appetite regulation that favor the development of obesity. Dietary fat usually implies an increase in energy consumption because it has a lower potential for inducing satiety than carbohydrates and protein. Hence, high-fat diets may play an important role in the increased prevalence of obesity and can be a triggering factor in the development of hyperglycaemia and hyperinsulinaemia (Jager et al. 2009). Moreover, the intake of dietary fats is usually accompanied by a higher intake of refined sweet carbohydrates (fast food and desserts), where the high intake of sucrose promotes weight gain, visceral adiposity and the development of diseases that are related to obesity, such as cardiovascular diseases (Stevenson et al. 2009). Therefore, low-fat diets often are prescribed in the prevention and treatment of obesity because a reduction in dietary lipids without restriction of total energy intake could cause weight loss.

Recent studies indicate that fat digestion is a prerequisite for the effects of fat on gastric emptying, gastrointestinal hormone secretion, appetite and energy intake. An increasing number of gastrointestinal enzymes involved in nutrient digestion are being identified and characterized, representing a rich pool of potential therapeutic targets for obesity and other metabolic disorders (Birari and Bhutani 2007). Enzymes that are especially related to dietary fat are interesting targets, which include preduodenal lipases (lingual and gastric lipases), pancreatic lipases, cholesterol-ester lipases and bile-salt stimulated lipases (Armand 2007). Most dietary fat is ingested as triglycerides (90–95%), and their hydrolysis starts in the mouth, then goes on through the stomach by an acid stable gastric lipase, and continues in the duodenum through the synergistic actions of gastric and colipase-dependent pancreatic lipases, leading to the formation of monoglycerides and free fatty acids (Figure 7.2). Free fatty acids are absorbed by the enterocyte to synthesize new triglyceride molecules, which are transported to the different organs via lipoproteins, especially chylomicrons, after a meal.

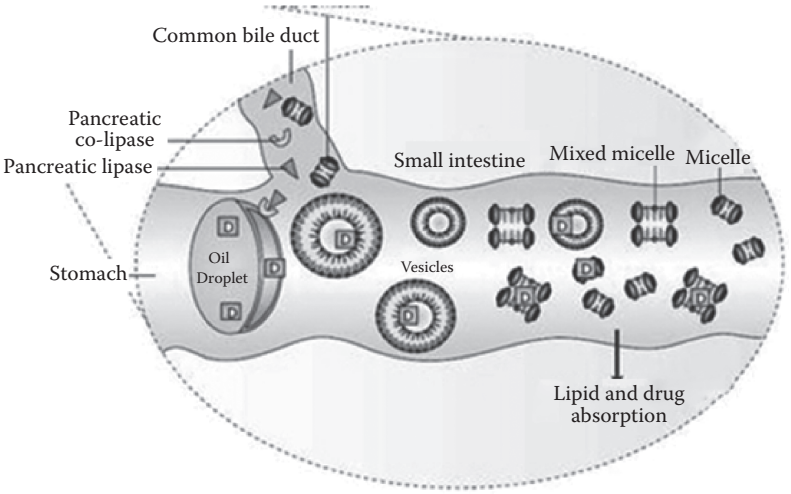

FIGURE 7.2 Role of pancreatic lipasema in fat digestion.