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# Chlordecone disappearance in tissues of growing goats after a one month decontamination period—effect of body fatness on chlordecone retention

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**Abstract** Chlordecone (CLD) is an organochlorine pesticide whose extended use led to the contamination of at least 20 % of agricultural soils from the French West Indies. Livestock reared on polluted areas are involuntary contaminated by CLD and their level of contamination may exceed the threshold values set by the European Union. Thus, characterizing the CLD behaviour in farm animals appear as a real issue in terms of food safety for local populations. The aim of this experiment was (i) to characterize the CLD disappearance in various tissues after exposure cessation and (ii) to evaluate the potential effect of body fatness on this process. Two groups of eight growing goats were submitted to either a basal diet or a high energy diet for 50 days before being intravenously contaminated with 1 mg CLD $kg^{-1}$ body weight. Two days after CLD contamination, half of the kids of each experimental group were slaughtered in order to determine pollutant levels in the serum, liver, adipose tissues, and empty carcass. The remaining animals were submitted to a 30-day decontamination period before slaughtering and measurements as described above. The implemented nutritional plan resulted in both groups of kids with significant differences in terms of body fatness. CLD was mainly concentrated in the liver of animals as described in the literature. It was found also in kids' empty carcass and adipose tissues; however its levels in the empty carcass (muscles and bones) were unexpected since they were higher than in fat. These results indicate that the lipophilic pollutant CLD is found mainly in liver but also in muscles and fat. Concerning the animals' depuration, a 30-d decontamination period was sufficient to observe a decrease of CLD levels by more than 75 % in both experimental groups and neither CLD concentrations nor CLD amounts were significantly affected by kids' body fatness.

**Keywords** Chlordecone · Growing goat · Decontamination · Fatness

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**Abbreviations**

| | |
|---|---|
| AT | Adipose tissues |
| BD | Basal diet |
| BW | Body weight |
| CLD | Chlordecone |
| CLD-OH | Chlordecone-alcohol |
| DM | Dry matter |
| FM | Fat matter |
| HED | High energy diet |
| LOQ | Limit of quantification. |

## Introduction

Chlordecone (CLD) is a chlorinated polycyclic ketone pesticide used from 1971 until 1993 in the French West Indies to fight against the banana black weevil (*Cosmopolites sordidus*). Its use resulted in an extended contamination of at least 20 % of agricultural soils (Le Deaut and Procaccia, 2009). Local water and food resources in polluted areas,

including fish, crustaceans, root vegetables and terrestrial animal products (meat, milk, and eggs) have been contaminated by CLD because of (i) its high bioaccumulation potential in environment (Bocquené and Franco, 2005, Dubuisson et al. 2007, Coat et al. 2011) and (ii) its persistence in soils which is thought to last five to seven centuries for the heaviest polluted andosols (Cabidoche et al. 2009). Until now, the main research efforts were focused on human health, environmental fate of CLD, edible crops, and marine or freshwater organism contamination. National survey plans were carried out in the French West Indies slaughterhouses in order to assess the CLD prevalence in ruminants originating from contaminated areas and to remove non regulatory carcasses from human consumption. In the European regulation (Commission Regulation (EC) n°839/2008), fat is considered as the reference matrix and for practical reasons peri-renal fat is used for CLD determination. The maximum residue limits (MRL) has been fixed at 100 μg CLD $kg^{-1}$ fat. The contamination of local ruminants is currently observed and for example, in 2011, the CLD concentrations in peri-renal adipose tissues of 12 % of slaughtered animals in Guadeloupe were found to be higher than the expected MRL. Backyard animal productions (pigs, small ruminants, and poultry) were not taken into account in the national survey plans while they could also represent an important source of CLD contamination for the local population. Thus, Martinique and Guadeloupe populations remain exposed to this pesticide through the consumption of contaminated food. There is an increasing demand for solutions that would enable to ensure safe animal products all the more that CLD is considered as a carcinogenic, mutagenic, and reprotoxic compound for all living beings including humans (Dallaire et al. 2012, Multigner et al. 2010). Since it remains difficult to avoid exposure of ruminants reared in contaminated areas especially in this island context, CLD behaviour in the organism of target species and its rate of decontamination must be studied in order to better predict the risk of contamination and to assess the time required for its remediation. It has to be recalled that CLD ingested by grazing ruminants is not longer sequestered by soil during the digestive process and therefore this compound will be strongly absorbed (Jurjanz et al. 2014).

