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MATERNAL MICROBIOTAS TRANSFER IMPACTS MICROBIOTA-GUT-BRAIN AXIS IN OFFSPRING

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Abstract Content: Introduction. The intestinal microbiota is a major player in the physiology of the host, particularly through its interactions with the intestinal epithelium. Initially transmitted from mother to child, the metabolism of primocolonizing bacteria can have an immediate and future impact on health. We previously demonstrated that neonatal transfer of different microbiota from obese-resistant (OR) or obese-prone (OP) dams has short- and long-term effects on the feeding behavior of offspring, supporting a risk of overweight. Regulation of food intake by the gut-brain axis involves enteroendocrine cells capable of detecting bacterial metabolites. **Objectives.** We looked for potential relationships between the composition and activity of transferred microbiota and gut endocrine function as a hypothesis for an early impact of microbiota-gut-brain on neurodevelopment and further eating behavior.

Method. Microbiota (vaginal, fecal, and milk-derived) from OP and OR dams, which differed in taxonomic composition, were inoculated into pups born to conventional Fischer F344 dams from birth to day 15 of life, constituting three groups: F-OP, F-OR, and F-Sham. **Results.** At 21 days of age, principal component analysis of caecocolic contents showed a discriminant separation of the 3 groups by metagenomic species and associated functions, which was reflected by only minor differences in metabolite abundance (NMR) in caecocolic supernatants. In vivo transcriptomic analysis of ileum and colon (RT-qPCR) and in vitro on the enteroendocrine STC-1 line incubated with the supernatants revealed differences in expression of some endocrine markers between the treated and F-Sham groups. The identification of the bacterial markers involved (short-chain fatty acids, neurotransmitters, bacterial peptides) is in progress via the search for statistical correlations. **Conclusion.** Maternal microbiotas transferred at birth altered the microbiota-gut-brain axis at least in part via the enteroendocrine function.