wt. remains unchanged, the new substance is believed to be the cis isomer of I. A. Baidins

Alkali metal *N,N*-dialkyldithiocarbamates. Stig Akerstrom and Anders Uhlin (Univ. Uppsala, Swed.). *Arkiv Kemi* 24(34), 503–4(1965)(Eng); cf. *CA* 61, 7924c. The following data are reported (metal in Bu_2NCS_2M, m.p., no. of associated molecules in $CHCl_3$, dn., detd. ebullioscopically): K, 134.5–6.5°, 6; Rb, 148–50°, 5; Cs, 158–9°, 4. The distribution coefficient (*f*) of R_2NCS_2Na (I) between $CHCl_3$–H_2O is summarized as follows (R in I, *f*): Me, 0.9 × 10^{-2}; Et, 28 × 10^{-2}; Pr, 270 × 10^{-2}; and Bu, 790 × 10^{-2}. Harry L. Yale

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Mamedov, M.: Vinilkhlorid (Vinyl Chloride). Baku: Azerb. Gos. Izd. 1964. 127 pp.

Smith, Peter A. S.: The Organic Chemistry of Open-Chain Nitrogen Compounds, Vol. I. New York: Benjamin. 1965. 418 pp.

Purified hydrocarbons. Esso Research and Engineering Co. Neth. Appl. 290,921 (Cl. C 07*c*), June 25, 1965 Appl. March 29, 1963; 17 pp. To obtain starting material for isomerization and paraffin alkylation, a mixt. of hydrocarbons should be purified from alkenes, aromatic hydrocarbons, and S-contg. compds. Therefore, at first this mixt. is heated to 93.3–176.7° and the vapor is brought to an absorption zone contg. a Linde 13X or 10X molecular sieve. So only most of the alkenes remained, which are removed by condensing the vapor and bringing the liquid to another Linde 10X or 13X molecular sieve at 21.1–65.6°. P. Vink

Isohexanes by the isomerization of *n*-hexane. I. A. Balashov and E. A. Timofeeva. U.S.S.R. 172,310 (Cl. C 07*c*), June 29, 1965, Appl. May 18, 1964. Isohexanes are prepd. by the isomerization of n-C_6H_{14} on $AlCl_3$ and to increase the yield, 0.5–1.5% Li stearate or MVP oil or TsIATIM-201 lubricant is added to the $AlCl_3$. From *Byul. Izobret. i Tovarnykh Znakov* 1965(13), 17. MHCL

4-Methylpent-1-ene. Toyo Rayon Co., Ltd. (by Ryoji Nakanishi and Akihisa Miyake). Japan. 20,373('65), Sept. 10, Appl. March 12, 1962; 2 pp. Dimerization of propylene in aliphatic hydrocarbon in the presence of a reaction product of K with ether was described. Thus, a mixt. of 40 cc. heptane, 4 g. K, 2.16 g. anisole, and 0.33 mole propylene is heated in 100 cc. autoclave at 150° for 10 hrs. to give 0.12 mole hexene composed of 4-methylpent-1-ene 78, 4-methylpent-2-ene 16, 1-hexene 5, and others 1%. Hiroshi Kataoka

Isomerization of the C_4 olefinic hydrocarbons. P. E. Tulupov and N. G. Polyanskii. U.S.S.R. 172,308 (Cl. C 07*c*), June 29, 1965, Appl. March 13, 1963. The C_4 olefinic hydrocarbons are isomerized by heating them on sulfo cation exchange resins. To increase the selectivity of the process and to inhibit the polymerization of the olefins, the resin is first hydrated to the limiting swelling and the process is carried out at 150°. From *Byul. Izobret. i Tovarnykh Znakov* 1965(13) 17. MHCL

Hydrogenation of 1-butene on an irradiated molecular sieve. James M. Caffrey, Jr. (to Texaco Inc.). U.S. 3,210,437 (Cl. 260–683.9), Oct. 5, 1965, Appl. Oct. 25, 1961; 3 pp. A molecular sieve of the Ca aluminosilicate type, such as Linde 10A, is exposed to H for 15 min. to 2 hrs. at 400–550° at subatmospheric pressure, then γ-irradiated with 0.1 × 10^6 rs. Contacting H and 0.5 to 50 cc. gaseous butene per g. of this adsorbent at 0–100° results in hydrogenation of the butene. L. R. Caswell