CLD decontamination kinetics were previously described in laying hens, Muscovy ducks (Jondreville et al. 2014a,b) and dairy cows (Smith and Arant, 1967). For these animal species, it is assumed that the fast CLD removal (half-lives of less than 3 weeks) is due to the important excretion routes through egg and milk (Naber and Ware, 1964, Smith and Arant, 1967, Jondreville et al. 2014a, b) and to metabolism. No information is currently available concerning CLD behaviour in non lactating ruminant species. After cessation of exposure, would CLD disappear in a same way from the various tissues? Would CLD disappearance be influenced by the body fatness? Indeed, knowing that CLD is a lipophilic contaminant, different body fatness could originate a different distribution of CLD among tissues and organs as well as different pollutant retention within animals' organs. Then, it may be assumed that the decontamination kinetic of animals may be influenced, also, by this body fatness. Such interaction has already been observed in a previous study in which a stronger retention of the lipophilic pollutant "pentachlorobenzene" was highlighted in fat animals compared to restricted-feeding ones (Umegaki et al. 1993). Therefore, a simultaneous determination of CLD in various tissues and organs as the serum, fat, and muscles should allow, for the first time, to get a real picture of the CLD distribution and disappearance in the growing goat. Thus, the objectives of the current study were to characterize, after a 30-d decontamination period, CLD disappearance in the serum, the peri-renal adipose tissues, the subcutaneous adipose tissues, the liver and the empty carcass of growing ruminants with two different levels of body fatness.

## Materials and methods

The experimental protocol was approved by the Lorraine Ethics Committee (CELMEA) and the French Ministry of Higher Education and Research under the project number 00270.02.

### Animals and experimental design

Sixteen Alpine male kids (*Capra hircus*), born in the experimental farm of Bouzule (Laneuvelotte, France) were used. Once weaned (9-week-old±1 week), they were allocated to two groups of eight kids each according to their body weight (BW) (group 1: 18.25±2.82 kg, group 2: 18.13±2.30 kg (mean±standard deviation)). Each experimental group ($n$=8) was kept in a box covered with straw and equipped with individual feeders of complete feed. The experiment consisted in a 50-day rearing period followed by a 9-day contamination period and a 30-day decontamination period. All animals were individually weighed every 7 days.

### Experimental diets

During the entire experimental period (90 days), the kids were submitted to two experimental diets with continuous access to fresh water and mineral blocks. The first diet was identified as the basal diet "BD" and was offered to kids from group 1. It was characterized by a standard level of energy and was composed of a complete feed (6.69 MJ of net energy $kg^{-1}$ of dry matter; Fluvia Junior, SANDERS®, Einville au Jard, France) completed by first-cut hay. The second diet was identified as the high energy diet "HED" and was offered to kids from group 2. This diet was based on BD supplemented with corn (8.40 MJ of net energy $kg^{-1}$ of dry matter) in order to (i)

provide the animals with a higher daily energy intake (+66 %) and to (ii) increase their fat deposition. First-cut hay was distributed ad libitum while complete feed and corn were distributed according to a nutritional plan based on kids' BW (BD animals: 20 g complete feed DM kg $BW^{-1}$ $day^{-1}$; HED animals: 20 g complete feed DM kg $BW^{-1}$ $day^{-1}$ and 13 g corn kg $BW^{-1}$ $d^{-1}$) and were offered daily in two equal meals.

