EFFECT OF CRUDE OIL AND THE FRACTIONS ON THE BLOOD HAEMOGLOBIN AND NEUTROPHIL CONCENTRATIONS IN *HETEROBRANCHUS BIDORSALIS* JUNVENILES

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Abstract

Studies on the effects of crude oil and its fractions on the blood haemoglobin (HB) and neutrophil (NP) concentration in the blood of *Heterobranchus bidorsalis* juveniles were carried out. Two study periods (toxicity and recovery) and four oil types (Bonny-light crude oil (BLCO), premium motor spirit (PMS), kerosene (DPK) and lubricating engine oil (LBO) at 1.00, 2.00, 4.00 and 8.00ml L^{-1} were used. There were significant differences ($P<0.05$, $p< 0.01$) on the HB and NP values in the blood of fish samples. The elevated values of HB levels in fish exposed to 2.00,4.00 and 8.00m1 L^{-1} BLCC, 2.00ml L^{-1} PMS and DPK; and 4.00ml L^{-1} LBO over the levels in the control fish were in consonance with the results of other workers. Increases in NP values in fish exposed to the four oil types (BLCO, PMS, DKP and LBO) relative to the control could be part of the immunological attempts by the fish to mobilize the body's defence mechanisms. The relatively high percentage mortality and low survival of *H. bidorsalis* juveniles exposed to 4.00 and 8.00ml L^{-1} concentration of the four oil types over the other concentration implied that these concentrations were probably very lethal to fish existence.

Keywords: *Heterobranchus bidorsalis*, haemoglobin, neutrophil, crude oil concentration, toxicity, recovery.

Introduction

The incessant oil spillage in Nigeria and the resultant spoilage of valuable food fish informs the need to study the impact of crude oil and its fractions on the haematology of *Heterobranchus bidorsalis* juveniles. Despite the reported cases of rapid uptake of crude oil compounds from water by fish and the concomitant bioaccumulation that do occur (Payne and Penrose, 1994), little is known about the metabolism of these compounds in *H. Bidorsalis*.

The high demand of this giant African catfish, owing to its rapid growth and flavour, necessitated an investigation into the blood haemoglobin and neutrophil concentrations of the fish stocked in water polluted with crude oil and its fractions. Stone *et al.* (2002) stated that about five million red blood cells (RBC) are contained in one cubic millimetre ($1m^3$) of blood sample of vertebrates. Neff and Anderson (1981) reported that the exposure of fish to crude oil compounds resulted in the destruction of the RBC and WBC components of the blood as well as the alteration of the immune response and liver metabolism of fish among other damages. The effect of crude oil on adult fishes of *C. gariepinus* has been reported to increase the mortality rate and changes in the haemoglobin content of blood (Tatem, 1979). The response of aquatic organisms to pollution is reflected in changes in key enzymes, especially those of biotransformation systems. These biomakers may be sensitive and specific early warning signs for aquatic pollution (Strmac and Braunbeck, 2000). Polyaromatic and halogenated hydrocarbons (PAHs), heavy metals, polychlorinated biphenyls (PCBs) and other pesticides enter freshwater systems from industrial waste- waters, urban discharges and agricultural activities. In the long term all the pollutants may eventually result in ecotoxicological effects (Ozmen *et al.*, 2005). Persistent organochlorine (OC) pesticides accumulate in adipose tissues of non-target organisms and biomagnify in the food chain (Henriksen et al., 2000).

Detailed chemical analyses are needed to determine the effect of crude oil infiltration into the blood system of *H. bidorsalis* juveniles. It has been suggested that the uptake and translocation of crude oil compounds in fish may be through the gills, gut or the intestinal walls (Roubal, 1977); where the parent compounds solublize in the cell membranes and are carried via the erythrocytes to the general circulation of the blood. The specific individual metabolites that result from the uptake and metabolic conversion of petroleum aromatic compounds (ACs) in fish have not been well characterized (Krahn *et al.*, 1992). Previous studies have identified only a few individual AC metabolites in the livers (Krahn *et al.*, 1992) or bile (Hellou and Payne, 1987) of fish that have been exposed to no. 2 fuel oil, a distillate fraction of petroleum that contains only a portion of the ACs found in crude oil.

Against this background, this paper presents the results of the blood haemoglobin and neutrophil concentrations in *H. bidorsalis* juveniles exposed to different concentrations of crude oil and its fractions. The aim was to ascertain the concentration of crude oil compounds that could reduce the potency of the haemoglobin and neutrophil components of fish blood.

