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Comparison of existing models to simulate anaerobic digestion of lipid-rich waste

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HIGHLIGHTS

- ADM1 cannot simulate anaerobic digestion of lipid-rich waste.
- LCFA inhibition has to be considered to simulate substrates and acids accumulation.
- Acetogenesis and methanogenesis are most sensitive to LCFA inhibition.
- Models fail to simulate pH when approaching 6.5–6.0.

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ABSTRACT

Models for anaerobic digestion of lipid-rich waste taking inhibition into account were reviewed and, if necessary, adjusted to the ADM1 model framework in order to compare them. Experimental data from anaerobic digestion of slaughterhouse waste at an organic loading rate (OLR) ranging from 0.3 to 1.9 kgVS m^{-3} d^{-1} were used to compare and evaluate models. Experimental data obtained at low OLRs were accurately modeled whatever the model thereby validating the stoichiometric parameters used and influent fractionation. However, at higher OLRs, although inhibition parameters were optimized to reduce differences between experimental and simulated data, no model was able to accurately simulate accumulation of substrates and intermediates, mainly due to the wrong simulation of pH. A simulation using pH based on experimental data showed that acetogenesis and methanogenesis were the most sensitive steps to LCFA inhibition and enabled identification of the inhibition parameters of both steps.

1. Introduction

Anaerobic digestion (AD) is increasingly used for the treatment of organic wastes not only due to the energy potential of the process but also to the simultaneous reduction in greenhouse gas (GHG) emissions, particularly when used for livestock manures (Pellerin et al., 2013). In addition to the advantage of using manure due to GHG abatement, manure provides nutrients required for biological processes and also a buffer capacity that facilitates management of the process. However, the energy yield of manures in relation to their volume is too low for them to be processed alone. Consequently, co-substrates with a potentially higher energy yield are added to make the process cost effective. Wastes from agro-industries are often used as co-substrates for livestock manures, particularly lipid rich wastes from slaughterhouses, food processing industries and food distribution. Such wastes are very attractive co-substrates thanks to their high energy potential, and co-digestion with manure generally improves biogas production with no major increase in cost. However, when applied in large amounts, lipid rich wastes are also well known inhibitors of anaerobic digestion. According to the literature (Hwu et al., 1998), inhibition is mainly due to the fast step of hydrolysis of lipids to long-chain fatty acids (LCFA) performed by extracellular lipases excreted by hydrolytic bacteria and further adsorption of LCFA onto the microbial surfaces, resulting in biochemical and physical inhibition. Firstly, adsorbed LCFA can act as a detergent on microbial surfaces and damage the membrane. Secondly, adsorbed LCFA can limit the transport of the substrate from the culture medium into the cell. Thus, excessive lipid rich wastes greatly reduce biogas production whereas the optimum amount improves it.

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Due to the advantages of using such wastes but also the difficulties involved, many studies have dealt with the co-digestion of manure or sludge with lipid rich wastes (Zhang et al., 2013; Silvestre et al., 2014; Noutsopoulos et al., 2013; Neumann et al., 2015; Astals et al., 2015, 2013; Gunay and Karadag, 2015; Rasit et al., 2015; Fierro et al., 2014; Pitk et al., 2014; Zhang and Banks, 2012) in order to identify the optimum quantity of lipid rich waste to add in the process. However, the results of each work are very specific to the substrates and conditions of the study, and a more generic approach to understanding and controlling the process is needed.

Modelling is a very powerful tool for such a generic approach, as it enables a good understanding of both the process and control. However, only a few papers among the many published on co-digestion were devoted to modelling (Mata-Alvarez et al., 2014). Some were published before and after the development and publication of the Anaerobic Digestion Model N°1 (Batstone et al., 2002) but the number of publications on modelling is very low compared to published experimental studies on the same topic.

The anaerobic digestion model n°1 (ADM1) published in 2002 does not account for either inhibition due to lipids or LCFA or the concentration of LCFA for the calculation of pH, meaning it is not really useful for modelling anaerobic digestion of lipid rich waste, as already mentioned in Batstone et al. (2002). Before the publication of ADM1, Angelidaki et al. (1999) published a model taking LCFA inhibition into account. In addition to the existing ADM1, this model described LCFA inhibitions and also VFA inhibition of hydrolysis. Other models of anaerobic digestion of lipid rich waste were published by Salminen et al. (2000) and Lokshina et al. (2003) before the publication of ADM1. The model was based on a previous model called <METHANE> (Vavilin et al., 1994). The authors considered LCFA inhibitions and VFA inhibition of hydrolysis. More recently, Palatsi et al. (2010) proposed two different models compatible with the ADM1 framework that take the lipid and LCFA inhibition into account during anaerobic digestion. A first model, inspired by the work of Angelidaki et al. (1999), included LCFA inhibition. A second model (Palatsi_MOD2) was proposed to account for the physical adsorption of LCFA onto the surface of biomass as the main inhibitory process. More recently, another model was published by the same researchers (Zonta et al., 2013) that differentiates LCFA between C16 and C18 and takes the physical processes of adsorption and desorption of biomass into account. As the conditions and data required are so different from ADM1 and this works, it is not included here. Ma et al. (2015) published a model that considers LCFA inhibition in the context of anaerobic digestion of algal biomass. This model was not developed in the ADM1 framework and dealt only with hydrolysis, acidogenesis and methanogenesis. LCFA inhibition was considered.