Olefin dimers. Monsanto Co. (by James M. Schuck and Robert G. Schultz). Fr. 1,403,273 (Cl. C 07*c*), June 18, 1965; U.S. Appl. July 12, 1963; 53 pp. Active C is treated with NH_3 and impregnated with a Co salt of an alkanoic acid or an O-contg. mineral acid, the products are activated at 200–300° under an inert gas to give Co/C. C_{2-4} α-olefins are polymerized in the presence of the prepared Co/C to give liquid dimers which can be used in the prepn. of biodegradable detergents; catalysts activated at 450–550° can be used to dimerize C_{2-4} alkenes contg. an internal double bond. The olefins are passed over the Co/C at 0.1–2 g./g. catalyst/hr. at 10–50° and 3.5–21 kg./cm.^{2} Thus, 300 g. BPL activated C is treated with 300 ml. concd. NH_3, dried 2 hrs. at 120°, treated with a soln. of 200 g. $Co(NO_3)_2 \cdot 6H_2O$ in 250 ml. H_2O, dried 3 hrs., heated *in vacuo* 18 hrs. at 120°, and activated *in vacuo* at ≤274°. A reactor is charged with 80 g. propylene, 2–4 g. catalyst, and 10 ml. heptane and the mixt. is heated to 85° in 1/2–1 hr., heated 5 hrs. at 85°, and cooled to room temp. to give 13.04 g. product/g. catalyst as compared with 8.02 for the control. BDPF

Catalytic dehydrogenation of *n*-butenes to butadiene on a nickel calcium phosphate catalyst. Prediction of catalyst life. Goodrich-Gulf Chemicals, Inc. (by William L. Kehl and Donald S. Maclver). Fr. 1,405,509 (Cl. B 01*j*, C 07*c*), July, 9, 1965; U.S. Appl. Oct. 29, 1963; 23 pp. A catalyst is prepd. contg. 20.5–31 wt.% Ca, 5.1–5.4% Ni, 51–57.5% PO_4, small quantities of Cr_2O_3 as a promotor and lubricant. By x-ray analysis of nonactivated catalyst with the $K\alpha$ frequency of Cu, the crystalline forms of Ca salts are identified as similar to $Ca_3(PO_4)_2$ and $Ca_5(OH)(PO_4)_3$. After activation to $Ca_5Ni(PO_4)_4$ life of catalyst during described dehydrogenation process is compared with $Ca_3(PO_4)_2/Ca_5(OH)(PO_4)_3$ (A/B) when this value is >2, butadiene produced/kg. catalyst is >500. For example, the tabulated results were obtained. The catalyst is steamed to total

catalyst	wt.% Ca	Ni	PO_4	Cr_2O_3	C	Ash	A/B	Butadiene/catalyst kg./kg.
1	30.24	5.17	55.89	1.85	2.89	—	2.16	644.85
2	29.56	5.14	54.23	1.91	2.62	—	2.22	638.62
3	29.60	5.37	56.66	1.75	2.31	—	1.37	184.30
4	30.84	5.30	56.46	1.66	2.40	0.53	1.37	176.22
5	29.97	5.20	55.69	1.84	2.24	0.43	1.96	254.32
6	30.93	5.29	56.62	1.70	2.31	0.48	1.49	208.04

activation in closed reactors at 300–400°. After burning the lubricant with 0, and steaming to 565–580°, several routes are described to bring the temp. to operating conditions. For example, temp. was kept at 566° for 8 hrs., increased by 2.8°/8 hrs. to 593°, 2.8°/12 hrs. to 610°, and 2.8°/24 hrs. to complete

catalyst	*n*-butenes vol./vol./hr.	steam/*n*-butenes	temp. feed	regeneration steam vol./vol./hr.	regeneration air	conversion	selectivity mole%
1	158.0	18.1	662°	762	145.7	38.7	92.4
2	158.9	18.8	656°	785	132.6	38.3	92.4
3	160.9	18.0	662°	829	148.5	38.7	92.8
4	157.3	18.1	652°	823	147.4	38.5	94.0
5	179.2	18.0	655°	875	149.3	38.8	93.5
6	158.1	18.0	657°	816	146.2	38.4	93.9

production. The process consists in alternative dehydrogenation of steam-diluted *n*-butenes and steam-air regeneration of catalyst. Typical conditions are given in the 2nd table. N. Russo

