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Acute Oral and Inhalation Toxicities in Rats With Cadmium Telluride

Joseph Zayed and Suzanne Philippe

Cadmium telluride (CdTe) thin film photovoltaic has become one of the leading technologies in the solar renewable energy market. Little is known about CdTe's toxicological profile and regulatory agencies usually apply cadmium (Cd) criteria as a best approximation. However, CdTe may have different toxicological properties. The goal of this study is to determine the median lethal concentration/dose of CdTe and to compare these values with those of Cd. Using a rat model, the method followed conforms to that described in the Organisation for Economic Co-operation and Development (OECD) guidelines and in the US Environmental Protection Agency (EPA) Health Effects Test Guidelines. The median lethal concentration of CdTe was established at 2.71 mg/L, showing a very low variability between genders. The median lethal dose was determined to be greater than 2000 mg/kg. These results clearly show that CdTe is less toxic than Cd.

Keywords: acute toxicity; cadmium telluride; inhalation exposure; oral exposure

Cadmium telluride (CdTe) is a semiconductor compound used for various applications, including photovoltaic (PV) cells, infrared and radiation detection, and electro-optical modulators. PV cells allow the conversion of a portion of the solar radiation into electricity and, therefore, provide a form of renewable energy. Several cells are connected on a photovoltaic solar module. Several modules are grouped to form a solar installation that can be put on a private rooftop, on a commercial or industrial building, or in a large free-field photovoltaic solar power plant.

CdTe thin-film PV is already an important cost-competitive PV electricity option and its international market should continue to grow significantly in the coming years, mainly due to the growing need for energy—particularly energy with significantly reduced greenhouse gas emissions—combined with the decline in easily recoverable oil and gas reserves and the increase of their cost. The market for PV is expected to grow from $220 million in 2006 to over $3 billion in 2013.[1] PV use produces no emissions, thus offsetting many potential environmental problems.

However, as mentioned in several studies, the principal environment, safety, and health issue for CdTe PV is the potential introduction of cadmium—a toxic heavy metal—into the environment.[2] Unfortunately, in the case of CdTe, we are confronted with the very limited specific toxicological data. In fact, until 1994, no toxicity studies were reported for the compound.[3]

Therefore, regulatory agencies usually apply Cd criteria which could be in some cases totally inappropriate.

One of the first studies published on the toxicity of CdTe examined the compound's acute pulmonary toxicity, as compared to that of 2 other compounds (copper gallium diselenide and copper indium diselenide) used in the PV industry,[4] while a follow-up study focused on pulmonary tissue absorption and distribution, and sub-chronic endpoints.[5] These studies, which administered CdTe intra-tracheally to simulate inhalation exposures at doses ranging

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from 12.5 to 100 mg/kg, showed inhibition of weight gain, possible kidney effects at the highest dose, lung inflammation (from mild to severe), pulmonary fibrosis, and mortality at the 2 highest doses.

In 1999, Fthenakis et al derived a Reference dose (RfD) of 0.0006 mg/kg/d for CdTe.[3] This RfD is similar to the 1 of 0.0005 mg/kg/d derived by EPA for cadmium oral exposure by means of drinking water. Due to the lack of confidence in the data used, an uncertainty factor of 10 000 and a modifying factor of 2 were used to derive the RfD for CdTe.

More recently, Zhang et al[6] conducted in vitro and in vivo toxicity tests using CdTe nanoparticles and found few signs of functional toxicity or clinical changes. After intravenous exposure in male rats (2 μmol/kg), motor activity was transiently reduced (2 hours after treatment) and then significantly increased at a later time point (24 hours after dosing).

The complete toxicological profile of CdTe remains difficult to establish due to the lack of specific data. The goal of this study is to determine the median lethal concentration and the median lethal dose of CdTe for the respiratory and the oral routes, and to compare these values with those of Cd. This is the first step of ongoing research aiming to establish the toxicity and the ecotoxicity of CdTe.

Methods

CdTe was supplied by 5N Plus Inc (Montreal, Canada) with a certificate of analysis attesting that the product was of very high purity (99.999%). The experimental study was performed by LAB Research in accordance with the Principles of Good Laboratory Practice.

