Diagnostic Aid to Identify Simulation and/ or Induction: Abuse by Pediatric Condition Falsification/ Caregiver-Fabricated Illness in a Child/ Medical Child Abuse

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Abuse by pediatric condition falsification/ caregiver-fabricated illness in a child/ medical child abuse (APCF/ CFIC/MCA) frequently goes undiscovered despite the fact that the victims of this type of abuse can spend a substantial amount of time being evaluated and/or treated. Table 1 summarizes a variety of ways that illnesses may be simulated or induced. This table can be utilized as a starting point, but it is not an exhaustive list of all the possible presentations of APCF/CFIC/MCA or means of simulation and induction.

Any illness or condition can be falsified by providing inaccurate information to clinicians and others, thus all descriptions of symptoms and disability made by suspected abusers (and their friends and family members) must be considered possibly inaccurate. Further, a suspected abuser cannot be relied upon to follow instructions to prepare or monitor a child for or during diagnostic assessments or treatments (APSAC Taskforce, 2018).

Methods of illness fabrication include the following: giving or producing false information; withholding pertinent information; exaggerating symptoms; simulating symptoms; withholding medications, nutrition, or treatments to exacerbate symptoms; and/ or inducing illness (APSAC Taskforce, 2018). Abusers may also coach others to collaborate or corroborate the false claims of the abuser.

Simulation and/or induction of illness due to poisoning can present as a challenge as routine toxicology screens only target common drugs of abuse and are not inclusive of all possible poisons. Clinicians should consider the utility of performing toxicology screens prior to diagnostic assessments in order to determine if an exogenous agent may be responsible for puzzling symptoms, disability presentations, and/ or unexpected diagnostic test results.

Consultation with a toxicologist may be helpful in attempting to narrow down possible toxins. Review of the child's presenting signs, symptoms, and laboratory and ancillary tests may clue one into possible toxins. Inquiring about other medications utilized by household members may divulge possible agents. Consultation with a pharmacologist may also be useful in understanding pharmacokinetics of various drugs. The information within the table below is derived from a review of the literature, case reports, the known effects of commonly used medications, and the authors' clinical experiences.

Acknowledgements
The authors would like to thank Drs. Randell Alexander, Brenda Bursch, Kenneth Feldman, and Marc Feldman for their expert recommendations.

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NEUROLOGY and PSYCHIATRY			
Altered mental status, central nervous system (CNS) depression	Baldwin, 1994; Bartsch, Risse, Schultz, Weigand, & Weiler, 2003; Kintz, Evans, Villain, Salquebre, & Cirimele, 2007; Lansky, 1974; MacGregor, 1995; Meadow, 1982; Rosenberg, 1987; Saladino & Shannon, 1991; Rogers et al., 1976; Woolf, Wynshaw-Boris, Rinald, & Levy, 1992	Alcohol, anticholinergics/ anti-histamines/ tricyclic antidepressants, anticonvulsants (e.g., barbiturates, benzodiazepines), aspirin (severe toxicity), chloral hydrate, clonidine, diphenoxylate and atropine (e.g., Lomotil); ethylene glycol, insulin, methaqualone, opioids, phenothiazines, salt poisoning; suffocation; repaglinide, sulfonylureas	Acunticholinergic toxidrome: blurred vision (mydriasis), hyperthermia, tachycardia, flushed skin, dry mouth/ skin, urinary retention, decreased bowel sounds, confusion/coma/psychosis/ seizures Toxicology: specific/selective testing for drugs not included within the routine toxicology screen (e.g., serum diphenhydramine level, clonidine level) Hair analysis (chronic exposure) Benzodiazepines: give selective benzodiazepine receptor antagonist flumazenil to immediately reverse effects Opioids: give antagonist drug naloxone to immediately reverse effects Aspirin: elevated anion gap metabolic acidosis Ethylene glycol: elevated anion gap metabolic acidosis, hyperglycinemia, urine organic acids with elevated glycolic acid, and/or urine with calcium oxalate crystalluria Exogenous insulin: *see Endocrinology: hypoglycemia Surreptitious repaglinide and/or sulfonylureas: *see Endocrinology: hypoglycemia Salt poisoning: *see Fluid, Electrolytes, Nutrition: diabetes insipidus