### CLD contamination

After the first 50 days of the experiment, each kid was intravenously administered a total dose of 1 mg CLD $kg^{-1}$ BW in three successive injections (days 50, 54, and 58). The administration of successive high doses of CLD was chosen (i) to expose all animals in a same manner (quantities, duration of exposure) and (ii) to ensure a high level of CLD impregnation for all goats' tissues and organs. CLD powder (Kepone®, ref.49046, 99.9 % purity, Sigma Aldrich®, France) was dissolved in a solubilizer and emulsifying agent (Cremophor® EL, ref.C5135, pH-range 6.0–8.0, Sigma Aldrich®, France) by a magnetic stirrer at 20 °C for 2 h followed by a 1-h sonication at 20 °C. The contaminated solution was concentrated to 10 mg CLD $g^{-1}$ Cremophor® and was infused into the 16 kids' jugular veins through a catheter thereafter rinsed with a physiological saline solution (NaCl solution, 0.9 %, Lavoisier, France).

### Sampling, measurements and chemical analysis

*Blood samplings and CLD analysis in serum*

Before slaughtering (days 60 and 90), blood samples of 30 mL were collected in all kids by jugular venipuncture into serum collection tubes in order to establish levels of CLD at the beginning and at the end of the decontamination period. All blood samples were allowed to clot for 2 h at room temperature and maintained at 4 °C for 24 h before the serum was separated by centrifugation at 1278*g* for 15 min. The obtained serum was stored in glass vials at −20 °C before analysis. Determination of CLD in serum was performed by Gas Chromatography Electron Capture Detector (GC-ECD) by the Center for Analytical Research and Technology at Liege University (CART, Belgium) according to Multigner et al. (2010). Limit of quantification (LOQ) was 0.06 μg $g^{-1}$ in the serum.

*Tissues, organs, and CLD analysis*

At day 60, each kid was weighed and four animals of each experimental group were selected according to their BW and slaughtered (electronarcosis followed by exsanguination). The whole liver, the peri-renal AT, and a part of the sternal subcutaneous AT were collected, weighed, and stored at −20 °C before freeze-drying and analysis. Thereafter, the digestive content was separated from the digestive tract, the body hairs were shorn, and the horns were cut in order to determine the empty body mass (defined as total body mass minus liver, peri-renal adipose tissue, digestive contents, hairs, and horns). Empty body was cut in five pieces and stored at −20 °C in plastic bags. The content of the bags was ground using industrial grinder and mixer at the PEGASE research unit of INRA (Rennes, France). A homogenized 1-kg aliquot was obtained for analyses. Dry matter was determined for each individual matrix after freeze-drying, before being ground. Final samples (liver, peri-renal and subcutaneous AT, and empty carcass) were analysed for dry matter (DM) by desiccation (103 °C, 48 h) and for ether extracted fat according to Folch et al. (1957). Dosages of CLD were also performed on these matrices by High-Performance Liquid Chromatography-Mass Spectrometry (HPLC-MS) according to the method described by Bordet et al. (2007), in the Departmental Analytical Laboratory of Morbihan (LDA 56, Saint-Ave, France). Limit of quantification was 2.0 μg CLD $kg^{-1}$ in these matrices. The four remaining animals of each experimental group were reared with the same nutritional plan (BD versus HED) for an additional 30-day period and the morning of day 90, these animals were slaughtered in the same conditions than the first goats. The following steps performed were similar to those described previously for the first eight kids.

### Calculations and statistical analyses

Statistical analyses were performed by means of the Statistical Analysis Systems software package (SAS, version 9.3, SAS Institute, Cary, NC). In order to compare body composition of experimental groups (BD versus HED) at the beginning and at the end of the decontamination period (day 60 versus d 90), data of body composition were analyzed as repeated measures using MIXED procedure. Thereafter, CLD concentrations and quantities in the studied matrices were analyzed as independent measures using GLM procedure. The kid was considered as the experimental unit and for each studied matrix (or element of the body composition), the model included the dates of sampling (days 60 and 90), the treatment (BD, HED) and their interaction as main effects. A Tukey–Kramer test was used for comparison of means. Differences were considered significant for $P$-values$<0.05$.