Removal of acetylenes from a mixture of hydrocarbons, mainly diolefins. Chemische Werke Huels A.-G. (by Manfred Reich). Ger. 1,196,642 (Cl. C 07*c*), July 15, 1965, Appl. July 10, 1963; 4 pp. Addn. to Ger. 1,190,456 (See Fr. 1,370,445, *CA* 62, 14497*d*). According to Ger. 1,190,456 acetylenes can be removed from a mixt. with diolefins by selective catalytic hydrogenation in the gas phase. The catalyst was composed of 5–50% Cu, 1–50% Cr, and 0.1–50% metals of the main group I and II of the periodic table in form of their oxides, not reducible by H up to 600°. It was found now that 0.01–20% of one or more metals of the group VIII and (or) Ag and (or) Zn addnl. to or instead of Cr gave an even better catalyst. The acetylenes were hydrogenated in presence of an equimolar amt or even of a great excess of H without reduction of the diolefins. Thus, $CuCO_3 \cdot Cu(OH)_2$, $NiCO_3$, and $CaCO_3$ were brought onto pumice, fixed by water-glass, and reduced at 200° by H. The catalyst thus produced contained 14.5% Cu, 0.9% Ni, and 1.6% Ca. A mixt. of 95.7 mole-% 1,3-butadiene, 0.162 mole-% vinyl-, Et- Me-acetylene, and 1 vol.-% H was transferred over (400 l per L catalyst per hr.). Leaving the reaction oven the mixt. contained 95.5 mole-% 1,3-butadiene. Acetylenes could not be indicated any more (<0.0003%). Several examples, varying the composition of the catalyst and the rates of the gas phase components are given in detail. Marianne Pauling

Butyl chloride. Organization of the State Committee for the Chemical Industry under the State Planning Committee of the Council of Ministers of the U.S.S.R. (by E. G. Serebrenko, I. M. Kosinskaya, I. L. Bogatyrev, and S. I. Cherepanov). U.S.S.R. 172,289 (Cl. C 07*c*), June 29, 1965, Appl. April 25, 1963. BuCl is prepd. from the reaction of BuOH with HCl in the presence of $ZnCl_2$ at elevated temp. To increase the yield and ensure a continuous process, the process is carried out in an aq. soln. of $ZnCl_2$. From *Byul. Izobret. i Tovarnykh Znakov* 1965 (13), 13. MHCL

Chlorination of alkynols. Chemische Werke Huels A.-G. Fr. 1,402,396 (Cl. C 07*c*), June 11, 1965; Ger. Appl. Aug. 3, 1963; 10 pp. Dichloroalkenols are prepd. by chlorination of alkynols in the presence of HCl at <−35°. Thus, 224 parts $CH{:}CCH_2OH$, b. 114–15°, satd. at −45° and 900 mm. with 220 parts HCl and treated at the same temp. and pressure with 284 parts Cl yielded 245 parts $ClHC{:}CClCH_2OH$, b_{10} 80–7°, and 132 parts $CHCl_2CCl_2CH_2OH$, b_9 75–95°. Similarly, $CH_2OH{-}CCl{:}CClCH_2OH$, b_5 136–48°, and $CH_2OHCCl_2CCl_2CH_2OH$, b_5 175–85°, are prepd. Basil Farah

