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Children's Exposure to Di(2-ethylhexyl)phthalate and Dibutylphthalate Plasticizers from School Meals

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ABSTRACT: Packed school meals for children 3−10 years old were studied to evaluate the levels of di(2-ethylhexyl)phthalate (DEHP) and di-*n*-butylphthalate (DBP) and the influence of the packaging process on meal contamination, and their contribution to daily intake was estimated. The packaging consisted of polyethylene-coated aluminum (PE/Al) dishes thermally welded by a polyethyleneterephthalate-coated aluminum (PET/Al) foil. Foodstuffs before processing were analyzed, too. Total meals before packaging and after packaging were collected. It was found that 92% of foodstuffs employed in meal preparation contained DEHP, and 76% of them DBP, at detectable levels. In cooked foods before packaging the DEHP median concentration levels varied from 111.4 to 154.8 ng/g ww and those of DBP between 32.5 and 59.5 ng/g ww. In packed meals the DEHP median values ranged from 127.0 to 253.3 ng/g ww, and DBP median values varied from 44.1 to 80.5 ng/g ww. The mean increases of median concentrations of DEHP in cooked foods before and after packaging were 113 and 125% for DBP. For nursery and primary school children DEHP intake via school meals can raise on average the respective EFSA TDI by 18 and 12% and that of DBP by 50 and 30%.

KEYWORDS: di(2-ethylhexyl)phthalate, dibutylphthalate, packed school meals, children's exposure

INTRODUCTION

Children are particularly susceptible and vulnerable to xenobiotics for different metabolic and physiological activities that rotate during the life cycle. The specific vulnerability of childhood is incidental to many intrinsic or expressed factors of children, such as greater pulmonary air volume, more permeable skin, and larger amounts of food and water ingested in relation to body weight, which is why exposure from respiration, through the skin, and via food is higher than in adults. Furthermore, children spend a considerable time indoors, such as at home, in a day nursery, and in school, where dust can represent a major source of exposure to xenobiotics;[1,2] ground contact, the habit of sucking and playing with pets treated with insecticides, and inadequate hand-washing can further increase children's chemical intake. Furthermore, with regard to food, as children typically consume milk, dairy products, and baby foods, the pollutants they ingest may be very different from those of adults.

In industrialized countries, exposure to some xenobiotics is considered to be probably involved in an increase of disorders in the immune system, neurobehavior, and puberty. Many ubiquitous pollutants such as polychlorinated and polybrominated biphenyls (PCBs, PBBs), dioxins, some biocides and metals, and, principally, phthalates (PAEs) are considered to be potentially involved in endocrine system interference and disruption. PAEs are synthetic organic chemicals introduced in the 1920s, among which, for their use as plasticizers in resins and polymers such as PVC, the main compounds are di(2-ethylhexyl)phthalate (DEHP) and di-*n*-butylphthalate (DBP).[3−4] PAE toxicological studies have shown negative effects on the development of the male reproductive system in rodents[5,6] and possibly in humans.[7] Such findings are thought to be due to the endocrine disruptive action of phthalates during the phase of sexual differentiation in the fetus.[8] DEHP and DBP and their metabolites have especially been shown to cause antiandrogenic effects manifested as decreased anogenital distance (AGD), cryptorchidism, decreased testosterone levels, and sperm production and infertility. According to the European Union (EU) criteria for classification and labeling of dangerous substances, DEHP has been classified in category 2 as toxic both for reproduction and for development (R60-61).[9] Recently Kim et al.,[10] on the basis of the results of a study on 261 Korean children, aged 8−11 years, on the relationship between the clinical syndrome of attention-deficit/hyperactivity disorder (ADHD) and phthalate exposure, suggested the possible association between phthalate metabolism and the inattention and hyperactive−impulsivity phenotype of ADHD.

