURFACE, ACTIVATES TRYPSINOGEN TO

TRYPSIN BY REMOVING A HEXAPEPTIDE. TRYPSIN THEN ACTIVATES OTHER ZYMOGENS, INITIATING A

CASCADE OF PROTEOLYTIC ACTIVITY.





DIGESTION ABNORMALITIES:

DEFICIENCIES IN PANCREATIC SECRETION, DUE TO CONDITIONS LIKE

CHRONIC PANCREATITIS OR CYSTIC FIBROSIS, LEAD TO INCOMPLETE

DIGESTION AND ABSORPTION OF FATS AND PROTEINS, RESULTING IN

STEATORRHEA AND UNDIGESTED PROTEINS IN FECES.

DIGESTION OF OLIGOPEPTIDES BY SMALL INTESTINE ENZYMES :

THE LUMINAL SURFACE OF THE ENTEROCYTES CONTAINS

AMINOPEPTIDASE, AN EXOPEPTIDASE THAT REPEATEDLY CLEAVES THE N-

TERMINAL RESIDUE FROM OLIGOPEPTIDES TO PRODUCE EVEN SMALLER PEPTIDES

AND FREE AMINO ACIDS.

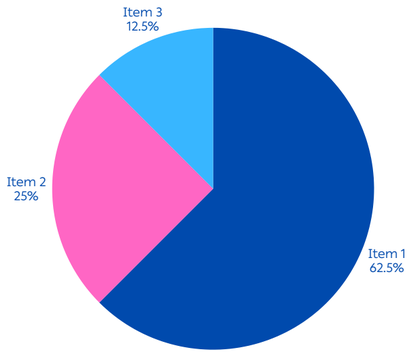
AMINO ACID AND SMALL PEPTIDE INTESTINAL ABSORPTION :

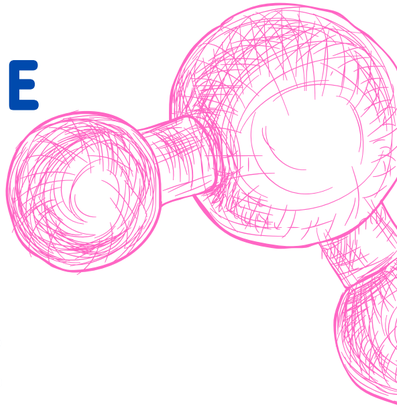
MOST FREE AMINO ACIDS ENTER ENTEROCYTES THROUGH SODIUM-

DEPENDENT SECONDARY ACTIVE TRANSPORT VIA SOLUTE CARRIER (SLC)

PROTEINS. DI- AND TRIPEPTIDES ARE ABSORBED BY A PROTON-LINKED

TRANSPORTER (PEPT1) AND HYDROLYZED INTO FREE AMINO ACIDS.





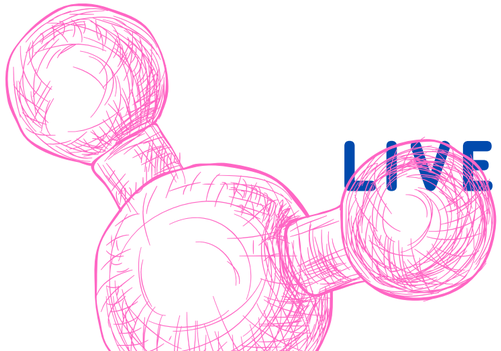
FREE AMINO ACIDS ARE THEN RELEASED INTO THE PORTAL SYSTEM THROUGH

SODIUM-INDEPENDENT TRANSPORTERS ON THE BASOLATERAL MEMBRANE.

CONSEQUENTLY, ONLY FREE AMINO ACIDS APPEAR IN THE PORTAL VEIN AFTER PROTEIN

MEALS. BRANCHED-CHAIN AMINO ACIDS (BCAAS) ARE NOT METABOLIZED BY THE

LIVER AND ARE INSTEAD SENT TO MUSCLE VIA THE BLOODSTREAM.



ABSORPTION ABNORMALITIES:

THE SMALL INTESTINE AND PROXIMAL KIDNEY TUBULES SHARE AMINO ACID TRANSPORT SYSTEMS, SO DEFECTS

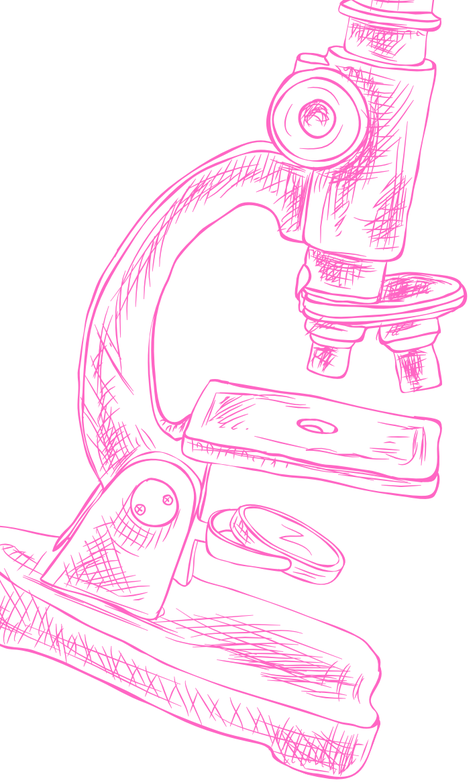
IN THESE SYSTEMS HINDER AMINO ACID ABSORPTION. IN CYSTINURIA, A DEFECT IN THE CARRIER FOR CYSTINE AND

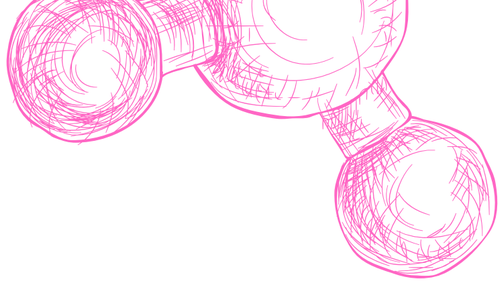
DIBASIC AMINO ACIDS (ORNITHINE, ARGININE, LYSINE) LEADS TO THEIR PRESENCE IN URINE, CAUSING KIDNEY

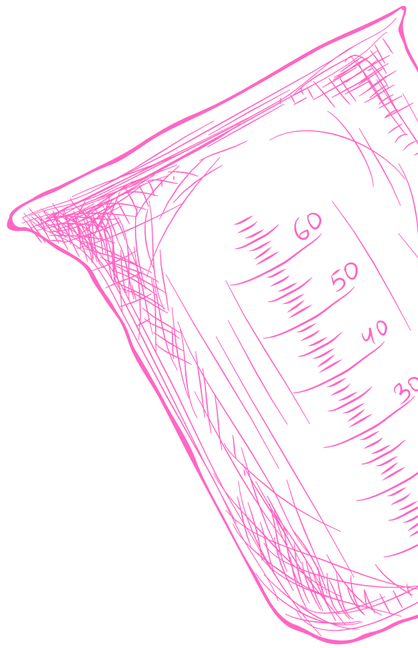
STONES. WITH A PREVALENCE OF 1 IN 7,000, CYSTINURIA IS A COMMON INHERITED DISORDER. ADDITIONALLY

DEFECTS IN TRYPTOPHAN UPTAKE CAN LEAD TO HARTNUP DISORDER, CAUSING DERMATOLOGIC AND

NEUROLOGIC SYMPTOMS.







THANK YOU FOR YOUR ATTENTION !