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Ataxia	Gomila et al., 2016; Martinovic, 1995; Poretti, Benson, Huisman, & Boltshauser, 2013; Rosenberg, 1987; Schreier, 2002	Alimemazine, barbiturates, benzodiazepines, phentermine, methaqualone, carbamazepine	Toxicology: specific/selective testing for drugs not included within the routine toxicology screen Hair analysis (chronic exposure) Benzodiazepines: give selective benzodiazepine receptor antagonist flumazenil to immediately reverse effects
Developmental disability (e.g., autistic spectrum disorder), learning disorders, attention deficit hyperactivity disorder (ADHD), and cognitive impairment	Ijff & Aldenkamp, 2013; Loring & Meador, 2004; Stevenson & Alexander, 1990	Anticholinergics/ tricyclic antidepressants, anticonvulsants, benzodiazepines	Anticholinergic toxidrome: *see Neurology: altered mental status Toxicology, specific/selective testing for drugs not included within the routine toxicology screen Psychiatric examination, collateral input from teachers and/or other independent third-party observers, and/or psychological testing
Childhood onset schizophrenia	Gochman, Miller, & Rapoport, 2011; Marcus, Ammermann, Bahro, & Schmidt, 1995; Shaw et al., 2006	Benzodiazepines	The onset of schizophrenia before puberty is extremely rare.
Seizures	Braham et al., 2017; Burton, Warren, Lapid, & Bostwick, 2015; Fernandez-Jaen,Martinez-Bermejo, Lopez-Martin, & Pascual-Castroviejo, 1998; Gomila et al., 2016; *cont.*	Anticholinergics/ antihistamine, tricyclic antidepressants, phenothiazines, exogenous insulin, lamotragine, hydrocarbons (e.g., camphor), rat poison (e.g., chloralose), repaglinide, sulfonylureas, salt poisoning, suffocation leading to hypoxic seizures, *cont.*	Anticholinergic toxidrome: *see Neurology: altered mental status Toxicology: specific/selective testing for drugs not included within the routine toxicology screen Hair analysis (chronic exposure) *cont.*

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Seizures, cont.	Holstege & Dobmeier, 2006; Martinovic, 1995; Meadow, 1993; Rosenberg, 1987; Shaw, Dermott, Lee, & Burbridge, 1959; Tekin, Gökben, & Serdaroğlu, 2015; Widdess-Walsh, Mostacci, Tinuper, & Devinsky, 2012; Willis, Roper, & Rabb, 2007	withholding anticonvulsants in a child who has a true seizure disorder	Camphor poisoning: associated nausea and vomiting followed by seizures tremor, hallucinations, delirium, respiratory failure Exogenous insulin, surreptitious repaglinide and/or sulfonylureas: *see Endocrinology: hypoglycemia Salt poisoning: *see Fluid, Electrolytes, Nutrition: diabetes insipidus Video telemetry: see "Video Surveillance" in APSAC Taskforce, 2018 Withholding of needed anticonvulsants: Check anticonvulsant level and consult with pharmacologist regarding drug half-life and metabolism. Unexplained fluctuations in serum levels may indicate improper dosing or failure to give dose.
Syncope and dizziness	MacGregor, 1995	Diuretics (e.g., chlorthalidone, furosemide)	Screen for specific diuretics
Tremors and movement disorders	Gomila et al., 2016; Kintz et al., 2007	Anticholinergics/antihistamines (e.g., benztropine, diphenhydramine), betablockers (e.g., propranolol), metoclopramide, phenothiazines and phenothiazine derivatives	Anticholinergic toxidrome: *see Neurology: altered mental status Toxicology: specific/selective testing for drugs not included within the routine toxicology screen Hair analysis (chronic exposure)
RESPIRATORY			