Food is the major source of several PAEs in humans and, particularly, of DEHP and DBP isomers,[11−13] depending on environmental pollution and processing, storing, and packaging practices. Because DEHP and DBP are not chemically bound to plastic matrices, they can easily migrate, due to thermal or mechanic stress, into food contacting plastic surfaces and equipment such as containers, tubes, gloves, and packaging, including adhesives and imprints. The available data show that retail-packaged foods are widely contaminated by phthalate plasticizers[2,12,14−17] and that cooked foods, such as retail or hospital ready meals, can also be highly contaminated.[18] This is why the EU has restricted phthalate use in food contact materials[19] and, to reduce the daily exposure to the more widely occurring phthalates, since 2005 the European Food Safety Authority (EFSA) has set tolerable daily intakes (TDIs) of 0.05 mg/kg body weight (bw) for DEHP and 0.01 mg/kg bw for DBP.[20−22]

Phthalate food contamination is considered to be of major importance, especially with regard to the age of consumers. Because

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Table 1. Composition and Mean Portion Sizes of School Meals Sampled

meal component	composition	mean portion size (g)	
		nursery school	primary school
first course	pasta or rice with tomato sauce and Parmesan cheese or with legumes; pasta or rice in vegetable; vegetable soup; potato dumplings with minced meat; gateau	191	231
second course	beef or pork stew or meatballs; frankfurters, ham; fish steaks; mozzarella or processed cheese	80	95
vegetables	peas, potatoes, spinach with cheese, runner beans, carrots, boiled chard dressed with olive oil, maize	80	108
fresh fruit	apple, peer, orange	180	180
bread	roll	100	100

the toxicological profile of phthalates shows that developmental effects are the greatest concern, children have to be considered the most susceptible category. Because in the majority of industrialized countries, children attending school consume school meals, careful monitoring is required in terms of safety and, in this case, phthalate contamination.

This study aimed to evaluate children's exposure to DEHP and DBP plasticizers from school meals and ascertain to what extent packaging can affect the concentration of such contaminants in ready-to-eat meals.

MATERIALS AND METHODS

Sampling. In Italy 3−10-year-old children make up the majority of refectory users. Generally, they have lunch at school, which is the main meal (40% of daily caloric contribution). According to local habits, a typical lunch is based on a first course of pasta or rice with tomato sauce or with legumes, vegetable soup, potato dumplings, etc.; a second course based on meat, fish, or dairy products; fresh or cooked vegetables; fresh fruit; and bread. The portions vary depending on the age of children (Table 1). The school meal service is supplied 5 or 6 days a week for about 8−10 months per year. In recent years, meals in Italian state schools have been mainly farmed out on contract to catering firms under the control of the Food Hygiene and Nutrition Service of the Italian Department of Health, which attempt with their nutritionists or dieticians to compile the diets to obtain well-balanced meals.

The production system consists mainly in food cooking, portioning quickly in disposable dishes that are sealed and transported up to 0.5 h away, maintaining the prescribed temperatures (>60 °C or <4−10 °C) during transport. The first courses are always cooked dishes, whereas the second courses may comprise cooked foods or foodstuffs not produced in the catering firms such as sliced cured meat (ham, salami) or dairy products (mozzarella, processed cheese) packed in plastic and served in their original industrial packaging. Bread consisted of rolls, also packed in sealed plastic by bakeries that deliver them to the catering firms on a daily basis.

To achieve our study aims, a catering firm supplying >1700 meals/day for nursery and primary schools in Naples (Italy) was involved. To evaluate the levels of DEHP and DBP and the influence of the packaging process in the meals supplied, the following were sampled from February to May 2010: (at the catering firm) foodstuffs before processing, including packed cured meats, dairy products, rolls, and fresh fruit, as well as cooked courses immediately before packaging; (at the schools supplied) packed ready meals at time of consumption.