Animals

Rats are the preferred species as historically they have been used for this type of study and they are specified by the appropriate regulatory authorities. They were obtained from Charles River (Europe) Laboratories Inc (TOXI-COOP KFT, 1103 Budapest, Hungary). After arrival, the animals' health was certified by the resident veterinarian and after an acclimatization period of 5 days the rats were accepted into this study. Before this acceptance the rats were randomized. At their inclusion, they were young adults, in the weight range of 224–299 g. Females were nulliparous and not pregnant. Each rat was identified by a unique number marked on the tail.

Husbandry

The rats were housed in groups of 3 (for the median lethal dose experiment) or 5 (for the median lethal concentration experiment) in solid-floor cages (Type III) with stainless steel mesh lids and softwood flake bedding. The environmental controls were set to achieve target values of 22° ± 3° C and 30–70% relative humidity. The animal room was ventilated at a rate of 8–12 air changes per hour and the lighting controlled to give 12 hours of continuous artificial light in each 24-hour period.

Diet and Water

Rats were provided with ssniff SM R/M-Z+H "Autoclavable Complete Feed for Rats and Mice – Breeding and Maintenance" (ssniff Spezialdiäten GmbH, D-59494 Soest, Germany) and tap water, as for human consumption, *ad libitum*. The diet and drinking water were routinely analyzed and are considered free from any contaminants that could reasonably be expected to affect the purpose or integrity of the study.

Necropsy

After exposure, rats were followed during a 14-day observation period to check morbidity/mortality, clinical signs, and body weight. At the end of this period, surviving rats were killed by exsanguination under anesthesia (intra-peritoneal injection of pentobarbital solution ('Euthasol 40%'; Lot No.: 07E29 8; Expiry 04-2009; Produced by AST Beheer B.V. Oudewater Netherlands [Produlab Pharma, Raamsdonksveer]) and gross necropsies were performed. All rats, including those that died during the study, were subject to a gross necropsy which included a detailed examination of the abdominal and thoracic cavities. Special attention was given to the respiratory tract for macroscopic signs of irritancy or local toxicity.

Acute Inhalation Toxicity

The method used followed that described in the US Environmental Protection Agency (EPA) Health

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Effects Test Guidelines, OPPTS 870.1300, Acute Inhalation Toxicity, August 1998. The method was also designed to meet OECD guideline 403 (May 1981) and EC Guideline (Method B2 of Commission Directive 92/69/EEC, Annex V, as amended by 93/21/EEC) specifications.

Three groups of 10 Wistar Crl:(WI) BR strain rats (5 females and 5 males each group) were nose-only exposed to CdTe aerosol during 4 hours continuously. TSE Rodent Exposure System (TSE Systems GmbH, Bad Homburg, Germany) was used. This system is comprised of 2 concentric anodized aluminum chambers and a computer control system incorporating pressure detectors and mass flow controllers.

Due to the granular nature of the material, it was grounded using a VEB Elmo Ball Mill. Fresh aerosol from the generation system was constantly supplied to the inner plenum (distribution chamber) of the exposure system from where, under positive pressure, it was distributed to the individual exposure ports. The rats were held in polycarbonate restraint tubes located around the chamber which allowed only the rat's nose to enter the exposure port. After passing through the animal's breathing zone, spent aerosol entered the outer cylinder from where it was exhausted through a suitable filter system. Atmosphere generation was, therefore, dynamic.

A target concentration of 5 mg/L was used for the first exposure group. Subsequent targets were based on the results of the preceding exposure to produce a range of mortality rates. Thus, the second and the third groups were exposed to 1 mg/L and 3 mg/L, respectively.

The test atmosphere was sampled at regular intervals during each exposure period. Samples were taken from an unoccupied exposure port by pulling a suitable, known volume of test atmosphere through weighed GF10 glass fiber filters (Schleicher & Schuell GmbH, Dassel, Germany or similar). The difference in the pre- and post-sampling weights, divided by the volume of atmosphere sampled, was equal to the actual achieved test atmosphere concentration.

The particle size of the test atmosphere was determined 3 times during the exposure period using a 7-stage impactor of Mercer style (TSE Systems GmbH, Bad Homburg, Germany). The total amount collected for each stage was used to determine the cumulative amount below each cut-off point size. In this way, the proportion (%) of aerosol less than 0.33, 0.5, 0.77, 1.21, 1.93, 3.13, and 5.09 μm was calculated and the mass median aerodynamic diameter (MMAD) and Geometric Standard Deviation were calculated. In addition, the proportion (%) of aerosol less than 4 μm (considered to be the respirable fraction) was determined.