Materials and Methods

Six hundred (600) juveniles of *Heterobranchus bidorsalis* (Geoffrey St. Hilaire, 1809) (mean weight, 14.08±0.12g) were transported from a private fish hatchery at Otor Oweh in Delta State, Nigeria, in five plastic containers (25L) to the Fisheries Laboratory of Enugu State University of Science and Technology, Enugu. Water temperature (27+ 1.00°C) was maintained during transportation with ice cubes held in a portable cooker. The fishes were acclimatized, in the fisheries laboratory for 14 days and were maintained on a chick starter diet at 1% body weight a day ($bw.d^{-1}$).

Four samples of crude oil and its fractions were used for the study: Bonny- light crude oil (BLCO), premium motor spirit (PMS), Kerosene (DPK) and lubricating engine oil (LBO). Aliguots (5ml) of each of these four oil samples were introduced in triplicate to 48 plastic containers (25L) at concentrations of 1.00, 200, 4.00 and $8.00mlL^{-1}$. Twelve (12) plastic containers with fish were not contaminated with any oil samples ($0.00mlL^{-1}$) and were left as the control. The fishes were randomly stocked in a completely randomized block Design (CRBD) in 60 plastic container (25L) at 10 fish per container. Each container was filled to 24L mark with dechlorinated tap water. Two experimental periods were adopted for the study. The toxicity period lasted for 4 days (96 hours); while the recovery period (42 days) was monitored fortnightly. At the end of the toxicity period, the surviving fish and plastic containers were thoroughly washed with clean tap water to allow for the 42 days recovery period to commence. A 38% crude protein diet was fed to fish at 3% $bw.d^{-1}$ during the toxicity and 5% $bw.d^{-1}$ during the recovery periods. Blood samples of fish were also collected during each experimental period. Records of fish mortality/survival rates were taken, while feeding and swimming activities of the fish were observed. The water temperature (25±1.50°C) and pH(6.65+0.05) were recorded with the aid of a maximum- minimum thermometer and a pH meter (model ph-1-20^{-L}) respectively.

Blood samples of fish from each triplicate treatment of crude oil (BLCO), its fractions (PMS, DPK, LBO) and the control, were collected with 2.50ml capacity syringes and hypodermal needles. Blood collected was via the dorso- anterior musculatures, just below the dorsal fin and around the operculum. Anti-coagulant (EDTA) fluid was used to condition both the syringes and needles prior to blood collection. Analyses of blood samples were carried out within 12 hours at the Bronilla Diagonistic Laboratory, Enugu, Nigeria.

At the laboratory, triplicate samples of blood from fish of each triplicate treatment of crude oil/fractions and the control were analyzed for their haemoglobin (HB) and neutrophil (NP) concentration estimates using the method described by Wed Meyer and Yasutate (1973). All the data obtained were subjected to analysis of variance (ANOVA) to determine whether differences existed among treatment means (Steel and Torrie, 1990).

Results and Discussion

Tables 1a and 1b show the gross and proximate compositions of the experimental diet used during the toxicity and recovery periods of the study. Table 2 summarizes the haemoglobin (Hb) concentrations in the blood of *H. biorsalis* juveniles stocked in polluted water with BLCO, PMS, DPK and LBO; and the control. Hb values virtually remained constant in the control fish than in those exposed to the various oil concentrations during the 4 days toxicity period (Table 2). The 2.00, 4.00 and 8.00 mlL^{-1}fish concentrations of BLCO elicited higher values of Hb in the fish blood than in the control fish (Table 2). Similarly, the 2.00ml L^{-1} concentrations of PMS and DPK resulted in higher Hb concentrations on fish than in the control fish. The exposure of the fish to 4.00 mlL^{-1} LBO in water gave the highest value of Hb (7.18±0.21mg. $100m1^{-1}$) in the fish than in those exposed to other oil concentrations and the control. As was indicated with the Hb concentrations, the exposure of the fishes to the four oil types (BLCO, PMS, DPK and LBO) and their concentrations (1.00, 2.00, 4.00 and 8.00$m1L^{-1}$) exhibited a similar pattern of variations in NP values both at the toxicity and recovery periods of the study (Table 3). Consistent increase in NP values, however, was recorded in fishes exposed to the various concentrations of the four oil types and the control as they recuperated between days 14 and 42 (Table 3). Both at the toxicity and recovery periods, NP values in the fish varied significantly as the various concentrations of the four oil types and the control was applied ($p<0.05$; $p<0.01$) (Table3).