## Results and discussion

### Effects of the diet on the growth parameters and the adipose status of kids

Over the first 60 days of the experiment, the average daily amounts of ingested BD and HED were respectively of 424

±37 g $d^{-1}$ complete feed (mean±standard error) and 441± 24 g $d^{-1}$ complete feed supplemented by 287±17 g $d^{-1}$ corn. During the following 30 days of decontamination, these values were respectively 499±33 g $d^{-1}$ complete feed for BD and 551±26 g $d^{-1}$ complete feed supplemented with 367±18 g $d^{-1}$ corn for HED. No feed refusal was recorded during the experiment. For each experimental period mentioned above, a significant weight gain was observed in all animals. The mean weight gain between the start (day 0) and the end of the experiment (day 90) was+9 kg for BD animals and+15 kg for HED animals (Fig. 1). Thus, CLD contamination did not affect the kids' development and the observed levels of growth correspond to usual figures for such kind of animal species (Urge et al. 2004). Indeed, the CLD dose used in this experiment (1 mg CLD $kg^{-1}$ BW) was much lower than the doses for which clinical signs of toxicity were previously observed in livestock: up to 80 mg CLD $kg^{-1}$ in growing pigs (Soine et al. 1983) and from 150 mg CLD $kg^{-1}$ food in poultry (Naber and Ware, 1964, McFarland and Lacy, 1969, Guzelian, 1982). For each experimental group, Table 1 details the average body composition of empty carcasses at slaughtering (days 60 and 90) in relation to diets and time. Both experimental diets (BD and HED,) resulted in animals with significant different anatomical measurements and body composition ($P<0.05$, Table 1) except for the weight of the digestive contents and the percentage of protein and ash ($P>0.05$, Table 1). The amounts of lipids and proteins of animals from HED (3.2 and 4.1 kg, respectively) were significantly higher than those of BD (2.2 and 3.4 kg, Table 1). The effects of "Time" were considered minor when compared to the effects of diets (Table 1). Finally, the nutritional plan (BD versus HED) applied in this study resulted in (i) a significant increase in the mean body weight of the animals between d 0 and d 90 ($P<0.05$, Fig. 1), (ii) a final body weight difference of nearly 6 kg between the two groups of kids (Fig. 1, Table 1) and (iii) a significant difference in the body fatness of the animals from both groups ($P<0.05$, Table 1).

## Chlordecone distribution

Tables 2 and 3 indicate the CLD levels in kids' organism at days 60 and 90. Both tables clearly show and emphasize the heterogeneous CLD distribution. Liver represents an important matrix of CLD storage (Tables 2, 3) as already mentioned in the literature (IPCS, 1984, Bouveret et al. 2013, Jondreville et al. 2014a, b, Jurjanz et al. 2014). For each experimental group, CLD levels in adipose tissues were lower than the CLD levels in the empty carcass (Tables 2, 3). These latter results in the empty carcass were unexpected for a lipophilic pollutant and suggest that CLD is not only distributed to the liver and the fat but also to muscles. Recently, Jondreville et al. (2014b) had already observed, in Muscovy ducks, a ten times higher CLD concentration in leg without skin than in abdominal fat. The following hypotheses may explain the specific CLD distribution in kids (liver, muscle, and fat). First, it is known in humans, rats, and pigs that CLD distribution depends on the affinity of pollutant to blood components. Indeed, several studies suggested a high affinity of CLD to albumin and high-density lipoprotein (HDL) (Skalsky et al. 1979, Guzelian, 1982, Soine et al. 1983). Albumin is an important protein which constitutes more than 40 % of the serum total protein concentration in goats (Alberghina et al. 2010) and HDL represent 80 % of lipoproteins carrying massively lipids throughout ruminants' body (Mills and Taylaur, 1971, Vitić and Stevanović 1993, Bauchart, 1993, Hocquette and Bauchart, 1999). Knowing that (i) the liver has many receptors for albumin and (ii) the blood flows throughout the body, the links "CLD-albumin" and "CLD-HDL" may explain the high concentration of this pollutant in the liver and the empty carcass of animals. CLD distribution in adipose tissues (AT) may be linked to the development of these tissues within the organism and this pollutant would be able to successively concentrate in intra-muscular, inter-muscular, visceral, and subcutaneous fat tissues according to the growth steps of the animals. CLD distribution may also depend on the different lipid classes which compose AT. Assuming that (i) CLD has a high affinity for HDL, (ii) HDL are richer in cholesterol and phospholipids than in triglycerides (Mills and Taylaur, 1971,

