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Regular paper**Milk lipid digestion in the neonatal dog: the combined actions of gastric and bile salt stimulated lipases**Sara J. Iverson^a, , Charlotte L. Kirk^a, Margit Hamosh^a and Joseph Newsome^b^a Department of Pediatrics, Georgetown University Medical Center, Washington, D.C., U.S.A.^b Research Resources Facility, Georgetown University Medical Center, Washington, D.C., U.S.A.

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Abstract

Intragastric lipolysis may be particularly important for the digestion of milk lipid since milk fat globules are resistant to pancreatic lipase without prior disruption; milk bile salt stimulated lipase (BSSL) may supplement further intestinal hydrolysis. Previous information on gastric lipolysis has been based primarily on in vitro studies using artificial lipid emulsions containing a single component fatty acid and have focused on the preferential release of medium-chain fatty acids. The actual contribution of these enzymes to overall fat digestion in vivo on natural substrates has rarely been studied, however. The neonatal dog is an excellent model in the study of lipid digestion because, like the human, milk lipids are high in long-chain unsaturated fatty acids, milk contains BSSL and gastric lipase is the predominant lipolytic enzyme acting in the stomach. We used a combination of in vivo studies with in vitro incubations to investigate digestion of milk lipid by gastric and milk (BSSL) lipases in the suckling dog. In the first 4 weeks postpartum, 14–41% and 42–60% of milk triacylglycerol was hydrolyzed to primarily diacylglycerol and free fatty acid (FFA) in the first 30 and 60 min in the stomach, respectively. Milk lipid contained high levels (63%) of long-chain unsaturated fatty acids, which were preferentially released as FFA during in vivo gastric lipolysis, consistent with the actions and stereospecificity of gastric lipase. While levels of hydrolysis in gastric aspirates were significantly different (by age and time in stomach) at the start of in vitro studies, total hydrolysis in all incubation systems plateaued at about 65%, suggesting product inhibition by the long-chain FFA, but to a much lesser degree than previously expected from in vitro studies. The magnitude of in vivo intragastric lipolysis was 3- to 6-times greater than that predicted by in vitro assays using either milk lipid or labeled emulsion as substrate, respectively. Prior exposure to intragastric lipolysis resulted in 30% hydrolysis by BSSL compared to 5% hydrolysis without prior exposure. We suggest that previous in vitro studies have largely underestimated the actual degree of intragastric lipolysis that can occur and its activity on long-chain fatty acids; this study indicates the importance of the combined mechanism of gastric lipase and BSSL to fat digestion in the suckling neonate.

Author Keywords: Bile salt stimulated lipase; Gastric digestion; Gastric lipase; Milk lipid; Suckling neonate**Article Toolbox**

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