In particular, almost 60 samples of foodstuffs, namely, cereals (pasta, rice), dry legumes (beans, lentils), potato dumplings, meat (fresh and cured), fish-based foods, dairy products (mozzarella cheese and processed cheese), vegetables (frozen spinach, peas, carrots, chard; potatoes; canned maize), and condiments (olive oil, tomato sauce, onion, milk, eggs, Parmesan cheese, etc.), were collected twice (in February and April). As the menu changed every week (from Monday to Friday), meal sampling was carried out over a period of 4 weeks, with 20 complete meals being sampled before and after packaging. The meals consisted of a first course, a second course, and vegetables (a total of 120 serving portions) plus bread (20 samples) and fruit (20 samples) (Table 1). The packaging in question consisted of polyethylene-coated aluminum (PE/Al) dishes thermally welded by polyethyleneterephthalate-coated aluminum (PET/Al) foil. The packed courses were placed on electrically powered isotherm serving carts, loaded onto vehicles, and delivered to schools. About 100 g of each food was put in glass jars that were hermetically closed by tops covered inside by an aluminum foil (rinsed twice with acetone and *n*-hexane) and transported to the laboratory, where the dry foods were ground and divided into 5 g aliquots; wet solid foods were homogenized, divided into 5 g aliquots, and lyophilized. As children consumed only peeled apples, pears, and oranges, the fresh fruit collected was peeled prior to being homogenized. Eggs were opened, and the content of each egg was weighed, homogenized, subdivided into 3 g aliquots, and lyophilized. Liquids (olive oil and milk) were divided into 1 and 15 g aliquots, respectively; milk samples were lyophilized.

A complete portion of each ready course was collected immediately prior to packaging, put in a glass jar capped as described above, and taken to the laboratory, where it was codified, weighed, homogenized, subdivided into 5 g aliquots, and lyophilized. The packaged courses sampled at school were codified, weighed, homogenized, subdivided into 5 g aliquots, and lyophilized. All of the analytical samples were stored in airtight glass jars in the dark at 4 °C until processed.

Chemicals. Acetonitrile, *n*-hexane, acetone for organic trace analysis, and anhydrous Na_2SO_4 were supplied by Merck (Darmstadt, Germany). Florisil (60/100 mesh) was furnished by Supelco (Bellefonte, PA) and Bondesil (PSA 40UM) by Varian (Palo Alto, CA). Di(2-ethylhexyl)phthalate and dibutylphthalate standards were purchased from Sigma-Aldrich (Dorset, U.K.).

DEHP and DBP Detection. As the PAEs are ubiquitous, during each analytical phase several precautions were adopted to avoid contamination. All of the glassware used in sampling and in analytical activities was thoroughly washed and rinsed twice with acetone and *n*-hexane.

In accordance with the Tsumura et al.[18] method, phthalates were extracted from food samples by 15 mL of acetonitrile three times in an ultrasound bath for 15 min; the samples were centrifuged at 2000 rpm for 10 min, transferring the acetonitrile layers to a separatory funnel; 10 mL of *n*-hexane saturated with acetonitrile was added, and the funnel was vigorously shaken for 5 min. The acetonitrile phase containing the phthalates was transferred to a flask and dried under vacuum at 55 °C. The dried extracts were reconstituted by 5 mL of *n*-hexane and cleaned

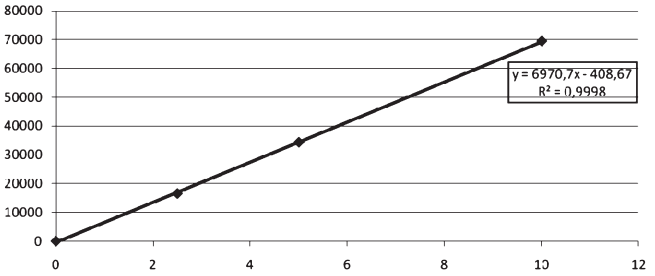

Figure 1. DEHP calibration curve in the range 2.50−10.00 μg/mL.