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Apnea/apparent life- threatening events (ALTE)	Flaherty, MacMillan, & Committee on Child Abuse and Neglect, 2013; Foto-Ozdemir et al., 2013; Griffith & Slovik, 1989; MacGregor, 1995; Mitchell, Brummitt, DeForest, & Fisher, 1993; Rosenberg, 1987; Rosen et al., 1983; Saulsbury, Chobanian, & Wilson, 1984	Injecting air through intravenous line, suffocation, tricyclic antidepressants, hydrocarbons containing naphtha such as kerosene or mineral oil	Anticholinergic toxidrome: *see Neurology: altered mental status Toxicology: specific/selective testing for drugs not included within the routine toxicology screen Sleep study, apnea monitor
Cystic fibrosis (CF)	Leonard et al., 2008; Orenstein & Wasserman, 1986	Tampering with laboratory specimens by adding salt solution to filter paper, adding fat to stool specimens, presenting sputum collected from actual CF patients as victim's specimen	Genetic analysis for CF gene mutations are negative Tampering with specimen: If administration of the sweat chloride test on the left and right arms simultaneously reveals significant differences between the two samples, this may indicate tampering with the specimen. Repeat test in absence of caregiver. If test results reveal a very low potassium concentration, this may indicate tampering of filter paper with an agent/solution that is potassium free, but sodium chloride enriched

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FLUID, ELECTROLYTES, AND NUTRITION			
Bartter syndrome (e.g., dehydration, polyuria, hypokalemia, metabolic alkalosis)	Chan, Salcedo, Atkins, & Ruley, 1986; D'Avanzo, Santinelli, Tolone, Bettinelli, & Bianchetti, 1995	Diuretics (e.g., furosemide)	A screen for diuretics is warranted to differentiate between Bartter syndrome and the ingestion of diuretics because blood and urine electrolytes alone cannot differentiate between the two.
Diabetes insipidus (DI), hypernatremia	Coulthard & Haycock, 2003; Meadow, 1993; Su,Shoykhet. & Bell, 2010; Wallace, Lichtarowicz-Krynska, & Bockenhauer, 2017	Salt poisoning	Salt poisoning is associated with vomiting, diarrhea, failure to thrive, coma, and seizures. Salt poisoning leads to an excess of total body Na+ and increased fluid intake, which can result in recent weight gain or a stable weight if there are ongoing losses secondary to vomiting and diarrhea. DI and dehydration leads to a loss of water, which can result in recent weight loss. Accurate calculations can be made for expected weight change if the hypernatremia was solely due to water loss by calculating free water deficit and comparing it with the observed weight change. Calculation of the fraction excretion of sodium/ FENA will allow one to differentiate between salt poisoning versus hypernatremia secondary to DI or hypernatremic dehydration. FENA for salt poisoning > 2%, but in DI and dehydration, FENA is <1%.

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GASTRO-INTESTINAL			
Chronic constipation/ chronic intestinal pseudo-obstruction (CIP)/gastroparesis	Baron, Beck, Vargas, & Ament, 1995; Hyman, Bursch, Beck, DiLorenzo, & Zeltzer, 2002; Rigaud et al., 1988; Roerig, Steffen, Mitchell, & Zunker,2010	Anticholinergics (e.g., diphenhydramine, cetirizine, fexofenadine), iron supplements, nonsteroidal anti-inflammatory drugs/ NSAIDs, opioids, chronic laxative use followed by abrupt termination, food and/or water restriction/ dietary manipulation	Anticholinergic toxidrome: *see Neurology: altered Mental status Toxicology: specific/selective testing for drugs not included within the routine toxicology screen Normal x-rays and GI motility testing are reassuring that it is not CIP or other motility disorder, but not reassuring related to APCF/ CFIC/MCA. Abnormal motility testing may be difficult to interpret due to variations in interpretations as well as it could be induced by starvation, poisoning, and/or diet.
Diarrhea	Gennari & Weise, 2008; Meadow, 1993; Mehl, Coble, & Johnson, 1990; Roerig et al., 2010; Rosenberg, 1987; Sadilek, Feldman, Murray, Young, & Mazor, 2010; Schreier, 1992; Topazian & Binder, 1994	Addition of diluent/water to stool, excessive volume and/or rate of tube feeds, laxatives, salt poisoning	Diluted stool: measured stool osmolality is found to be significantly lower than plasma osmolality Laxatives: measure stool electrolyte concentrations and osmolality. Calculate osmotic gap (osmotic gap is the difference between stool osmolality and twice the sum of the stool sodium and potassium concentrations). Osmotic gap >50 may indicate the presence of an unabsorbed agent/laxative in stool. Check serum/stool magnesium levels. Salt poisoning: *see Fluid, Electrolytes, Nutrition: diabetes insipidus