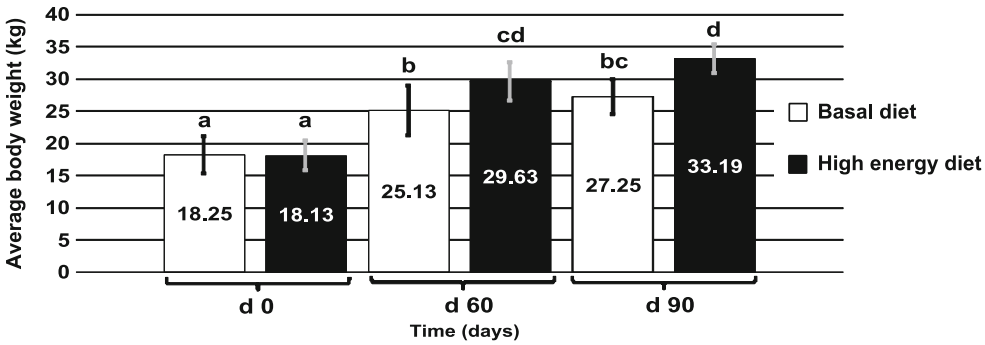


**Fig. 1** Evolution of the average weight of the kids during the experimental period

Hocquette and Bauchart, 1999) and (iii) each AT has a specific composition (Bas et al. 1985, Morand-Fehr et al. 1985), CLD would have a higher affinity for polar lipids composing cell membranes which are abundant in liver (e.g., phospholipids and free cholesterol) than for non-polar lipids like triglycerides which is one of the components of fat tissues. Thus, the results of these studies suggest a specific behaviour of CLD which would need to be thoroughly investigated, especially the pathways followed by CLD to concentrate in tissues and organs.

## CLD concentrations in the studied matrices at days 60 and 90

Table 2 indicates the CLD concentrations at days 60 and 90 and for each time point the levels obtained according to the implemented processing. At day 60, CLD concentrations were similar in the empty carcass or in the serum of BD and HED animals whereas a significant difference was found in both adipose tissues of these animals. Indeed, CLD levels in adipose tissues of fatty animals (HED) were significantly higher than in animals from BD group ($P<0.05$, Table 2). These results are not surprising since the lipid amounts in HED animals were found to be quantitatively higher than in BD animals (3.2 and 2.2 kg, respectively, Table 1) and it can be assumed that the adipose tissues were potentially more developed (adipocyte volume, metabolic activity) in HED animals compared to BD animals. Furthermore, previous observations have shown a positive correlation between the concentration of some lipophilic organochlorine pollutants and the fat tissue mass or the body weight of animals (Rozman et al. 1983, Nakashima and Ikegami, 2003). At day 90, there were no significant differences between the CLD concentrations in both experimental groups whatever the biological matrix. It may be noted that CLD concentration at day 90 in fat tissues and in serum was close to the LOQ (2 μg CLD $kg^{-1}$ in fat; 0.06 μg CLD $g^{-1}$ in serum). For each studied matrix, the CLD concentrations were found to be significantly lower at day 90 when compared to day 60 ($P<0.05$, Table 2). Thus, over the experimental period, the levels decreased by more than 75 % in all studied matrices. This fast and important disappearance of CLD from the organism reflects either (i) a CLD dilution, (ii) its excretion from the organism or (iii) its potential biotransformation in CLD metabolites, as for example chlordecone-alcohol (CLD-OH). To our knowledge, only one study carried out in pigs indicated the presence of CLD-OH in livestock (Soine et al. 1983). Overall, for similar ages and exposure conditions, the high energy diet used in this study did not really modulate the decontamination process in kids. From a global point of view, the rates of CLD disappearance observed in all studied matrices and in all animals were in a same order of magnitude then those found in other species. A decrease up to 80 % of the CLD concentrations was, for example, observed in the milk of dairy cows decontaminated during 40 days (Smith and Arant, 1967). In Muscovy ducks placed in confinement for 6 weeks,