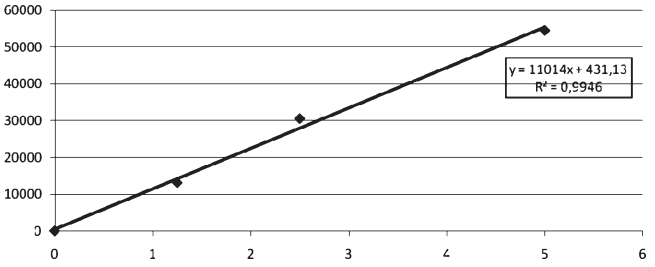

Figure 2. DBP calibration curve in the range 1.25−5.00 μg/mL.

up on a column consisting of 2 g of Florisil, activated for 2 h at 200 °C, 0.5 g of Bondesil, and 1 g of anhydrous Na_2SO_4. The column was eluted three times with 10 mL of *n*-hexane/acetone (100:5 v/v), the eluates being collected. The eluates were evaporated under vacuum at 40 °C and reconstituted with 2 mL of *n*-hexane for GC analysis. PAE analyses were carried out by a Shimadzu GC-17 (Shimadzu, Kyoto, Japan) capillary gas chromatograph with a flame ionization detector (FID), injecting 1 μL of each extract on an HP-5 (cross-linked 5% PHME Siloxane, 30 m length, 0.32 i.d., 0.25 μm film thickness) glass capillary column. Helium was used as carrier and hydrogen/air for the flame. The injection mode was splitless, the injector temperature was 260 °C, and the detector temperature was 310 °C. The temperature program was 100 °C for 1 min, increasing by 15 °C/min to 280 °C and staying at this temperature for 10 min.

Quantification and Quality Parameters. The calibration curves were made using DEHP and DBP standard solutions at three different levels, 2.50, 5.00, and 10.00 μg/mL for DEHP and 1.25, 2.50, and 5.00 μg/mL for DBP. Each concentration level was injected three times, and mean area value was considered to make the calibration curve. The regression coefficients (R) were >0.99 for both DEHP and DBP (Figures 1 and 2). The PAE concentrations (ng/g) were calculated by comparison with them.

To test the accuracy and validity of the method, the recoveries of DEHP and DBP from various spiked foods and meals were assessed. As PAE certified matrices are not available on the market, some representative samples of different foodstuffs and serving portions were submitted to recovery tests, adding 1 mL of three standard solutions containing DEHP at 10.0, 20.0, and 40.0 μg/mL and DBP at 5.0, 10.0, and 20.0 μg/mL and processing them as food samples. The recoveries obtained were 80.3 ± 3.5% for DEHP and 102.8 ± 4.4% for DBP.

To highlight possible contaminations a blank sample was tried with every series of analysis, and the value was subtracted from detected PAE values. Twenty blanks, obtained by submitting only the reagents to the analytical procedure, were analyzed, and LOD and LOQ values were obtained. For DEHP the LOD was 5.0 ng/g and the LOQ 15.0 ng/g, whereas for DBP the two values were 7.5 and 22.5 ng/g, respectively.

Statistical Analysis. To evaluate the influence of packaging on DEHP and DBP contamination levels, PAE contents in foods before and after packaging were compared by log-transforming the values to approximate a normal distribution of the data. All mean results are upper estimates that assume phthalates not detected as present at the limit of detection. Analysis of variance was carried out by ANOVA. The level of significance was set at $p < 0.05$. Statistical analysis was performed by SPSS 13.0.

RESULTS AND DISCUSSION

Figure 3 shows some GC chromatograms: one blank, two foodstuffs (raw pasta and cooked ham), and three courses after packaging (rice with tomato sauce, pork stew, and maize pan cooked).

The DEHP and DBP concentrations in foodstuffs are shown in Table 2. Among the foodstuffs are considered bread rolls and fruit results also, because these products were not processed and packaged in the catering industry. DEHP was found at detectable levels on average in 92% of foodstuffs analyzed and DBP in 76% of them. Meat, fish, and diary products, fresh fruit, and bread showed a 100% prevalence of DEHP contamination, followed by cereals and legumes (93%), vegetables (80%), and condiments (66%). Fish-based foods, fresh fruit, and bread showed a 100% prevalence of DBP contamination, followed by meat (85%), cereals and legumes (72%), dairy products (67%), vegetables (60%), and condiments (45%) (data not shown).