The median lethal concentration values and 95% confidence intervals were calculated by probit analysis.[7] Statistical analysis was performed by SPSS/PC software package.

Acute Oral Toxicity

This acute oral toxicity study was conducted according to the requirements of the US EPA Health Effects Test Guidelines, OPPTS 870.1100, Acute Oral Toxicity, August 1998, the OECD Guidelines for Testing of Chemicals 1981 No. 423 "Acute Oral Toxicity – Acute Toxic Class Method," and the Directive 2004/73/EC (B.1 Tris 08 June 2004) of the European Community.

A group of 3 female Wistar Crl:(WI) BR strain rats was treated orally with a suspension of the CdTe, in 1% Carboxymethylcellulose. The animals were treated at a dose level of 2000 mg/kg, followed by a 14-day observation period. In the absence of clinical signs after treatment, a second group of 3 females was treated in the same manner and at the same dose level to confirm results.

On the day before treatment, food was withheld from the animals overnight; water was available to them during this period. On the day of treatment, animals were weighed before dosing. The exact volume to be given was calculated based on each animal's body weight and a constant dose of 10 mL/kg. The test item formulation was stirred continuously throughout the dosing procedure to ensure each rat was treated with a homogenous solution. A single administration by oral gavage was given. Food was made available to the animals 3 hours after the treatment which was followed by a 14-day observation period.

Results

Acute Inhalation Toxicity

As shown in Table 1, the mean atmospheric concentrations to which the 3 groups of rats were exposed were 5.73, 1.07, and 3.03 mg/L with Mean Mass Median Aerodynamic Diameters (MMADs) of 2.03, 3.18, and 3.58 μm, respectively. The total deaths

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Table 1. Mortality of Rats After Acute Nose-Only Exposure to CdTe and Median Lethal Concentrations

Group No.	Mean Atmospheric Concentration (mg/L)	Mean Mass Median Aerodynamic Diameter (μm ± GSD)	Death		
			Male	Female	Total
1	5.73 ± 1.52	2.03 ± 1.99	5/5	5/5	10/10
2	1.07 ± 0.12	3.18 ± 2.29	0/5	0/5	0/10
3	3.03 ± 0.18	3.58 ± 2.13	4/5	3/5	7/10
Median lethal concentration (mg/L)			2.53	2.87	2.71

Table 2. Body Weight of Rats Before and After Acute Inhalation Exposure to CdTe

Group No. and Gender	Body Weight (g)				Weakly Increase (g)	
	Day 0	Day 7	Day 14	At death	Week 1	Week 2
1 – M	228	-	-	193	-	-
1 – M	233	-	-	211	-	-
1 – M	230	-	-	188	-	-
1 – M	235	-	-	188	-	-
1 – M	236	-	-	214	-	-
1 – F	226	-	-	188	-	-
1 – F	224	-	-	194	-	-
1 – F	229	-	-	165	-	-
1 – F	226	-	-	178	-	-
1 – F	226	-	-	183	-	-
2 – M	232	222	240	240	-10	18
2 – M	234	241	266	266	7	25
2 – M	232	238	279	279	6	41
2 – M	234	209	204	204	-25	-5
2 – M	238	229	249	249	-9	20
2 – F	229	225	249	249	-4	24
2 – F	238	201	184	184	-37	-17
2 – F	226	234	249	249	8	15
2 – F	234	203	221	221	-31	18
2 – F	238	236	253	253	-2	17
3 – M	289	-	-	222	-	-
3 – M	299	327	354	354	28	27
3 – M	294	-	-	242	-	-
3 – M	294	-	-	248	-	-
3 – M	298	-	-	243	-	-
3 – F	235	203	209	209	-32	6
3 – F	251	-	-	196	-	-
3 – F	248	198	186	186	-50	-12
3 – F	262	-	-	209	-	-
3 – F	248	-	-	188	-	-

were respectively 10/10, 0/10, and 7/10. The median lethal concentration for all animals was calculated to be 2.71 mg/L, and showed a very low variability between genders.

The majority of surviving rats (9/13) showed body weight losses after the first week of exposure, with an average overall weight loss of 11.6 g (4.84% of the body weight at the day of exposure) (Table 2). At the end of the second week of observation, the body weight of only 5/13 rats was still lower, while the overall body weight for all rats exceeded the initial one by 2 g (0.83%).