3,3-Dichloro-1,1,2,2-tetrafluoropropane. Imperial Chemical Industries Ltd. (by Brain G. Dutton and James Raventos). Brit. 1,004,606 (Cl. C 07*c*), Sept. 15, 1965, Appl. May 15, 1962; 3 pp. Process for the prepn. of the title compd. (I). Cl_2 at 3.9 l./hr. is bubbled through 225 g. $HF_2CCF_2CH_2Cl$ in a flask under a packed glass column surmounted by a trap cooled with solid CO_2. The mixt. was kept at the reflux temp. by irradiating with a Hg vapor lamp for 7 hrs. The product of 4 runs weighed 280 g. and was sepd. by gas phase chromatography to give 193 g. I, b. 77°, of 99.7% purity. A rabbit inhaling 3% I in O_2 for 10 min., then 1½% for 1 hr. was anesthetized with very little fall in blood-pressure and no cardiac irregularities. In pathogenative tests, the ratio $L.C._{50}/AC_{50}$ for I is 2.1:0.3, that is 7 where AC_{50} is the minimum concn. by vol. to fully anesthetize 50% of the mice and $L.C._{50}$ is the minimum concn. by vol. to kill 50% of the mice within 30 min.). Comparable ratios are: ether 10.1:3.51, $CHCl_3$ 2.03 -

activity. G. V. Martiuyan. *Biol. Zh. Armenii* 19(8), 36–41 (1966)(Armenian). In previous studies it was established that the decompn. of proteins is accelerated by poisoning with chloroprene. This process can be interrupted and the protein contained in cells rapidly restored with hyposulfite soln. by an unknown mechanism. Studies made to elucidate the influence of chloroprene and hyposulfite on cathepsin enzymes were inconclusive, and it is suggested that the most important factor in this modification is related to the transaminases. Expts. were conducted using normal rats (control) and rats poisoned with chloroprene (8 mg./l., 2 hrs. daily, 2–3 months). The activities of glutamic-oxalacetic transaminase and glutamic-pyruvic transaminase were measured by using the Reitman and Frankel method (*CA* 51, 18080*h*) and are expressed in Vroblevski units. The activities of the enzymes decreased 10–19% in blood, liver, and kidneys and 41–51% in the spleen of poisoned animals. In in vitro expts. the decrease in enzyme activity in normal tissues was 4.7–8.4% and in poisoned animals 2.3–7.1%; in the spleen the decrease was 0.4–0.5%. Hyposulfite also lowers the enzymic activity; however, the combination, hyposulfite–chloroprene restores it to the initial level and in some cases even a higher level is reached. Chloroprene does not affect the activity of pyridoxal phosphate. These results lead to the assumption that the inhibitory action of chloroprene can be explained by its reaction with the protein part of the enzyme. L. B. Bedighian

1231c **Changes in the esterified fatty acids and cholesterol in serum and liver of rats after application of orotic acid and hepatotoxic substances.** F. Musil and J. Sueva (Skoda-Werke-Krankenhauses, Plzen, Czech). *Fette Seifen Anstrichm.* 68(9), 748–50 (1966)(Ger). Changes in the serum and hepatic levels of cholesterol and esterified fatty acids following oral administration of CCl_4 (0.5 ml./kg.) for the intraperitoneal administration of ethionine (250 mg./kg.) to rats were in some cases normalized following oral administration of orotic acid (100–250 mg./kg.) given as either a preventive or as a therapeutic agent. CMJG

1232k **Acute (mouse and rat) and short-term (rat) toxicity studies on dibutyl(diethylene glycol bisphthalate).** D. E. Hall, Patricia Austin, and F. A. Fourweather (British Ind. Biol. Res. Assoc., Carshalton, Engl.). *Food Cosmet. Toxicol.* 4(4), 383–8(1966)(Eng). Acute and short-term feeding studies have been carried out on a sample contg. 80% dibutyl (diethylene glycol bisphthalate) (DDGB). The intraperitoneal $L.D._{50}$ in both mice and rats was approx. 11.2 g./kg. This dose when given orally was tolerated by both species without ill effect. The toxicity by either route in mice was enhanced when DDGB was administered in arachis oil (50% vol./vol.). In a 90-day study in rats given dietary levels of 0.0 (control), 0.25, 1.0, and 2.5% DDGB, significant growth retardation occurred in both sexes at all levels apart from males on 0.25%. This effect was attributable, at least in part, to reduced food intake, although this effect was less marked in females. There were no hematological changes or effects on kidney function. At the 1.0 and 2.5% dietary levels, the relative liver and heart wts. in males and the relative brain wts. in both sexes were significantly increased. No pathol. changes were found that could be attributed to DDGB. In view of the effect on growth at the lowest dietary level of 0.25%, a no-effect level in the diet of rats for 90 days could not be established for the particular batch of DDGB tested. RCTT

