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Effect of Prehydrolysis of Milk Fat on its Conversion to Biogas

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ABSTRACT

Milk fat is considered to be the main limiting component of the kinetics of dairy wastewater anaerobic digestion. The objective of this work was to give a better understanding of the nonelucidated anaerobic degradation steps of milk fat. For that purpose, the kinetics of fat degradation was quantified in comparison with other milk components (lactose, proteins), regarding the milk fat polluting load and structure [globular (native state), triglycerides]. This work confirms that milk fat is degraded after a lag phase of several days, with a maximal degradation rate 2 to 5 times less than the degradation rate of the other milk components. It was shown that (1) the structure of the fat does not influence the limits of its anaerobic degradation; (2) the lag phase before biogas production is mainly due to unsaturated free fatty acids (FFA); and (3) conversion to biogas occurs at a lower rate for saturated than for unsaturated FFA. Therefore, the prehydrolysis of fat, which increases the instantaneous concentration of unsaturated FFA, sharply increases the length of the lag phase with no significant change in the maximal biogas production rate. To reduce the delay imposed in the biogas production, it is necessary to reduce the concentration of unsaturated FFA.
Key words: milk fat, hydrolysis, methanization, anaerobic degradation

INTRODUCTION

Due to stricter environmental regulations, the dairy industry is pushed to reduce both the volume (1 to 5 L per liter of processed milk) and the polluting load [0.5 to 5 g/L of chemical oxygen demand (**COD**)] of its wastewater reaching water treatment stations. The polluting load is mainly composed of milk components that can easily be converted into energy (biogas) by methanization (Li et al., 1984; Gutierrez et al., 1991). Among the milk components, milk fat, which represents 4 to 22% of the dry matter of the dairy process waters (Sage, 2005), is often considered as the main limiting component of the kinetics of dairy wastewater anaerobic digestion (Petruy and Lettinga, 1997; Vidal et al., 2000). Therefore, fat is usually separated from the waste waters and degraded with biological aerobic digestion generating extra costs: aeration for aerobic digestion and further sludge treatment processes. Moreover, because of the high specific COD of the milk fat (about 2 kg_{COD}/kg_{fat}), its removal leads to 8 to 44% potentially lost energy (methane) compared with anaerobic digestion.

Little has been reported on the comparison of anaerobic degradation kinetics of milk fat and other milk components (Perle et al., 1995; Vidal et al., 2000). Perle et al. (1995) have shown that at similar polluting load the maximum production rate of biogas from caseins, amino acids, and VFA is 2 times greater than production rate obtained from oleic acid [major milk long-chain fatty acid (**LCFA**)] and 4 times greater than the production rate obtained from anhydrous milk fat (**AMF**).

Milk fat is mainly composed of fat globules and is constituted by more than 97% of triglycerides (esters of fatty acid and glycerol, neutral hydrophobic molecules). Its anaerobic degradation follows the same steps as in aerobic digestion (Figure 1). Biodegradation is initiated by lipid hydrolysis (Weng and Jeris, 1976): hydrolysis of triglycerides to FFA and glycerol. Glycerol is metabolized to propionate; FFA are degraded into acetate and hydrogen through ß-oxidation, after a saturation step in the case of unsaturated acids. Acetate and H_2 are then converted to methane by acetoclastic and hydrogenotrophic methanogens in the methanogenesis step.

The impact of milk fat in the anaerobic degradation process is complex and 2 major limitations have been reported: physical and chemical inhibition.

Physical Inhibition

Milk fat globules mostly contain triglycerides, the neutral structure of which limits their solubility in