Bread and fish showed the highest DEHP concentrations, with median values, respectively, of 314.0 and 136.5 ng/g wet weight (ww). The highest DBP contents were found in bread rolls (101.0 ng/g ww), fresh fruit (66.0 ng/g ww), and fish (60.4 ng/g ww). The lowest concentrations of both DEHP and DBP were found in condiments (DEHP median concentration, 18.6 ng/g ww; range, 5.0−60.4 ng/g ww; DBP median, 7.5 ng/g ww; range, 7.5−19.2 ng/g ww). Except fresh fruit, the analyzed foods were less contaminated than cereals, bread, biscuits, cakes, nuts, spices, fat, and oil from Germany, the United Kingdom, and Japan (DEHP and DBP concentrations up to about 10 mg/kg) as reported by Wormuth et al.[12]

DEHP and DBP levels in cooked foods before and after packaging are respectively shown in Tables 3 and 4. Before packaging, the DEHP median concentration levels varied from 111.4 to 154.8 ng/g ww; the lowest value was found among vegetables in a "spinach with cheese" dish (22.0 ng/g ww) and the highest among the first courses in "rice in vegetable soup" (379.4 ng/g ww).

In packaged meals sampled at schools the DEHP median values ranged from 127.0 to 253.3 ng/g ww; the lowest value was found again in "spinach with cheese" (21.3 ng/g ww) and the highest in a first course of "rice with tomato sauce" (1050.8 ng/g ww) (Table 3).

The median concentrations of DBP before packaging ranged between 32.5 and 59.5 ng/g ww; the lowest value was found among vegetables in a sample of "boiled chard dressed with olive oil" (11.0 ng/g ww) and the highest among the first courses in "potato dumplings with minced meat" (165.4 ng/g ww). After packaging, the DBP median values ranged from 44.1 and 80.5 ng/g ww, with the lowest value (18.3 ng/g ww) among the second courses and in vegetables, respectively in "frankfurters" and "maize"; the most DBP-contaminated course was again "potato dumplings with minced meat" (775.0 ng/g ww) (Table 4). The DEHP and DBP median concentrations in cooked foods before packaging were lower than those after packaging at the time of consumption, with mean increases of 113% for DEHP and 125% for DBP and differences statistically significant between the first courses