Some clinical observations were made. Excluding those related to the fur (wet or staining), the main observations after exposure are presented in Table 3

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and are related to emaciation, labored respiration, and increase of respiratory rate.

Acute Oral Toxicity

There was no mortality or clinical sign observed during the 14-day observation period among the 2 groups of rats. Normal body weight development was observed, with the exception of a single female which showed a slight body weight loss during week 2 only (Table 4).

Occasional occurrences of pale, raised areas were observed in the lungs of 2 animals at necropsy. Several instances of pin-prick sized hemorrhages in the lungs were also noted but such alteration is not unusual. Finally, mild hydrometra was noted in 1 female. This is a sporadic occurrence in laboratory maintained rat and is without toxicological significance.

Under the experimental conditions of this study, no deaths occurred among the 2 groups of 3 female rats treated orally at a dose level of 2000 mg/kg. The median oral lethal dose was, therefore, considered to be greater than 2000 mg/kg.

Discussion

This study showed that the median lethal concentration of CdTe is 2.71 mg/L/4 hours while its median lethal oral dose was considered to be greater than 2000 mg/kg.

According to *The Registry of Toxic Effects of Chemical Substances data bank* (http://www.cdc.gov/niosh/rtecs/eu958940.html, visited in July 2008), the median lethal concentration for Cd is 0.025 mg/L/30 minutes for rats. For comparison, Haber rule can be used (ie, $Cxt = k$, where C = exposure concentration, t = exposure duration, and k = a constant).[8] Accordingly, for 4 hours of exposure, this concentration leads to a value of 0.0031 mg/L/4 hours, which is very much lower than the value for CdTe (2.71 mg/L/4 hours). However, it must be remembered that the production of CdTe involves the use of Cd and the risk of toxic effects may be still significant for the exposed workers.

According to the *Hazardous Substances Data Bank*, the median lethal dose for Cd is 225 mg/kg.[9] The comparison between the median lethal doses suffers by the limitation of this study which was dictated by the OECD 423 guidelines. According to these guidelines, the dose level to be used as the starting dose has to be selected from 1 of 4 fixed levels, 5, 50, 300, and 2000 mg/kg body weight.[10] The starting dose level should be that which is most likely to produce mortality in some of the dosed animals. In addition, if the test was performed at the highest stipulated starting dose and induced no mortality, the guidelines do not permit testing higher doses. We agree with the OECD guidelines and we believe that the use of a higher dose is not scientifically justified. However, we should keep in mind that the 2000 mg/kg used in this study should not be interpreted as the exact value of the median lethal dose; it is a lower limit.

Because no mortality and no clinical sign were observed during the course of the study and considering that the increase of body weight was almost normal, we can reasonably make the hypothesis that the median lethal dose should be expected to be much higher. However, taking this value as a comparison basis, the median lethal dose for Cd is at least 8.9 times (225 mg/kg vs 2000 mg/kg) lower than that for CdTe. As was the case for the inhalation route, Cd appears to be more toxic than that for CdTe for the oral route.

Because the median lethal dose determined in this study is higher than 2000 mg/kg, CdTe should be ranked into Category 5 of the Globally Harmonized Classification System and according to 2001/59/EC Directive, classification of CdTe by the oral route is not required. According to the guidance provided by the US EPA, CdTe should be ranked into Toxicity Category III "Caution."

Comparing the data obtained in the present study to those related to tellurium also gives an interesting perspective. The median lethal concentration for Te was established by TNO Nutrition and Food Research as higher than 2.42 mg/L.[11] This level was the highest concentration reached by the laboratory and should not be interpreted as an exact median lethal concentration, but as a minimum value. This level is quite near the value of CdTe found in the present study (2.71 mg/L). Again, caution must be exercised as the level of 2.42 mg/L for Te did not lead to any death and the exact value is expected to be higher. It appears reasonable to hypothesize that a real median lethal concentration for Te should be higher than the one for CdTe, but these results do indicate that the acute inhalation toxicity of CdTe resembles that of Te more than that of the toxic Cd.