The percentage mortality and survival of *H. bidorsalis* juveniles during the toxicity and recovery periods of the study are shown in Tables 4. The fish exposed to 4.00 and 8.00$m1L^{-1}$ concentrations of crude oil (BLCO) and its fractions (PMS, DPK and LBO) exhibited higher percent mortality and lower percent survival than those exposed to the other concentrations (1.00 and 2.00$m1L^{-1}$,) and the control (0.00$m1L^{-1}$) (Table 4). This situation was applicable both at the 4 days toxicity and the 42 days recovery periods of the study. However, lower percent mortality of fish was recorded fortnightly as the fishes recovered than during the toxicity period. These trends in Hb concentrations due to fish exposure to the four oil types and their concentrations were exhibited both at the toxicity (4 days) and the fortnightly recovery periods of the study (Table 2). Significantly, there were consistent increases in Hb concentrations as the fishes recuperated between days 14 and 42 recovery periods. Thee increases in the Hb concentration with time were also expressed in the control fish. The results also indicated that both at the toxicity and the recovery periods of the study, Hb values varied significantly when *H. bidorsalis* juveniles were exposed to the various concentrations of the oil types and the control ($p<0.05$; $p<0.01$) (Table 2).

Table 3 shows the concentrations of the neutrophil (NP) components of the WBCs of *H. bidorsalis* juveniles. NP concentrations also remained virtually constant in the control fish than in those exposed to the oil pollutants during the toxicity period (Table 3). However, the 4.00 and 8.00 mlL^{-1} concentrations of BLCO gave higher values of NP in the fish than the 1.00 and 2.00mlL^{-1}BLCO and the control (0.00$m1L^{-1}$) (Table 3). Also at the toxicity period, the fishes exposed to 2.00$m1L^{-1}$PMS (204+1.04mg. $100m1^{-1}$) DPK (40.11+206mg. 100^{-1} and LBO (10.20+0.25mg. 100^{-1}) had higher values of HB. HB increases were recorded in fishes exposed to fertilizer (Van Vuren, 1986). The results of this study, however, varied with those of Hilmy *et al.*, (1980) and Annune *et al.* (1994) who reported decreases in Hb levels when *Aphanus dispar* (marine teleost) was exposed to mercury and *Clarias gariepinus* was exposed to zinc respectively. From the present study, it could be deduced that the higher Hb values in fish exposed to some concentrations of the toxicity and the recovery

periods (Table 2) may represent a physiological adaptation to increase the oxygen carrying capacity in the fish (Oluah, 2001). Van Vuren (1986) stated that the usefulness of haematological variables in the diagnosis of stress and / or ecosystem disequilibria derives from the assumption that they respond quickly to stressful conditions or pollution.

The higher neutrophil (NP) values recorded with 4.00 and 8.00mlL^{-1}BLCO; and 2.00mlL^{-1}PMS, DPK and LBO than with the control (0.00mlL-1) (Table 3) are consistent with the significant increase ($p<0.05$) in the total WBC counts recorded by Oluah (2001) for *C. gariepinus* juveniles exposed to sublethal levels of cadmium over the control. In addition, the higher percent survivals was recorded during the recovery period than during the toxicity period (Table 4). From these results, it was apparent that the 4.00 and 8.00mlL^{-1} concentrations of BLCO PMS, DPK and LBO caused higher mortality and lower survival of *H. bidorsalis* both at the toxicity and recovery periods of the study than other oil concentrations applied. Changes in the aquatic ecosystems have been monitored by the assessment of the haematological parameters of fish. Oluah (2001) indicated that in order to assess the sub-lethal and non-lethal effects of heavy metals on the physiology of aquatic organisms especially fish, some haematological parameters of the exposed fish should be monitored as indicators of sub-lethal perturbations in the environment. The elevated values of haemoglobin (Hb) levels in the fish exposed to 2.00,4.00 and 8.00ml L^{-1} BLCO, 2.00mlL^{-1} PMS and DPK; and 4.00mlL^{-1} LBO relative to the control at both toxicity and recovery periods of the study (Table 2) are consistent with the results of other workers, such as Allen (1993) for *Oreochromis aureus* exposed to 0.1ppm cadmium and lead for 24 hrs. (Oluah 2001), Nussey *et al.* 1995).