**Table 1** Anatomical measurements and body composition of goats receiving diets supplemented or not with corn and slaughtered at the beginning (day 60) and the end (day 90) of the decontamination period

| | Diet | | Time (days) | | Significance | | |
|---|---|---|---|---|---|---|---|
| | BD | HED | 60 | 90 | Diet | Time | SEM |
| Anatomical measurements at slaughter (kg) | | | | | | | |
| Body weight | 26.6 | 31.4 | 27.7 | 30.2 | ** | NS | 1.01 |
| Digestive contents (DC) | 7.1 | 7.1 | 6.6 | 7.6 | NS | NS | 0.38 |
| Empty carcass | 19.3 | 24.1 | 20.9 | 22.4 | *** | NS | 0.71 |
| Chemical composition (kg) | | | | | | | |
| Total water | 19.2 | 21.8 | 19.8 | 21.2 | * | NS | 0.69 |
| Water of empty carcass | 13.1 | 15.8 | 14.2 | 14.8 | *** | NS | 0.46 |
| Lipid | 2.2 | 3.2 | 2.4 | 2.9 | ** | NS | 0.19 |
| Protein | 3.4 | 4.1 | 3.6 | 3.9 | *** | NS | 0.12 |
| Ash | 0.7 | 0.9 | 0.8 | 0.9 | ** | NS | 0.03 |
| Energy (MJ) | 163.2 | 217.6 | 175.7 | 205.0 | *** | NS | 2.2 |
| Proportions of empty carcass (%) | | | | | | | |
| Water | 68.0 | 65.9 | 67.9 | 66 | * | * | 0.54 |
| Lipid | 11.1 | 13.1 | 11.3 | 12.8 | * | NS | 0.63 |
| Protein | 17.5 | 17.2 | 17.3 | 17.4 | NS | NS | 0.13 |
| Ash | 3.8 | 3.8 | 3.8 | 3.9 | NS | NS | 0.09 |

Values are presented as means ($n=8$). Differences between Basal Diet (*BD*) and High Energy Diet (*HED*) groups according to the parameter "*diet*" and differences between kids according to the parameter "*time*" are noticed as follows: no significant (*NS*) for $P\geq0.05$; *for $P<0.05$; **for $P<0.01$; ***for $P<0.001$

**Table 2** Chlordecone (CLD) concentrations in empty carcass, liver, peri-renal and subcutaneous adipose tissues and serum of both experimental groups at the beginning (day 60) and the end (day 90) of the decontamination period

| | Time (days) | | | | | | Significance | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Day 60 | | Day 90 | | Day 60–90 | | | | | | |
| Matrices | BD | HED | BD | HED | BD (%) | HED (%) | Diet | Time | Diet x time | $R^2$ | SEM |
| Solid matrices (μg CLD $g^{-1}$ DM) | | | | | | | | | | | |
| Empty carcass | 1.58 (b) | 1.41 (b) | 0.20 (a) | 0.24 (a) | 87 | 83 | NS | ** | NS | 0.99 | 0.086 |
| Liver | 15.79 (b) | 15.13 (b) | 2.29 (a) | 2.71 (a) | 85 | 82 | NS | ** | NS | 0.99 | 1.309 |
| Peri-renal fat | 0.54 (b) | 0.67 (c) | 0.12 (a) | 0.14 (a) | 77 | 79 | * | ** | * | 0.99 | 0.039 |
| Subcutaneous fat | 0.51 (b) | 0.78 (c) | 0.08 (a) | 0.10 (a) | 85 | 87 | * | ** | * | 0.98 | 0.074 |
| Other matrix (μg CLD.$g^{-1}$ serum) | | | | | | | | | | | |
| Serum | 0.84 (b) | 0.74 (b) | 0.15 (a) | 0.17 (a) | 82 | 77 | NS | ** | NS | 0.88 | 0.15 |

