LUCY READING-IKKANDA
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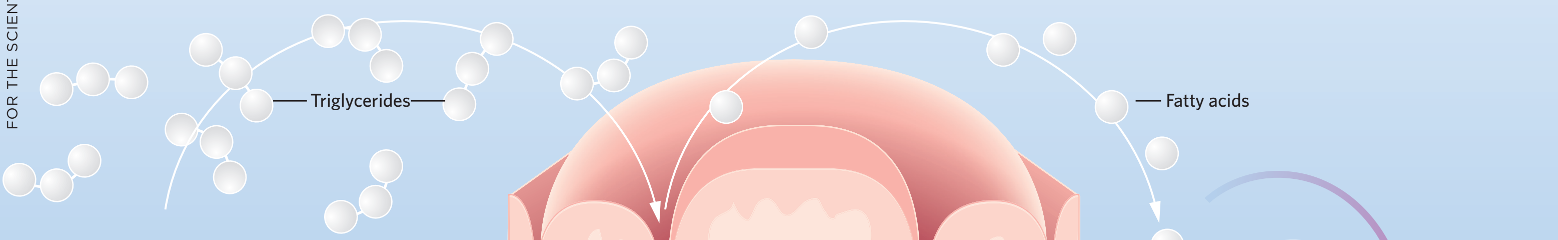
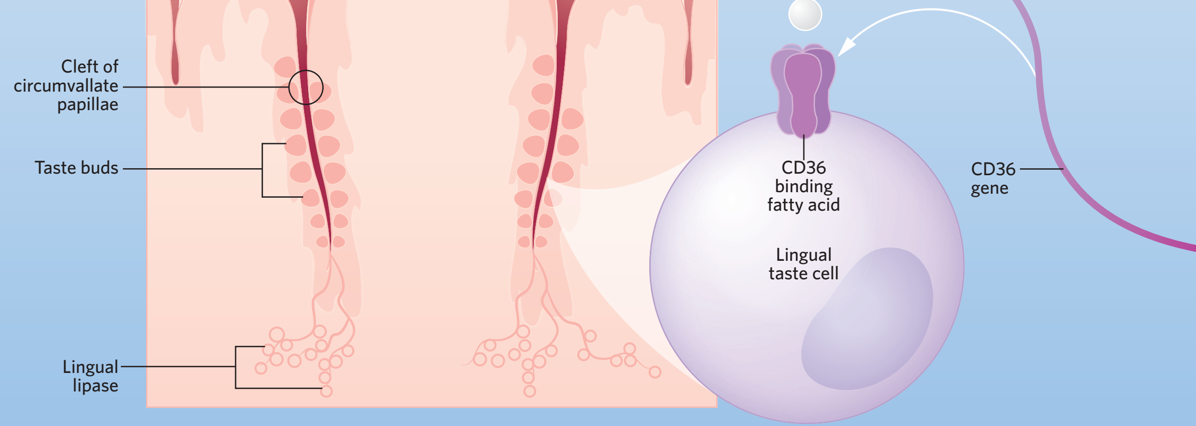
CAN WE TASTE FATS?

Although *gustin* and *TAS2R38* contribute to the supertaster phenotype and may contribute to the perception of fat texture, researchers are still looking for a receptor directly triggered by fat. One promising candidate is the protein CD36, which binds long-chain fatty acids in mice, and is expressed on taste buds. The mechanism by which the CD36 carrier protein initiates a neural signal is poorly understood. CD36 may serve as a carrier protein that transfers the fatty acid to another receptor or it may activate an ion channel that alters the excitability of taste cells.





DETECTING FATTY ACIDS

Although CD36 binds long-chain free fatty acids, most of the fat we eat is composed of triglycerides (three fatty acids bound to a glycerol backbone), which CD36 cannot bind. However, lingual lipase, an enzyme that humans secrete in small quantities, breaks down triglycerides into free fatty acids, producing low levels of fatty acids that bind to CD36 in animals and may affect fat preference.

HOW DOES THIS LEAD TO OBESITY?

Recent work has shown that people who had a particular single nucleotide difference in their CD36 gene perceived high levels of creaminess in foods regardless of the fat level. These individuals, showed high preferences for creamy, usually fattier, foods. Although the mechanism remains unclear, this finding raises the possibility that disruptions in this gene lead to both persistently high responsiveness to the oral sensation of fat and an elevated preference for fat which could lead to obesity over time.