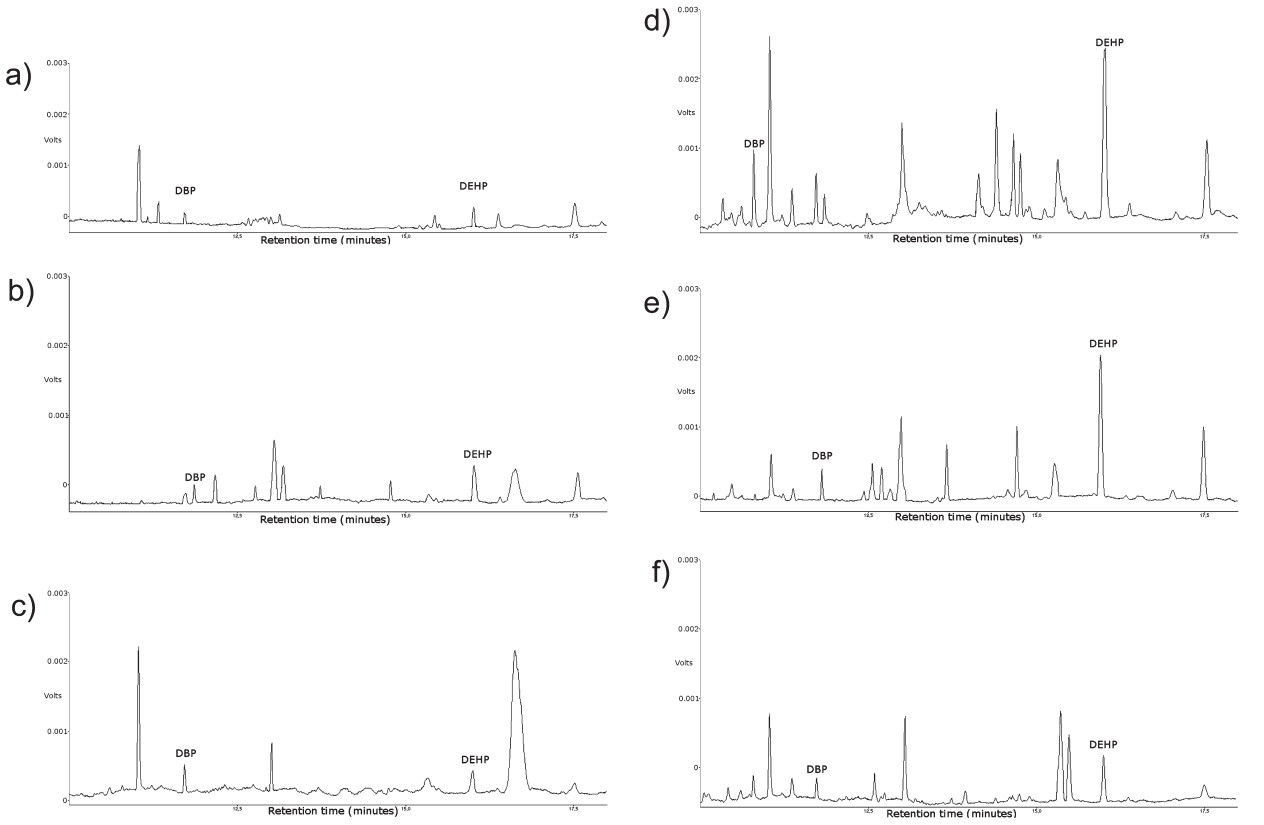

Figure 3. GC chromatograms of a blank (a), raw pasta (b), cooked ham (c), rice with tomato sauce after packaging (d), pork stew after packaging (e), and maize pan cooked after packaging (f).

Table 2. DEHP and DBP Concentrations in Foodstuffs (n = 60), Fresh Fruit (n = 20), and Bread Rolls (n = 20) Sampled at the Catering Industry Involved in the Study

	DEHP (ng/g ww)		DBP (ng/g ww)	
foodstuff	mean ± SD (min−max)	median	mean ± SD (min−max)	median
cereals and legumes	75.1 ± 87.8 (5.0−270.4)	38.4	50.5 ± 50.0 (7.5−152.4)	25.7
meat based	101.7 ± 88.4 (5.5−350.0)	80.0	52.2 ± 43.5 (7.5−147.0)	43.6
fish based	140.0 ± 52.2 (93.6−193.5)	136.5	81.1 ± 70.5 (23.8−180.0)	60.4
dairy	161.8 ± 235.5 (8.8−433.0)	43.6	32.2 ± 28.4 (7.5−63.2)	26.0
vegetables	87.2 ± 82.8 (5.0−265.2)	56.0	38.1 ± 46.2 (7.5−173.2)	20.2
condiments	22.8 ± 20.4 (5.0−60.4)	18.6	10.2 ± 4.0 (7.5−19.2)	7.5
fresh fruit	77.3 ± 34.9 (40.0−109.0)	83.0	57.0 ± 23.8 (30.0−75.0)	66.0
bread	270.3 ± 142.6 (111.0−386.0)	314.0	142.8 ± 78.0 (93.0−232.0)	101.0

Table 3. DEHP Concentrations in Ready Courses before (n = 60) and after Packaging at Consuming Time (n = 60)

	DEHP (ng/g ww)			
	before packaging		after packaging	
course	mean ± SD (min−max)	median	mean ± SD (min−max)	median
first[a]	146.6 ± 99.7 (37.9−379.4)	112.6	311.4 ± 255.1 (36.6−1050.8)	224.6
second	182.4 ± 100.3 (24.6−329.5)	154.8	250.4 ± 163.4 (43.6−497.2)	253.3
vegetables	117.0 ± 70.0 (22.6−365.0)	111.4	183.0 ± 140.4 (21.3−365.0)	127.0

[a] First course values showed statistically significant differences between before and after packaging (p = 0.02).