The higher percentage mortality and the low percentage survival of *H. bidorsalis* juveniles when exposed to 4.00 and 8.00mlL^{-1} concentrations of BLCO, PMS, DPK and LBO imply that the above concentrations were probably very lethal to fish. Neff and Anderson (1981) enumerated certain physiological and metabolic malfunctions associated with fish exposure to crude oil compounds to include: alteration of liver metabolism, adrenal tissue damage and even haemorrhaging. These ailments must have emanated during this study and have resulted in fish mortality.

Significant differences ($p < 0.05$) in the NP components of the WBCs recorded among treatment groups of fishes in this study (Table 3) also tallied with the those recorded by Oluah (2001) for *C. gariepinus* juveniles exposed to sub-lethal concentrations of cadmium. The present results are with the reports of Allen (1994) for *O. aureus* exposed to 0.10ppm of mercury for 7days; and Nussey *et al.* (1995) for *O. mossabicus* exposed to 0.20ppm of copper. Cases of leucocytosis have been reported in fish exposed to zinc (Flos *et al.*, 1987), chromium (Wepener *et al.*, 1992), iron and manganese (Wepener *et al.*, 1992).

The observed increases in NP values in the exposed fish to some concentrations of the four oil types relative to the control, and the NP increase between the toxicity and recovery periods of this study could be part of the immunological attempts by the fish to mobilize the body's defence mechanism (Oluah, 2001). The phargocytic propensity of the NPs of the WBCs of the fish might have been activated to mop up the encroaching xenobiotics (crude oil compounds) in the blood, or even annul the associated inflammatory reactions of these compounds. Leucocytosis as a usual defence mechanism in fish against infection has been reported by Hilmy *et al.* (1980) in *Clarias lazera* exposed to mercury pollutant.

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Table 1a. Gross Composition of Experimental Diet

Ingredients	% Composition
Yellow maize	29
Soyabean meal	54.84
Fish meal	16.65
Blood meal	10.97
Palm oil	5.00
Salt	0.25
Vitamin Mix	10.60
Mineral mix	22.40
Total	**100.00**

Table 1b. Proximate composition of experimental Diet.

Nutrient (%)	% Composition
Crude protein	37.58
Ether Extra	5.18
Ash	10.48
Dry Matter	11.80
Nitrogen free extracts	34.46
Total	**100.00**

1. Vitamin mix provided the following constituents diluted in cellulose (mg/kg in diet): thiamine, 10; riboflavin, 20; pyridoxine, 10; folacin, 5; pantotheruc acid, 40; choline chloride, 2000; niacin, 150; vitamin B12, 0.06; retinyl acetate (5000, 000 1u/g), 6; menadion = N-bisulphate, 80; inositol. 400; biotin, 2; vitamin C, 200; alphatoopherol, 50; chloecalcipherol (1000,001u/g).

2. Contained as g/kg of premix; $FeS04.7H_20,5$; $MgS04.7H_20,5$; $MgS04.7H_20;132$; K_2S04, 329.90 K1, 0.15; $MnS04.H_20,0.7$, and cellulose.

Table 2. Blood haemoglobin (HB) Concentrations (mg.100ml^{-1}) in *H. bidorsalis* juveniles per mm3 of blood sample within 12 hours of collection

