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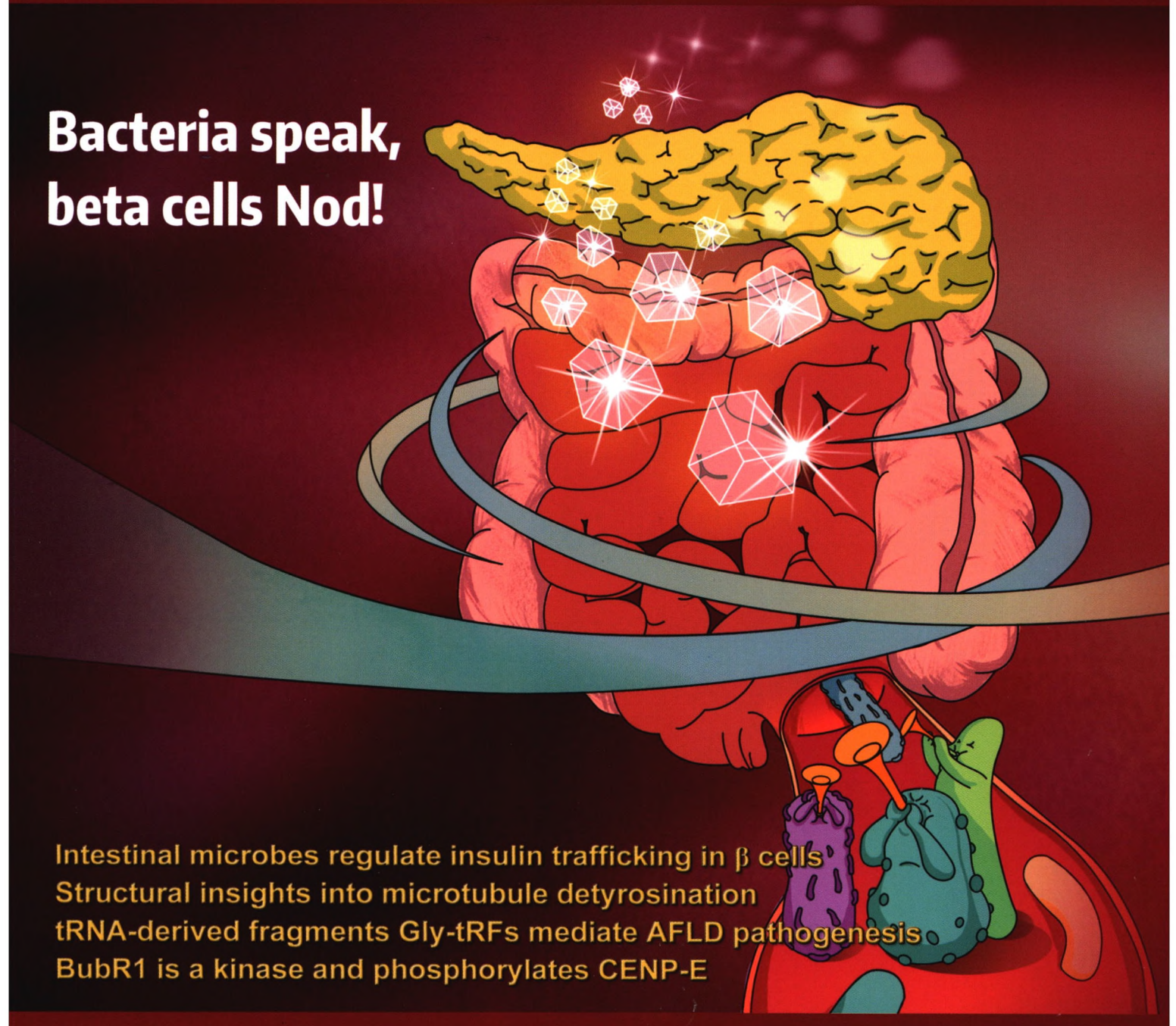
Bacteria speak, beta cells Nod!



Intestinal microbes regulate insulin trafficking in β cells
Structural insights into microtubule detyrosination
tRNA-derived fragments Gly-tRFs mediate AFLD pathogenesis
BubR1 is a kinase and phosphorylates CENP-E

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Coordinating Editor for this issue
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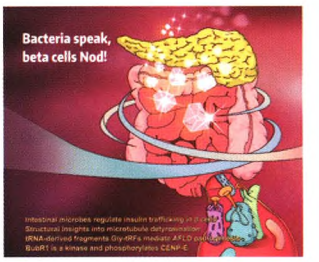
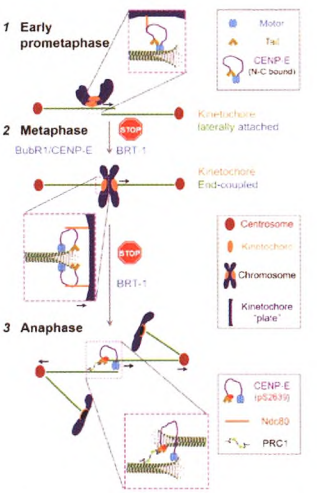


Cover: The impact of intestinal microbes goes well beyond intestine. Molecular signals derived from intestinal microbes can prompt islet beta cells to secrete insulin in a Nod1-dependent manner. When bacteria speak, islet beta cells nod. See page 516-532 by Qin Zhang et al. for details.



A working model shows that BubR1-elicited Cenp-E phosphorylation plays a central role in kinetochore-microtubule capture and central spindle assembly. See page 562-578 by Yuejia Huang et al. for details.

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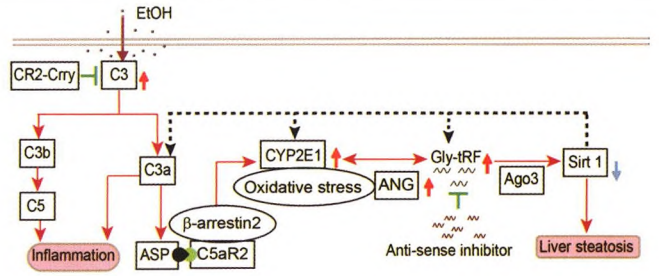

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