Values are presented as means ($n$=4). In each column, means with *different letters* are significantly different at the $P<0.05$ level. Differences between Basal Diet (*BD*) and High Energy Diet (*HED*) groups according to parameters "*diet*" and "*time*" are noticed as follows: no significant (*NS*) for $P\geq0.05$; *for $P<0.05$; **for $P<0.0001$. BD (%) and HED (%) correspond to the percentage of CLD disappeared for each experimental group between day 60 and day 90

a decrease up to 85 % was found in the liver, abdominal fat, and leg with or without skin (Jondreville et al. 2014b) and in laying hens, these values were about 94 % in egg, abdominal fat and serum after a 21-d depuration period (Jondreville et al. 2014a).

### CLD amounts in the studied matrices at days 60 and 90

Table 3 indicates the amounts of CLD recovered at days 60 and 90 in matrices from each experimental group and the percentages of CLD disappearance after the 30-d decontamination period. The subcutaneous adipose tissues do not appear in Table 3 because non-exhaustive samplings of these tissues could be realized (section 2.4.2). All amounts of CLD presented in Table 3 were found to be higher (although, mostly non significant) in HED than in BD. These data are related to the administered doses (chapter 2.3.) but also to the average weight of the studied matrices which were 1.1 to 2.1 times heavier in fatty animals (HED) than in standard animals (BD). At the whole organism level, average amounts of CLD recovered at days 60 and 90 were not significantly different between experimental groups ($P>0.05$, Table 3). The lack of differences observed between both groups of animals for a given time point is in line with the results presented in section 3.3. and tends to demonstrate that kids' body fatness does not modulate CLD decontamination processes. A highly significant time effect was found ($P<0.0001$, Table 3) and BD and HED animals lost respectively 85 and 81 % of CLD in 30 days

**Table 3** Chlordecone amounts (μg) in matrices of both experimental groups at the beginning (day 60) and the end (day 90) of the decontamination period

| | Time | | | | | | Significance | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Day 60 | | Day 90 | | Day 60–90 | | | | | | |
| Matrices | BD | HED | BD | HED | BD (%) | HED (%) | Diet | Time | Diet x days | $R^2$ | ETR |
| Total average amount of CLD in the whole organism (μg) | | | | | | | | | | | |
| [1]Total Amount | 10 615 (b) | 12 823 (b) | 1 561 (a) | 2 460 (a) | 85 | 81 | NS | *** | NS | 0.98 | 1201 |
| Average amount of CLD in each studied matrix (μg) | | | | | | | | | | | |
| Empty carcass | 8722 (b) | 10208 (b) | 1248 (a) | 1981 (a) | 86 | 81 | NS | *** | NS | 0.97 | 1060 |
| Liver | 1835 (b) | 2480 (c) | 295 (a) | 436 (a) | 84 | 82 | * | *** | * | 0.98 | 192 |
| Peri-renal fat | 58 (a) | 135 (b) | 18 (a) | 43 (a) | 69 | 68 | * | ** | NS | 0.90 | 26 |

Values are presented as means ($n$=4). In each column, means with different letters are significantly different at the $P<0.05$ level. Differences between Basal Diet (*BD*) and High Energy Diet (*HED*) groups according to parameters "diet" and "time" are noticed as follows: no significant (*NS*) for $P\geq0.05$; *for $P<0.05$; **for $P<0.01$; ***for $P<0.0001$. BD (%) and HED (%) correspond to the percentage of CLD disappeared for each experimental group between day 60 and day 90