No comparison of the models has been made since the ADM1 model was developed. In addition, few calibrations and validations have been performed using data from continuous or semi-continuous processes. Consequently, after being adjusted to fit the ADM1 framework if necessary, the models were used to model continuous processes and the results were compared with experimental data to evaluate them and enable their calibration.

2. Materials and methods

2.1. Experimental data

Results of previously published experiments corresponding to mesophilic anaerobic digestion of slaughterhouse waste (Rodriguez-Mendez, 2015) were used for this work. Four slaughterhouse wastes (SW) consisting of different dilutions of a mixture of blood and viscera were used. The main characteristics of these SW are listed in Table 2 and more details can be found in Rodriguez-Mendez (2015).

Five AD trials (T1 to T5) were carried out using these SW and hydraulic residence time (HRT) ranging from 20 to 50 days, and an organic loading (OLR) rate ranging from 0.3 to 1.9 kgVS m^{-3} d^{-1}, as described in Table 3. All the trials lasted longer than three hydraulic residence times to reach steady state. The characteristics of the effluents were analyzed throughout the duration of the trials particularly during steady-state conditions. The results are presented in Rodriguez-Mendez (2015).

At the start of each experiment, sludge from a digester treating primary and secondary sludge from Valcartier wastewater treatment plant (Québec, Canada) was collected and used to fill the digester. All the experiments were performed at bench scale in continuous CSTR with 11 L/3 L liquid/gas.

2.2. Description of the models

Several models were tested in this study for a better understanding of the anaerobic digestion of lipid rich waste. As previously mentioned, ADM1 is not really useful for modelling anaerobic digestion of lipid-rich waste so it was modified to take the LCFA into account in the calculation of pH. This slightly modified version of ADM1 is called ADM1_pHLCFA in the remainder of the paper. All the other processes were similar to the version proposed by Batstone et al. (2002) and Rosen and Jeppsson (2006). In addition to ADM1_pHLCFA, all models presented in the introduction were evaluated during this work. The model published by Angelidaki et al. (1999) was adjusted to fit the ADM1 framework (Table 1) and is called Angelidaki_MOD in the remainder of the paper. In addition to the existing ADM1, the Angelidaki_MOD described non-competitive LCFA inhibition of the (i) acidogenic glucose degrading step, (ii) lipolytic step, (iii) VFA-degrading acetogenic step and (iv) acetoclastic methanogenic processes and a Haldane LCFA inhibition of LCFA-degrading acetogenic step. Also in addition to ADM1, Angelidaki_MOD described non-competitive total VFA inhibition of carbohydrate and protein hydrolysis. For LCFA inhibition, only one constant (Ki,lcfa) was used for both competitive and Haldane inhibitions. The model published by Salminen et al. (2000) and Lokshina et al. (2003) is called Salminen_MOD in the remainder of this paper. This model was then adapted to the ADM1 framework as described in Table 1. The authors considered (i) non-competitive LCFA inhibition of acetogenesis of other VFA than acetate and of acetoclastic methanogenesis and (ii) a non-competitive VFA other than acetate inhibition of hydrolysis. The first model proposed by Palatsi et al. (2010), called Palatsi_MOD1 in the remainder of this paper, included LCFA inhibition of LCFA and acetate uptake but also of hydrogen uptake (Table 1). A second model (Palatsi_MOD2) was proposed to account for the physical adsorption of LCFA onto the surface of biomass as the main inhibitory process. To this end, the authors replaced the inhibitory constant (K_I) by a new inhibitory term including the ratio of the biomass to LCFA ($K_I \cdot X_{lcfa}/S_{lcfa}$). The model published by Ma et al. (2015) was not developed in the ADM1 framework and dealt only with hydrolysis, acidogenesis and methanogenesis. Non-competitive LCFA inhibition was considered and a specific inhibition constant was calibrated for each step. Even if the acetogenesis step was not directly tackled in this work, the inhibition of the LCFA uptake step was. The model was adapted to fit the ADM1 framework and is described in Table 1. This model is called Ma_MOD in the remainder of the paper. For the inhibition process, like in Palatsi_MOD2, the ratio of active biomass to LCFA was used considering that LCFA inhibition is mainly due to adsorption onto the surface of the biomass. For each model, any processes not listed in Table 1 were similar to those in ADM1_pHLCFA.