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Failure to thrive (FTT) and malnourishment	Christ, 2000; Feldman, Christopher, & Opheim, 1989; Ginies et al., 1989; Meadow, 1993; Rosenberg, 1997	Poor nutrition, intentional withholding of food, salt poisoning, misuse of feeding tube, chronic barbiturate intoxication, ipecac poisoning	Toxicology: specific/selective testing for drugs not included within the routine toxicology screen Chronic barbiturate intoxication: Hair analysis Ipecac poisoning: see *Gastrointestinal: Nausea/Vomiting Salt poisoning: *see Fluid, Electrolytes, Nutrition: diabetes insipidus
Nausea/vomiting, gastroesophageal reflux disease (GERD), esophagitis	Beard, 2007; Clin, Ferrant, Dupont, & Papin, 2009; Feldman et al., 1989; Holstege & Dobmeier, 2006; Manno & Manno, 1977; Meadow, 1993; Rosenberg, 1987; Yamashita, Yamashita, & Azuma, 2002	Ipecac (emetine/ cephaeline) administration, salt poisoning, surreptitious pumping of air into a feeding tube, excessive volume and/or rate of tube feeds, ingestion of limescale remover	Acute ipecac ingestion: Emesis typically occurs within 20 minutes and can last up to 2 hours. Serum emetine/cephaeline typically peaks within 1 hour after ingestion of ipecac and may be undetectable within 6 hours. Urine emetine and cephaeline can be detected in the urine 40 minutes after administration and may be present in urine for several weeks. Variability in absorption and excretion may alter test results. In cases of suspected ipecac poisoning, testing serum, urine and/or gastric aspirate/vomitus should be performed with consultation with a toxicologist regarding test assay Chronic ipecac ingestion: May present with cardiomyopathy, proximal muscle weakness, and high CPK level Salt poisoning: *see Fluid, Electrolytes, Nutrition: diabetes insipidus

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RENAL			
Hematuria	Bertulli & Cochat, 2017; Feldman, Feldman, Grady, Burns, & McDonald, 2007; Fleisher & Ament, 1977; Lech, 2014; Malatack, Wiener, Gartner, Zitelli, & Brunetti, 1985; Meadow, 1982; Outwater, Lipnick, Luban, Ravenscroft, & Ruley, 1981; Souid, Korins, Dubansky, & Sadowitz, 1993; Tsai et al., 2012	Contamination of urine specimen with colored substance (e.g., phenolphthalein) Contamination of urine specimen with victim, caregiver's, or other source of blood Poisoning with anti-coagulant, mercury, or phenolphthalein Manipulation/injury of urethra or mitrofanoff stoma with catheter	Test specimen for presence of blood (e.g. urinalysis/dipstick) Blood group typing (major & minor), human leukocyte antigen testing, and Y chromosome staining of specimen may be used to assess for victim versus other source of blood type. Consider forensic tests such as DNA short tandem repeats analysis. Anticoagulant poisoning: Abnormal coagulation panel compatible with vitamin K deficiency. Check serum anticoagulant level (e.g., warfarin/ superwarfarin). Mercury poisoning: blood and hair analysis should be performed with consultation with a toxicologist regarding different test assays and their sensitivities
Proteinuria	Bertulli & Cochat, 2017; Feldman et al., 2007	Addition of exogenous source of protein to urine specimen	Protein electrophoresis may reveal exogenous source of protein in urine
Renal calculi	Bertulli & Cochat, 2017; Feldman et al., 2007; Senocack, Türken, & Büyükpamukçu, 1995	Addition of sediment or pebbles into specimen and/ or into urethra	Absence of signs/symptoms common with renal stones, such as renal colic, macrohematuria, and/or urinary tract dilatation. Calculi analysis by using infra-red spectrometry
Renal failure	Abuelo, 1990; Mantan, Dhingra, Gupta, & Sethi, 2015; Feldman et al., 2007	Addition of urine to blood samples to mimic uremia, pre-renal azotemia secondary to dehydration, renal toxins	Monitor electrolytes, blood urea nitrogen/BUN, creatinine, and assess for dehydration. Toxicology: testing for renal toxins