1233t **Reaction of hydroxocobalamin with thiols.** Norman Adler, Thomas Medwick, and T. J. Poznanski (Merck Chem. Div., Merck & Co., Inc., Rahway, N.J.). *J. Am. Chem. Soc.* 88(21), 5018–20(1966)(Eng). Hydroxocobalamin reacts with thiol compds., as exemplified by glutathione, to form relatively weak 1:1 inner coordination complexes. Previously reported inconsistencies in the generality of this reaction are explained in terms of the simultaneous role of thiol compds. as complexing and reducing agents. RCJC

1234a **Neurotoxic effects induced by alkylating agents.** M. G. Donelli, R. Rosso, and S. Garattini. *J. Pharm. Pharmacol.* 18(11), 760–2(1966)(Eng). In decreasing order of effectiveness, DL-tryptophan mustard, D-sarcolysine, glycine mustard, L-sarcolysine, DL-sarcolysine, and alanine mustard produced a typical pattern of neurotoxicity when administered intracerebrally to rats. The typical pattern of neurotoxicity included a latency time of about 30–60 min., followed by signs of central stimulation, incoordinate movements, and stereotype behavior, and ending with clonic convulsions and occasional tonic extensions. Mannitol mustard, tetramine, cyclophosphamide, and chlorambucil were not neurotoxic when administered in doses of 100 γ/rat. Intraperitoneal administration of Na phenytoin (100 mg./kg.) was ineffective in preventing the neurotoxic effects induced by the alkylating agents, whereas Na phenobarbitone showed a clear protective effect. Cysteine and thiourea (1 g./kg., intraperitoneally) were also ineffective in preventing the neurotoxic effects. CMJN

1235h **Effect of chronic nortriptyline pretreatment on the acute toxicity of various medicinal agents in rats.** D. B. Meyers, D. O. Kanyuck, and R. C. Anderson (Eli Lilly & Co., Greenfield, Indiana). *J. Pharm. Sci.* 55(11), 1317–18(1966)(Eng). A method using rats that can be of value in predicting the effects of chronic treatment with psychotherapeutics on the acute toxicity of other medicinal agents is described. Results obtained by this method showed that nortriptyline, like amitriptyline, enhanced the toxicity of most cerebral depressants in an additive manner. Both of the antidepressive compds. markedly potentiated the toxicity of physostigmine and to a lesser degree pilocarpine. Ephedrine toxicity was significantly antagonized by thymoleptic pretreatment. The test failed to verify reports that the tricyclic antidepressives dangerously potentiate the toxicity of alc. or monoamine oxidase inhibitors. Results indicated that a variety of medicinal agents can be used in the nortriptyline treated patient without any problem of significant drug interaction. RCYG

1236r **Chlorinated anilines and their sanitary-toxicological characteristics.** G. D. Khamuev. *Khim. Faktory Vneshn. Sredy i ikh Gigien. Znachenie, Sb., Moscow* 1965, 108–10(Russ). Monochloroaniline is used in the production of insecticides and dyes. *m*-Monochloroaniline (I) and *p*-monochloroaniline (II) dissolve in H_2O (5 g./l.), in which they cause an unpleasant taste and odor. The odor and taste perception thresholds were 2.6 and 7.2 for I and 1.5 and 3–6 mg./l. for II, resp. In a concn. of 150 mg./l., I and II lowered the B.O.D. of water. Threshold concns. are 1.5 mg./l. for I and 0.75 mg./l. for II. The $L.D._{50}$ values (stomach administration) for I and II, resp., were 1104 and 401 for white mice, 1104 and 368 for white rats, and 750 and 350 mg./kg. for guinea pigs. A 3-month sub-acute expt., conducted on white rats with stomach administration of 0.1 $L.D._{50}$ levels, caused the formation of methemoglobin and reduced the hemoglobin level and the erythrocyte count. From *Ref. Zh., Khim.* 1966(8), Pt. II, Abstr. No. 81414. MVRK