[1] Sum of CLD amounts in empty carcass, liver and peri-renal fat

(Table 3). At the level of the studied matrices, at day 60, only amounts of CLD in peri-renal fat and liver appeared to be affected by diet ($P<0.05$, Table 3). Values in empty carcass were not significantly different between BD and HED animals ($P>0.05$, Table 3) and these data seemed to be explained by the fact that all kids ($n=16$) received the same CLD dose (1 mg CLD $kg^{-1}$ BW). Regarding the significant differences observed in peri-renal fat and liver, previous studies have demonstrated a correlation between metabolic rate and body size (Kleiber, 1947). Therefore, it can be assumed that the high CLD amount was promoted by their metabolic activity and that these phenomenons would be more important in HED than in BD. Indeed, a significant increased lipid deposition and liver size were observed in HED (Table 3) and it is recognized that internal visceral adipose tissues as peri-renal tissues have high lipid content and lipoprotein lipase activity (Bas et al. 1985, Morand-Fehr et al. 1985, Robelin and Casteilla, 1990). At day 90, there were no significant differences between groups and the time effect was strongly marked for all animals ($P<0.05$, Table 3). The obtained data demonstrate a significant decrease of CLD amounts between days 60 and 90. The average disappearance percentage of CLD in the empty carcass and in the liver ranged from 81 to 86 %, whereas it reached only 69 % in the peri-renal fat. These values which were in a same order of magnitude to those observed in the whole organism demonstrate a high disappearance of CLD from the organism. The pollutant was not diluted in adipose tissues of fatty animals (HED) and both experimental groups presented similar CLD levels at the end of the decontamination period. Thus, the CLD decrease observed for both concentrations (Table 2) and amounts (Table 3) suggest no effects of diets or lipogenesis on CLD behaviour. The CLD disappearance from kids' organism could be explained either by metabolism or excretion. It has to be recalled that CLD metabolites as CLD-OH were not analyzed in the current study.

## Conclusion

The nutritional plan implemented in this protocol resulted in two significant experimental groups of kids in terms of anatomical measurements and body composition. Animals, who received a high energy diet (HED), presented a 1.5 times higher lipid deposits than BD animals. At the beginning and the end of the decontamination period, the pollutant Chlordecone (CLD) in its native form was principally found in liver, empty carcass and, to a lesser degree, in peri-renal adipose tissues of growing kids. However, its high concentration and amounts in the empty carcass was unexpected and raises additional questions about the tissue distribution of this pollutant; in particular, because the empty carcass (without liver, blood and subcutaneous and peri-renal fat tissues) was mainly composed by muscles and bones. Thus, it appears that the muscle is also a major target tissue for CLD.

CLD levels were found to be very close between animals of both groups and for most all studied matrices, neither CLD concentrations nor CLD amounts were significantly affected by the diet. Furthermore, whatever the initial distribution of CLD in the organism, this compound was not found to be specifically retained in one or the other matrix. Indeed, after a 30-d decontamination period, all kids strongly eliminated CLD with a global disappearance of more than 75 % in the studied matrices. The current study showed a lack of modulation of CLD decontamination by goats' fatness but the cessation of CLD exposure appears to be an effective way to decontaminate young ruminants.

The results of this study are really promising since they suggest that sustainability of farming and rearing practices used in the French West Indies remains possible with the condition that the contaminated animals undergo a sufficient long period of decontamination which needs to further characterized. Indeed, an adequate period of decontamination needs to be adjusted to each specific kind of animals and their initial level of contamination. Concerning the behaviour of CLD in ruminants' organism, no CLD metabolites were analyzed in this study and further research need to be conducted to determine if the CLD levels decrease is related to the excretion of the CLD-parent or its biotransformation. Additional studies carried on (i) CLD affinity to muscles and (ii) receptors implemented during its cellular transport would allow, also, for better understanding of its metabolism and its distribution within organism.

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