Table 4. DBP Concentrations in Ready Courses before (n = 60) and after Packaging at Consuming Time (n = 60)

	DBP (ng/g ww)			
	before packaging		after packaging	
course	mean ± SD (min−max)	median	mean ± SD (min−max)	median
first[a]	65.4 ± 40.6 (16.4−165.4)	59.5	169.3 ± 217.7 (19.9−775.0)	80.5
second	51.9 ± 35.5 (19.8−112.5)	33.1	86.8 ± 93.4 (18.3−336.8)	44.1
vegetables	42.1 ± 27.4 (11.0−91.0)	32.5	92.9 ± 82.5 (18.3−236.1)	68.8

[a] First course values showed statistically significant differences between before and after packaging (p = 0.012).

Table 5. DEHP and DBP Contents in Nursery and Primary School Meals before and after Packaging

	PAEs (mean ± SD (range), μg)			
	nursery school		primary school	
PAE	before packaging	after packaging	before packaging	after packaging
DEHP	101.9 ± 22.9 (69.0−152.0)	144.9 ± 48.8 (72.0−301.0)	125.3 ± 40.6 (71.0−228.0)	170.0 ± 63.9 (76.0−348.0)
DBP	50.3 ± 11.5 (36.4−79.4)	91.2 ± 71.6 (41.4−272.8)	53.9 ± 11.0 (38.4−81.5)	86.8 ± 57.3 (45.4−243.4)

Table 6. DEHP and DBP Intake by School Meal for Children of Nursery and Primary Schools

	intake (mean ± SD (min−max), μg/kg bw/meal)	
PAE	nursery school	primary school
DEHP	8.6 ± 2.9 (4.2−17.7)	6.3 ± 2.4 (2.8−12.9)
DBP	5.4 ± 4.3 (2.4−16.9)	3.2 ± 2.1 (1.7−9.0)

(for DEHP, p = 0.002; for DBP, p = 0.012). The total amount of DEHP and DBP for a meal, considering the first and second courses and the vegetables contributions, was calculated by multiplying the concentrations for the weight of each portion and summing the values obtained. Both DEHP and DBP contents showed a significant increase (p = 0.02) at time of consumption in comparison with the contents in unpacked courses (Table 5).

To estimate the school meal contribution to daily intake of DEHP and DBP in children, we determined the ratios of the total DEHP and DBP amounts per meal (first and second courses, vegetables, bread roll, and fruit) at time of consumption to children's body weight. As suggested by the statistical reports of the Italian Society of Human Nutrition,[23] we considered for Italian children 3−5 years old (nursery school) a mean body weight (bw) of 17 kg and for those 6−10 years old (primary school) a mean body weight of 27 kg. We obtained a mean DEHP intake/meal of 8.6 ± 2.9 μg/kg bw (range, 4.2−17.7 μg/kg bw) in nursery school children and 6.3 ± 2.4 μg/kg bw (range, 2.8−12.9 μg/kg bw) in primary school children; the meal with highest intake levels consisted of rice with tomato sauce, ham and peas, bread roll, and apple. For DBP the mean intake/meal proved to be 5.4 ± 4.3 μg/kg bw (range, 2.4−16.9 μg/kg bw) in children 3−5 years old and 3.2 ± 2.1 μg/kg bw (range, 1.7−9.0 μg/kg bw) in those 6−10 years old (Table 6); the largest contributions to children's daily intake were given by three meals comprising potato dumplings, pork stew and potatoes, bread roll, and apple; potato dumplings, processed cheese and peas, bread roll and apple; lentils with pasta, mozzarella cheese, and maize, bread roll, and apple (data not shown). In light of the TDI values (50 μg/kg bw for DEHP and 10 μg/kg bw for DBP) established by the EFSA for adults of 70 kg bw,[20] we estimated on the basis of the mean and maximum values obtained that, in the study conditions, for nursery and primary school children DEHP intake via school meals can raise on average the respective TDI by 18% (maximum, 35%) and 12% (maximum, 26%) and DBP intake by 50% (maximum, 169%) and 30% (maximum, 90%). With regard to the maximum DBP contamination levels found in the meals at consumption, the intakes exceed the TDI in nursery school children and represent 90% of the TDI in primary school pupils. If we were to calculate intakes by DBP mean concentrations obtained on meals before packaging, respectively 50.3 μg in nursery school meals and 53.9 μg in primary school meals, we would obtain much lower DBP intake levels/meal, never exceeding the TDI.