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Urinary tract infections	Bertulli & Cochat, 2017; Feldman et al., 2007	Manipulation/ contamination of urethra or mitrofanoff stoma and/ or urine specimens, interfering/ withholding administration of antibiotics (e.g., disconnecting intravenous infusions of antibiotics)	Microbiology studies reveal multiple and/or unusual pathogens (e.g., oral or fecal pathogens) Interference/withholding of antibiotic administration: check for therapeutic concentration of antibiotics
HEMATOLOGY/ ONCOLOGY			
Anemia, iron-deficiency	Clayton, Counahan, & Chantler, 1978; Ernst & Philip, 1986; Souid et al., 1993; Zahner & Schneider, 1994	Intentional restriction of dietary intake of iron or iron supplements, diluting blood samples, phlebotomy	Anemia resistant to treatment with iron therapy at home, but responsive while hospitalized
Bleeding (e.g., bleeding disorder, hemoptysis, hematemesis, hematochezia, menorrhagia) * bleeding ears & epistaxis: see Head & Neck *hematuria: see Renal	Bourchier, 1983; Boyd, Ritchie, & Likhari, 2014; Feldman et al., 2007; Fleisher & Ament, 1977; Lee, 1979; Malatack et al., 1985; Meadow 1982; Mills & Burke, 1990; Rosenberg, 1987; Souid et al., 1993; Tsai et al., 2012; White, Voter, & Perry, 1985	Exogenous source of blood (e.g., victim, caregiver's, or other source of blood); simulation with colored substance, inflicted trauma; poisoning with anti-coagulant (e.g., warfarin/ superwarfarin); phenolphthalein.	Test specimen for presence of blood (e.g., vomitus, gastric aspirate, guaiac stools) Blood group typing (major & minor), human leukocyte antigen testing, and Y chromosome staining of specimen may be used to assess for child versus other source of blood. Consider forensic tests such as DNA short tandem repeats analysis. Anticoagulant poisoning: Abnormal coagulation panel compatible with vitamin K deficiency. Check serum anticoagulant level (e.g., warfarin/ superwarfarin).

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INFECTIOUS DISEASE			
Fever	Meadow, 1982; Rosenberg, 1987	Tampering with thermometer by rubbing, immersing in hot liquids, contact with hot object	
Sepsis, immunodeficiency	Blyth et al., 2007; Feldman & Hickman, 1998; Galanos et al., 2003; Hodge, Schwartz, Sargent, Bodurtha, & Starr, 1982; Kohl, Pickering, & Dupree, 1978; Liston, Levine, & Anderson, 1983; Schreier, 2002	Contamination of intravenous line, indwelling catheter, interfering/ withholding administration of antibiotics (e.g., disconnecting intravenous infusions of antibiotics)	Microbiology studies reveal multiple and/or unusual pathogens (e.g., oral or fecal pathogens) Interference/withholding antibiotic administration: check for therapeutic concentration of antibiotics
ALLERGY/ IMMUNOLOGY			
Angioedema	Boyd et al., 2014; Wittkowski et al., 2017	Mechanical manipulation, application of creams/ patches containing patch capsaicin containing capsaicin (used for pain relief), ingestion of known allergic substances	
Autoinflammatory disease/ autoimmune	Tamay et al., 2007; Tlacuilo-Parra, Guevara-Gutierrez, & Garcia-De La Torre, 2000; Wittkowski et al., 2017	Mechanical manipulation, application of topical creams/patches containg capsaicin (used for pain relief), ingestion of caustic substances (e.g., sodium hydroxide)	