Study Period	Duration (Days)	Oil Types	Oil Concentrations (m1L^{-1})				Control	Overall Mean
			1.00	4.00	2.00	8.00	0.00	
Toxicity Period	4	BLCO	4.90^{a}+0.12	7.65^{b}+0.14	6.53^{c}+0.15	8.44^{d}+0.16	5.80^{c}+0.12	6.62^{f}+0.13
		PMS	3.42^{a}+0.08	5.26^{b}+0.16	6.04^{c}+0.14	4.82^{d}+0.12	5.60^{d}+0.16	5.03^{e}+0.12
		DPK	2.83^{a}+0.05	4.55^{b}+0.08	5.62^{c}+0.13	3.45^{d}+0.06	5.60^{c}+0.12	4.41^{b}+0.06
		LBO	5.62^{a}+0.13	7.12^{b}+0.16	5.64^{a}+0.14	5.33^{a}+0.13	5.60^{d}+0.14	5.86^{a}+0.13
	14	BLCO	5.88^{a}+0.12	9.18^{b}+0.21	7.84^{c}+0.16	10.13^{d}+0.16	6.72^{e}+0.16	7.95^{f}+0.14
		PMS	4.10^{a}+0.12	6.31^{b}+0.17	7.27^{c}+0.14	5.78^{d}+0.15	6.70^{e}+0.14	6.04^{c}+0.15
		DPK	3.40^{a}+0.06	5.46^{b}+0.14	6.74^{c}+0.08	4.14^{d}+0.09	6.74^{c}+0.16	5.30^{b}+0.07
		LBO	6.74^{a}+0.15	8.54^{b}+0.22	6.73^{a}+0.17	6.40^{a}+0.16	6.70^{a}+0.15	7.03^{c}+0.16
Recovery Period	28	BLCO	6.47^{a}+0.18	9.64^{b}+0.24	8.23^{c}+0.20	10.64^{d}+0.16	7.39^{e}+0.12	7.00^{e}+0.18
		PMS	4.51^{a}+0.14	6.63^{b}+0.15	7.63^{c}+0.16	6.36^{b}+0.16	7.40^{c}+0.16	8.51^{b}+0.15
		DPK	3.74^{a}+0.06	5.73^{b}+0.13	7.08^{c}+0.14	4.55^{d}+0.09	7.41^{c}+0.12	5.70^{b}+0.08
		LBO	7.44^{a}+0.16	8.97^{b}+0.21	7.07^{a}+0.16	5.86^{c}+0.14	7.40^{a}+0.14	7.34^{a}+0.17
	42	BLCO	6.79^{a}+0.18	10.12^{b}+0.28	8.64^{c}+0.21	11.17^{d}+0.23	7.76^{e}+0.12	8.90^{f}+0.24
		PMS	4.74^{a}+0.12	6.96^{b}+0.16	8.01^{c}+0.20	6.68^{b}+0.18	7.77^{d}+0.16	6.83^{b}+0.16
		DPK	3.93^{a}+0.02	6.02^{b}+0.14	7.43^{c}+0.15	4.78^{d}+.08	7.78^{c}+0.12	5.99^{f}+0.08
		LBO	7.78^{a}+0.18	9.42^{b}+0.21	7.42^{a}+0.16	6.15^{c}+0.14	7.70^{a}+0.14	7.71^{a}+0.18

BLCO = Bonny-light crude oil, PMS = Premium motor spirit, DPK = Kerosene, LBO = Lubricating engine oil, Figures in the same row with different superscripts differ significantly, (P>0.05; P>0.01)

Table 3. Blood neutrophil (NP) Concentrations (mg.100ml^{-1}) in *H. bidorsalis* juveniles per mm3 of blood sample within 12 hours of collection