Various studies have estimated DEHP and DBP total daily intakes from food in adult populations (70 kg bw).[13−28] Mean total daily intakes for an adult of 70 kg bw varied from 2.1 to 4.9 μg/kg bw for DEHP and from 0.2 to 4.2 μg/kg bw for DBP. For Danish children aged 1−6 and 7−14 years, Müller et al.[29] estimated mean TDIs, respectively, of 11.0 and 26.0 μg/kg bw for DEHP and 3.5 and 8.0 μg/kg bw for DBP. In the course of 1999 Tsumura et al. carried out two diverse studies on plasticizer contamination in meals served in three hospitals and in retail-packed lunches and set lunches from restaurants in Japan. They found DEHP contamination levels ranging from 10 to 4400 ng/g in composite meals from hospitals, estimating a mean intake of 519 μg/day for hospitalized patients. Disposable PVC gloves used during the preparation of meals were suspected as the source of the high DEHP content.[18] In the retail packed lunches analyzed, the levels of DEHP were much higher (346−11800 ng/g), whereas set lunches from restaurants were much less contaminated (12−304 ng/g). Disposable PVC gloves, sprayed with 68% ethanol to sterilize them, used in the factories producing the packed lunches, were confirmed as the principal source of DEHP contamination.[30] A further study carried out in 2001 by the same authors, following the regulation of DEHP-containing PVC gloves in Japan, showed that the DEHP levels in the hospital meals were

much lower (6−675 ng/g) ,and hence the estimated DEHP mean daily intake decreased to 160 μg.[31]

In conclusion, foodstuffs used to prepare meals analyzed in the present study showed diffuse contamination by DEHP and DBP, confirming the ubiquity of the contamination sources. The highest concentrations of DEHP and DBP were found mainly in the processed and packed foodstuffs employed in supplying school meals, suggesting that manufacturing and contact with plastic wrapping can play a major role in the phthalate contamination of foods, as shown also by the high levels of DEHP and DBP found in bread rolls that are quickly packed after baking at a temperature that can favor plasticizer release. The increase in DEHP and DBP concentrations in the courses after packaging testified to the influence of the contact of cooked foods with the kind of packaging (PE/Al dishes thermally welded by PET/Al foil) used in the catering firm considered. Among the various courses comprising the meal, the first courses showed the highest concentrations, with a significant (p = 0.02) increase of both DEHP and DBP after packaging. Those that, as described above, were based on pasta or rice with tomato sauce or legumes, vegetable soup, etc., and various condiments are the courses with the more complex composition that can maintain a high temperature (>60 °C) for a time longer than the other courses. Furthermore, their weight and volume are much higher than those of the other courses, so the first courses may establish maximum contact with package surfaces. The presence of tomato sauce or other liquid fat condiments (olive oil, milk, cream, etc.) could further influence the release of the package plasticizers. It may also be considered that after cooking, foods are quickly portioned and packed to be transported (up to 0.5 h) and served to children, but, as observed during the collection of the meals at the served schools, the time of transport can noticeably vary up to 1−2 h depending on traffic.

The DEHP and DBP intake values estimated in the present study are noticeably variable and, even if referred only to the lunch, can reach levels near or up to the TDI. School meals supplied in the described conditions could be improved to increase the safety of foods, reducing the phthalate contamination levels. Even if in the present study the influence of the temperature and time of contact or the specific composition of the courses, for example, the lipidic content, has not been studied, it is plausible that these parameters could influence the migration of the plasticizers from the packages into the foods. The reduction of the times between the cooking and serving of the meals and/or the choice of an alternative packaging could be good options to reduce precautionally the risks for the children's health.

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