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DERMATOLOGY			
Rashes, skin infections, irritations	Boyd et al., 2014; Foto-Ozdemir et al., 2013; Harth, Taube, & Gieler, 2010; Jones, 1983; Rosenberg, 1987; Tamay et al., 2007; Venneman et al., 2006; Wittkowski et al., 2017	Mechanical manipulation (e.g., scratching, rubbing, squeezing), applying caustic (e.g., sodium hydroxide, capsaicin containing ointment) or thermal agents, poisoning, painting skin (e.g., blue dye simulating Raynaud), injecting foreign matter into skin, anti-wart patches	
ENDOCRINOLOGY			
Cushing syndrome	Cizza et al., 1996; Thynne, White, & Burt, 2014; Witt & Ginsberg-Fellner, 1981	Exogenous glucocorticoids	Due to cross-reactivity of synthetic corticosteroids and their metabolites with immunoassays measuring plasma and urinary cortisol the study of choice is high pressure liquid chromatography tandem mass spectrometry (HPLC-MS/MS) for the analysis of plasma and urine cortisol as well as for analysis of synthetic steroids. Variable or suppressed urine-free cortisol levels and abnormally low/suppressed serum adrenocorticotropin /ACTH are typical findings. However, intermittent corticosteroid ingestion may present with a clinical picture of Cushing without the complete suppression of the hypothalamic-pituitary adrenal axis. Radiologic imaging reveals absence of pituitary microadenoma, and small/atrophic adrenal glands.

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Hypoglycemia	Akin et al., 2016; Giurgea et al., 2005; Hirshberg et al., 2001; Holstege & Dobmeier, 2006; Rabbone et al., 2015	Insulin, repaglinide, sulfonylureas	Exogenous insulin is associated with hypoglycemia, elevated insulin, and low c-peptide levels Surreptitious repaglinide and/or sulfonylurea induced hypoglycemia accompanied by elevated insulin and elevated c-peptide levels which can mimic an insulinoma. Surreptitious administration progesterone along with insulin and/or sulfonylureas has been known to mimic the hirsutism and blood glucose changes characteristic of the insulin receptoropathy, Rabson-Mendenhall syndrome
HEAD AND NECK			
Conjunctivitis	Baskin, Stein, Coats, & Paysse, 2003	Caustic agent applied to eyes	
Ear discharge (e.g., outer ear infection, otorrhea; bloody discharge) Epistaxis	Rees et al., 2017; Zohar, Avidan, Shvili, & Laurian, 1987; Tsai et al., 2012	Trauma and/or application of caustic substances; insertion of foreign body and/or inflicting lesions to ear canal or nares; exogenous source of blood (e.g., victim, caregiver's, or other source of blood); simulation with colored substance, poisoning with anti-coagulant (e.g., warfarin/ superwarfarin)	Test specimen for actual blood (e.g., vomitus, gastric aspirate, guaiac stools). Blood group typing (major & minor), human leukocyte antigen testing, and Y chromosome staining of specimen may be used to assess for child versus other source of blood. Consider forensic tests such as DNA short tandem repeats analysis. Anticoagulant poisoning: Abnormal coagulation panel compatible with vitamin K deficiency. Check serum anticoagulant level (e.g., warfarin/ superwarfarin).

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Oral lesions, dental injury	Clin et al., 2009; Olczak-Kowalczyk, Wolska-Kusnierz, & Bernatowska, 2015; Tamay et al., 2007	Caustic agents, induced trauma	
MUSCULO-SKELETAL			
Fractures, osteomyelitis	Libow, 1995	Inflicted trauma, contamination of wounds	
MISCELLANEOUS			
Mitochondrial disease	Cameron et al., 2016	Inhaled βeta-2 agonist presenting with recurrent hypokalemia, supraventricular tachycardia, and lactic acidosis	Toxicology: Specific/selective drug assay should be sought out with consultation with a toxicologist regarding possible agents which can produce the given symptomatology.

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