<table>
<tr><th colspan="4">Obesity</th></tr>
<tr><td>Is</td><td colspan="3">Inc 20% of normal body weight</td></tr>
<tr><td>It Risks</td><td colspan="3">-Hypercholesterolemia -High blood triglycerides
-CVS disorders (Hypertension…) -arthritis (gout…)
-tumros -Gallstones -Mortality (death…) -DM</td></tr>
<tr><td>BMI</td><td colspan="3">-Body Mass Index (what we use to diagnose obesity & rank it)
-uses: height, weight & body fat
-high BMI = high risks
underweight <18.5 / normal: 18.5-24.5 / overweight: 25.0 – 29.9
obese 1: 30.0 – 34.9 / obese 2: 35.0 – 39.9 / obese 3: ≥ 40</td></tr>
<tr><td rowspan="4">Anatomical fat deposition</td><td></td><td>Android</td><td>Gynoid</td></tr>
<tr><td>AKA</td><td>apple-shaped</td><td>pear-shaped</td></tr>
<tr><td>site</td><td>upper (abdominal)</td><td>lower
(around hips or gluteal)</td></tr>
<tr><td>risks</td><td>-CVS
(hypertension, coronary thrm…)
-DM
(insulin resistance…)
-dyslipidemia
(abnormal lipid lvls in blood)</td><td>-much less risks</td></tr>
<tr><td>Fat depos. Specif. site</td><td colspan="3">Subcutaneous
-in abdominal & gluteal-femoral region (butt…)
-85%
Visceral
-omental & mesenteric (near GIT)</td></tr>
<tr><td rowspan="4">Fat sites differences</td><td></td><td>Abdomen</td><td>Gluteal</td></tr>
<tr><td>Adipocytes</td><td>Small</td><td>Large</td></tr>
<tr><td>Hormones response</td><td>Very good
(both SC & visc.)</td><td>Weak</td></tr>
<tr><td>Contents release locat.</td><td>Into portal vein</td><td>Directly into circulation</td></tr>
<tr><td>Adipocytes</td><td colspan="3">-main site for <u>Triacylglycerols</u> storage
-starts initially as pre-adipocytes only when prolonged overnutrition, then can grow to mature fat cell وهلم جره...
-overnutrition causes hyperplastic hypertrophic fat tissue
-they never underego apoptosis, but they can smallen in size</td></tr>
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Ectopic fat	-once adipocytes are completely filled with fat, and overnutrition is still persistent, fat **spills over** to adjacent tissues to be stored in muscles, liver… -strongly associated with insulin resistance
Obesity causing risks	-Genetics (familial) -Sex (women) -lack activity (potato life) -Psychogenic (depression) -OH -Smoking -meds (tricyclic derivatives)
Etiology	-<u>prolonged</u> intake of energy more than output of it (it can be reversed, More out + less in = weight loss) -HT diseases: it's the center for hunger and satiety(feeling full) -ECS diseases
Appetite regulation	-by: Afferent N, hormones 1-once hungry or energy deprived Afferent N signals **HT** to <u>release its H & trigger efferent N</u> to stimulate appetite 2-Adipocytes work as ECS & releases: Leptin, adiponectin, resistin (they're released once hungry or energy deprived)
Metabolic Changes	Adipocytes send abnormal signals causing: Dyslipidemia, Glucose intolerance, Insulin resistance & Hypertension
Im obese, why lose weight?	-Lower BP -Lower blood triacylglycerols -Lower blood glc -inc blood HDL -Lower mortality -good BMR -dec energy requirement -Slow weight loss is more stable tho
Treatment	**Physical** -good diet & exercises -Restrict excessive energy diet **Meds** -Orlistat: pancreatic & gastric lipase inhibitor (inh fat digestion) -Lorcaserin: stimulates satiety **Surgery** -only with BMI >40 -only when other treatment have failed -reduces food consumption

Hormones	
Leptin	-Signals info in regard of stored fat -contributes in fat regulation by inh appetite & inc energy usage (causes weight loss) -Suppressed in starvation (shrinkage of fat vesicles) -Enhanced in well-fed state (expansion of fat vesicles) -if injected in high amount in drops body weight -in prolonged obesity body builds resistance to it -its receptor is in HT (encoded in db gene - mutation causes massive resistance, thus exagg weight gain)
Adiponectin	-abundant -more fat = less Adiponectin -more HDL = more Adiponectin -metabolic syndrome & DM = less Adiponectin -stimulates glc & FFA uptake by: muscles & liver -inh synth of: FFA & gluconeogenesis by hepatocytes -inc insulin sensitivity -improve glucose tolerance (body responses good to glc)
Ghrelin	-secreted by stomach -Stimulates appetite -its lvls drop immediately after meal intake -dec fat catabolism & energy usage (promotes weight gain) -very high levels in starvation or dieters
CCK	-secreted by almost all GIT -Sends satiety signals
Insulin	Promotes metabolisms