1237y **Toxicity of thioacetamide.** Z. Hruban, W. Gradman, A. Siesers, and M. Lubran (Univ. of Chicago). *Lab. Invest.* 15 (11), 1748–60(1966)(Eng). Thioacetamide (I), fed to rats in a diet contg. 50 mg./kg. body wt./day for 5 days, produced high serum lactic dehydrogenase levels and centrolobular necrosis of the livers within 24 hrs.; the enzyme levels decreased and the necrotic areas were resorbed within 3 days of feeding the diet. Severe degenerative changes in the kidneys, hematuria, and increased serum urea N and serum creatinine levels occurred within 48 hrs. of treatment. The lethal effects of the above level of I were correlated with the renal damage. Excess dietary casein hydrolysate, methionine, or guanosine reduced the mortality and renal toxicity of I, although these compds. did not prevent the nuclear and nucleolar enlargement of hepatic cells and of the cells of the proximal convoluted tubules of the kidney occurring in treated rats. 67 references. BPJN

1238f **Poisoning by grass.** C. H. Gallagher, J. H. Koch, and H. Hoffmann (Univ. Sydney). *New Scientist* 31(510), 412–14(1966)(Eng). Acute and chronic neuromuscular disorders occur in sheep feeding on the pasture grass *Phalaris tuberosa*. This grass contained 0.3% (dry wt.) tryptamine alkaloids. Tissues from sheep undergoing acute poisoning showed no histol. alterations, whereas the brain and kidneys of chronically poisoned animals showed deposits of green pigment. The pigment was noted in the mitochondria cells of the mid-brain, brain stem, and spinal cord. It is suggested that these changes are related to the oxidative deamination of *N,N*-dimethyltryptamine and 5-methoxy-*N,N*-dimethyltryptamine. The protective action of Co against the chronic form of the disease, reported by others, may be related to the prevention of degenerative changes in nerve tissue or to the acceleration of alkaloid metabolism. W. L. Downs

1239p **Vinyl chloride; industrial toxicologic aspects.** I. Grigorescu and Gh. Teba. *Rev. Chim.* (Bucharest) 17(8), 499–501(1966)(Rom). A hypothesis was put forward, claiming that $CH_2{:}CHCl$ (I) could react with H_2O at the level of the hepatic cell, yielding ethylene monochlorohydrin, which could be oxidized to chloral and chloroacetic acid (II). II could be eliminated as such in the urine or be metabolized further through amination to glycocol. An anal. method was developed for detection of II in urine, assuming that the only other source of II could be massive ingestion of wine yeast or inhalation of trichloroethylene. An aliquot (200 cc.) of urine, acidified with 50% H_2SO_4 (2 cc.) is extd. with Et_2O (200 cc.), shaking vigorously and settling for 24 hrs. The ext. is dried (anhyd. Na_2SO_4), evapd. to dryness under reduced pressure, and the residue is dissolved in 50% aq. alc. soln. (0.5 cc.). One drop of the soln. (25–30 μl.) is placed on Whatman No. 1 paper strips (25 × 3 cm.), dried, and chromatographed by ascending method, using as eluant the mixt. NH_4OH–EtOH–H_2O (1:20:4) for a period of 6 hrs. The strips, dried in air, are kept 24 hrs. in a concd. NH_3 atm., heated for 5 min. at 80°, sprayed with 0.1% ninhydrin in MeOH, and dried. The II is indicated by reddish-violet spots. For distinct development, the strips are sprayed finally with 10% $Cu(NO_3)_2$ (carmine red spots). The R_f at 24° was established with a soln. of 20–30 μg. II/cc., as 0.80. The limit of sensitivity is 0.1 μg. II, and is improved by the use of $Cu(NO_3)_2$. The extn. of II from urine was demonstrated by treating a soln. of 25 μg. II/cc. in urine as above, cutting the center