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Table 3. Clinical Observations on Rats After Acute Inhalation Exposure to CdTe

	Animal No.	Day 1	2	3	4	5	6	7-14
Group 1 (5.73 mg/L) Male	9944	RL-2	X	-	-	-	-	-
	9945	X	-	-	-	-	-	-
	9947	RL-2	X	-	-	-	-	-
	9953	RL-2	RL-2	X	-	-	-	-
	9959	X	-	-	-	-	-	-
Group 1 (5.73 mg/L) Female	9962	RL-2	X	-	-	-	-	-
	9963	RL-2	X	-	-	-	-	-
	9964	RL-2	RL-2	RL-2, EMA-1	EMA-2, RL-2, LGY-1	X	-	-
	9974	RL-2	RL-2	X	-	-	-	-
	9979	RL-2	RL-2	X	-	-	-	-
Group 2 (1.07 mg/L) Male	401	N	N	EMA-2	EMA-2	EMA-2	EMA-2	EMA-1 (day7-11)N (day12-14)
	402	N	N	EMA-1	EMA-1	EMA-1	EMA-1	N
	419	N	N	EMA-2	EMA-2	EMA-1	EMA-1	N
	420	N	N	EMA-2	EMA-2	EMA-2	EMA-2	EMA-2
	421	N	N	EMA-2	EMA-2	EMA-2	EMA-2	EMA-1 (day 7-9)N (day 10-14)
Group 2 (1.07 mg/L) Female	441	N	N	EMA-2	EMA-2	EMA-1	EMA-1	EMA-1 (day 7-9)N (day10-14)
	442	N	N	EMA-2	EMA-2, RBH	EMA-1, RBH	EMA-1, RBH	EMA-2, RBH
	447	N	N	EMA-1	EMA-1, RBH	EMA-1, RBH	RBH	RBH
	448	N	N	EMA-2	EMA-2	EMA-2	EMA-2	EMA-2 (day 7-9)EMA-1 (day 10-13)RBH (day11-14)
	449	N	N	EMA-2	EMA-2	EMA-1	EMA-1	N
Group 3 (3.03 mg/L) Male	404	RL-1	RL-1, EMA-2	RL-1, RI-1EMA-2	X	-	-	-
	406	N	N	RBH	RBH	RBH	RBH	RBH
	411	N	RL-1, EMA-2	X	-	-	-	-
	414	RI-1	RI-1, EMA-2	X	-	-	-	-
	417	N	RL-1, EMA-2	X	-	-	-	-
Group 3 (3.03 mg/L) Female	436	N	EMA-1	EMA-1, RL-1	EMA-1, RL-1	EMA-2, RI-1	EMA-2, RI-1	EMA-2, RI-1
	438	N	EMA-2	X	-	-	-	-
	440	N	EMA-2	EMA-2, RL-1	EMA-2, RL-1	EMA-3, RI-1	EMA-3, RI-1	EMA3, RI-1
	450	RL-2	EMA-2, RL-1	X	-	-	-	-
	451	RL-1	EMA-2, RL-1	EMA-2, RL-1	X	-	-	-

EMA, emaciation; LGY, lethargy; N, no abnormalities detected; RBH, red-brown staining of head; RI, respiratory rate increased; RL, laboured respiration; X, found dead.
1: slight or small or few; 2: moderate or several, 3: severe or large or many.

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Table 4. Body Weight of Female Rats Before and After Acute Oral Exposure to 2 000 mg/kg of CdTe

	Body Weight (g)			Weakly Increase (g)	
Group No.	Day 0	Day 7	Day 14	Week 1	Week 2
1	196	236	256	40	20
1	197	237	254	40	17
1	198	239	256	41	17
2	202	250	261	48	11
2	205	235	230	30	-5
2	203	241	264	38	23

For the oral pathway, it is difficult to compare acute toxicity of CdTe (>2000 mg/kg) with that of Te (>5000 mg/kg),[12] because in both cases, we only have access to a minimum value.

Conclusion

With a median lethal concentration of 2.71 mg/L and a median lethal oral dose higher than 2000 mg/kg, CdTe has been shown to be far less toxic than Cd. This clearly shows that care must absolutely be taken when trying to read-across toxicological data from 1 parent element (Cd) to the compound (CdTe). This conclusion supports that of Sinha et al[13] who consider that policies, when possible, should not only be based on the precautionary principle, because this could potentially lead to restrictions of use of substances such as CdTe due to their cadmium content, while data indicate that these substances have significantly different toxicological properties. However, there is still an important lack of adequate toxicological and ecotoxicological information and further studies are needed to provide successful implementation of evidence-based risk assessment approaches.

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