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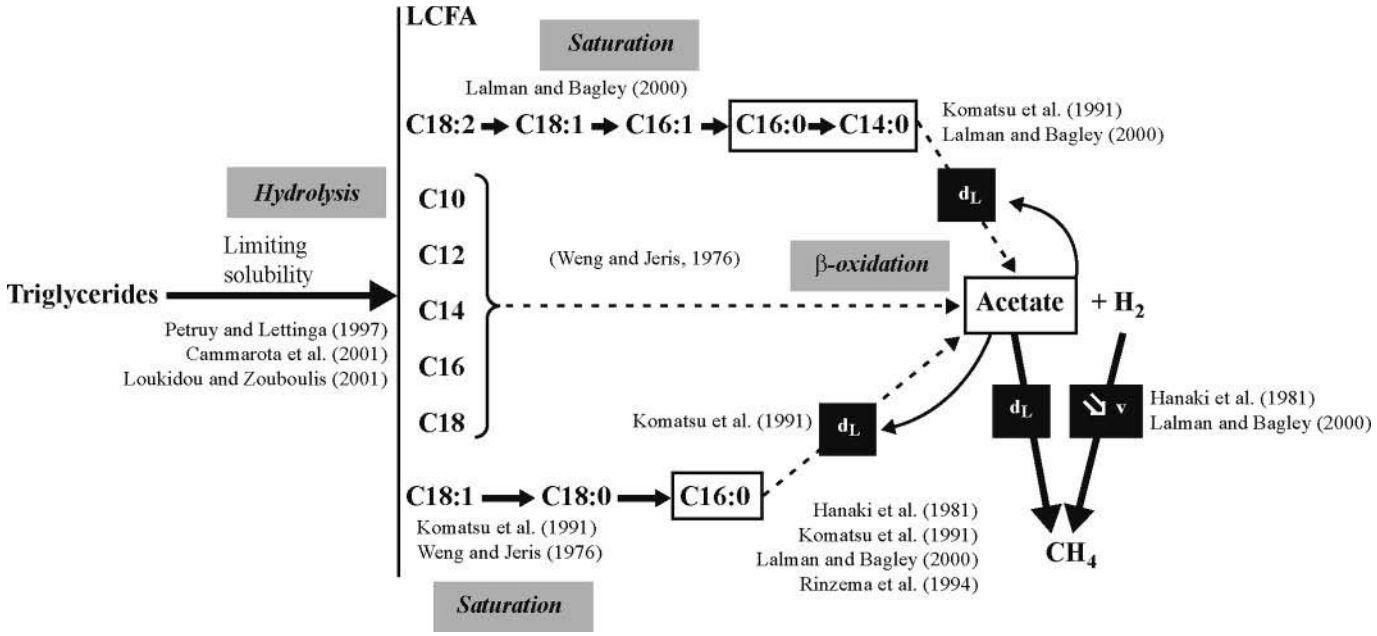

Figure 1. Limiting steps of anaerobic degradation of milk fat from triglyceride to methane. This figure is a synthesis of literature information available for each step of the degradation: hydrolysis of triglycerides; saturation of unsaturated fatty acids; successive ß-oxidations of saturated fatty acids (C10 to C18) and corresponding inhibition due to the acetate produced; conversion of acetate to methane. Dotted lines indicate several reaction steps. White boxes = accumulation; black boxes = consequence of inhibitions; d_L = lag phase; ⇩v = decrease of conversion velocity; LCFA = long-chain fatty acids; C10 = capric acid; C12 = lauric acid; C14 = myristic acid; C16 = palmitic acid; C18 = stearic acid; C16:1 = palmitoleic acid; C18:1 = oleic acid; C18:2 = linoleic acid.

the aqueous phase (Petruy and Lettinga, 1997). This low solubility leeds to adsorption of fat into biomass, decantation difficulties (Vidal et al., 2000), low bioassimilability and low accessibility of other substrates to bacteria (Petruy and Lettinga, 1997). Hanaki et al. (1981) and Petruy and Lettinga (1997) showed, in batch mode, that the anaerobic digestion of a mixture of long chain fatty acid, LCFA (C10–C18, 0.35 kg_{LCFA}/kg_{VSS} [**VSS** (volatile suspended solids); 40% of saturated acids (weight basis), 60% of unsaturated acids] led actually to a 60% temporary aggregation of LCFA and then to a lag phase of 10 d before methane production.

Chemical Inhibition

Chemical inhibition can be due to the toxicity of a given number of fatty acids on anaerobic microorganisms. Hanaki et al. (1981) showed that FFA (C10 to C18, 40% saturated, 60% unsaturated) inhibit H_2-producing bacteria responsible for ß-oxidation on one hand, and acetoclastic (acetate → CH_4) and hydrogenotrophic (H_2 → CH_4) methanogens on the other hand. Furthermore, the inhibition of hydrogenotrophic archaea leads to a reduction of the rate of hydrogen conversion into methane, which is correlated to FFA concentration (Lalman and Bagley, 2000). The inhibition of acetogens and acetoclastic methanogens leeds to a pronounced lag phase. The inhibition intensity varies with FFA nature (chain length and number of C=C double bond; Kim et al., 2004). Koster and Cramer (1987) showed that LCFA (C10, C12, C14, C18:1) have a variable toxicity power onto methanogens: C12 and C18:1 (30% of milk fatty acids) are the more toxic acid and the presence of sub-toxic concentration of C10 enhances C12 and C14 toxicity. Hanaki et al. (1981) observed a similar synergic effect with C18:1 and a LCFA mixture (C10 to C18, 40% saturated, 60% unsaturated). Komatsu et al. (1991) also reported that the lag phase length before biogas production from a given chain length FFA increases with the number of its double bonds (C18:0, C18:1, C18:2).

The exact mechanisms implied in the inhibitions described are complex (Figure 1) and not yet fully elucidated. It seems as if inhibitions occur in the very early steps of LCFA degradation, which vary according to the LCFA nature. In the case of milk fat, inhibitions observed during anaerobic degradation seem to be mainly due to the presence of unsaturated fatty acids. Indeed, C18:1 (30% in mass) and C18:2 (3%) were reported to cause a significant lag phase before acidification and methanogenesis, which is not the case with C18:0 (14%), C16:0 (27%), and C:14:0 (11%; Komatsu