Study Period	Duration (Days)	Oil Types	Oil Concentrations (m1L^{-1})				Control	Overall Mean
			1.00	2.00	4.00	8.00	0.00	
Toxicity Period	4	BLCO	3.24^{a}+0.06	6.34^{b}+0.05	20.23^{c}+1.12	10.24^{d}+0.29	10.11^{d}+0.24	10.3^{d}+0.60
		PMS	10.12^{a}+0.26	20.04^{b}+1.04	6.66^{c}+0.05	10.14^{a}+0.27	10.12^{a}+0.20	11.42^{d}+0.72
		DPK	4.20^{a}+0.08	40.1^{b}+2.06	6.64^{c}+0.06	10.01^{d}+0.26	10.11^{d}+0.21	14.21^{c}+0.04
		LBO	10.06^{a}+0.24	10.26^{b}+0.25	10.38^{b}+0.25	10.20^{b}+0.28	10.13^{b}+0.22	10.21^{b}+0.25
	14	BLCO	3.89^{a}+0.07	7.61^{b}+0.08	24.28^{e}+1.02	12.29^{d}+0.34	12.13^{d}+0.26	12.04^{d}+0.54
		PMS	12.14^{a}+0.24	22.04^{b}+1.14	7.99^{c}+0.09	12.19^{d}+0.35	12.14^{d}+0.24	13.30^{e}+0.51
		DPK	5.04^{a}+0.08	48.13^{b}+2.01	7.99^{c}+0.08	12.01^{d}+0.38	12.13^{d}+0.24	17.06^{e}+0.82
		LBO	12.07^{a}+0.28	10.26^{b}+0.26	12.46^{c}+0.36	12.24^{c}+0.40	12.16^{d}+0.22	11.84^{d}+0.46
Recovery Period	28	BLCO	.28^{a}+0.07	8.27^{b}+0.07	26.71^{c}+0.12	13.52^{d}+0.41	13.34^{d}+0.27	13.24^{d}+0.52
		PMS	13.35^{a}+0.29	24.24^{b}+0.11	8.79^{c}+0.07	13.41^{a}+0.38	13.35^{a}+0.26	14.63^{d}+0.60
		DPK	5.54^{a}+0.06	52.94^{b}+2.08	8.77^{b}+0.06	13.21^{c}+0.36	13.34^{c}+0.31	18.76^{d}+0.70
		LBO	13.28^{a}+0.31	11.29^{b}+0.32	3.71^{c}+0.25	13.46^{d}+0.33	13.38^{d}+0.33	13.02^{e}+0.28
	42	BLCO	4.49^{a}+0.02	8.79^{b}+0.10	28.05^{c}+0.28	14.20^{d}+0.32	14.01^{d}+0.30	11.11^{e}+0.06
		PMS	14.69^{a}+0.34	25.45^{b}+1.36	9.23^{c}+0.12	14.08^{d}+0.31	14.02^{d}+0.38	15.49^{e}+0.6
		DPK	6.09^{a}+0.06	55.59^{b}+2.46	9.21^{c}+0.11	13.57^{d}+0.30	14.01^{c}+0.36	19.75^{c}+0.8
		LBO	14.01^{a}+0.28	11.85^{b}+0.36	14.40^{c}+0.36	14.13^{c}+0.40	14.05^{d}+0.32	13.81^{d}+0.6

BLCO = Bonny-light crude oil, PMS = Premium motor spirit, DPK = Kerosene, LBO = Lubricating engine oil, Figures in the same row with different superscripts differ significantly, (P>0.05; P>0.01)

Table 4. Percentage mortality and survival of in *H. bidorsalis* juveniles exposed to different concentrations of crude oil and its fractions.

Study Period	Duration (Days)	Crude oil and its fractions	% Mortality Concentration of crude oil and fractions				Control	Concentration of crude oil and fractions				Control
			1.00	2.00	4.00	8.00	0.00	1.00	2.00	4.00	8.00	0.00
Toxicity Period	4	BLCO	10.00	0.00	40.00	50.00	0.00	90.00	100.00	60.00	50.00	100.00
		PMS	0.00	0.00	20.00	30.00	0.00	100.00	100.00	80.00	70.00	100.00
		DPK	0.00	0.00	40.00	50.00	0.00	100.00	100.00	60.00	50.00	100.00
		LBO	0.00	10.00	50.00	70.00	0.00	100.00	100.00	50.00	30.00	100.00
	14	BLCO	8.00	6.00	32.00	40.00	0.00	92.00	92.00	68.00	60.00	100.00
		PMS	5.00	4.00	16.00	24.00	0.00	95.00	95.00	84.00	76.00	100.00
		DPK	3.00	2.00	33.00	42.00	0.00	97.00	97.00	67.00	58.00	100.00
		LBO	4.00	3.00	42.00	55.00	0.00	96.00	96.00	58.00	45.00	100.00
Recovery Period	28	BLCO	2.00	1.00	24.00	36.00	0.00	98.00	99.00	76.00	64.00	100.00
		PMS	2.00	1.00	12.00	18.00	0.00	98.00	99.00	88.00	82.00	100. 00
		DPK	1.00	1.00	26.00	34.00	0.00	99.00	99.00	74.00	66.00	100.00
		LBO	1.00	0.00	35.00	42.0 0	0.00	99.00	100.00	65.00	58.00	100.00
	42	BLCO	1.00	0.00	16.00	26.00	0.00	99.00	100.00	84.00	74.00	100.00
		PMS	0.00	0.00	6.00	14.00	0.00	100.00	100.00	94.00	86.00	100.00
		DPK	0.00	0.00	18.00	24.00	0.00	100.00	100.00	82.00	76.00	100.00
		LBO	0.00	0.00	25.00	36.00	0.00	100.00	100.00	7600	64.00	100.00

BLCO = Bonny-light crude oil, PMS = Premium motor spirit, DPK = Kerosene, LBO = Lubricating engine oil,