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A case-series on chocolate poisoning in four Terrier dogs in Tehran

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Chocolate poisoning occurs most commonly in dogs, although many species are susceptible. Chocolate toxicosis may result in potentially life-threatening cardiac arrhythmias and CNS dysfunction. Contributing factors include indiscriminate eating habits and readily available sources of chocolate. Deaths have also been reported in livestock fed cocoa byproducts and in animals consuming much from cocoa-bean hulls. Chocolate is derived from the roasted seeds of *Theobroma cacao*. The toxic principles in chocolate are the methylxanthines theobromine & caffeine. Although the concentration of theobromine in chocolate is 3-10 times of caffeine, both constituents contribute to the clinical syndrome seen in chocolate toxicosis. However, in general, the total methylxanthine concentration of dry cocoa powder is 28.5 mg/g unsweetened (baker's) chocolate is 16 mg/g, semisweet chocolate and sweet dark chocolate is 5.4-5.7 mg/g, and milk chocolate is 2.3 mg/g. White chocolate is an insignificant source of methylxanthines. Cocoa bean hulls contain 9.1 mg/g methylxanthines. The LD₅₀ of caffeine and theobromine are reportedly 100-200 mg/kg, but severe signs and deaths may occur at much lower doses and individual sensitivity to methylxanthines varies. In general, mild signs may be seen in dogs ingesting 20 mg/kg, cardiotoxic effects may be seen at 40-50 mg/kg, and seizures may occur at doses ≥ 60 mg/kg. In the summer of 2007 four (2 male & 2 female) Terrier dogs were referred to a private veterinary clinic in Tehran because of their abnormal signs, such as polydipsia, vomiting, diarrhea, abdominal distention, restlessness, hyperactivity, polyuria, ataxia, tremors, and seizures. In ECG, tachycardia and premature ventricular contractions were seen. Diagnosis is based on history of exposure, along with clinical signs that occurred in 8-12 hours of ingestion. Methocarbamol (100 mg/kg, slow IV) and diazepam (0.5 mg/kg, slow IV) administered for tremors and/or mild seizures. Arrhythmias treated with propranolol (0.05 mg/kg). Fluid diuresis used for stabilizing cardiovascular function and hasten urinary excretion of methylxanthines. Partial improvement was seen by 3 days and all of the clinical signs were completely suppressed one week later.

Keywords: Chocolate, poisoning, Terrier, dog

Separation, purification and overexpression of anti-MUC1 nanobody from VHH phagemid library

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One of the most important goals of antibody engineering was to reduce the size of antibodies in order to reduce side effects, resulting in the production of, single chain variable fragments (ScvF) and single domain antibodies (SdAbs). Natural SdAbs were discovered in camelidae and llamas in 1993 by chance. They are part of the humoral response of camelidae (Camelidae dromedaries and Camelidae bactrianus) and llamas (*Lama glama*) consisting of the variable heavy chain (VH) of the whole antibody. They are called VHH in order to avoid the confusion with the ordinary VH in human beings. Their small size in the nanometer range gained them the nick name of nanobodies. They are easily separated from immunized and non-immunized phagemid libraries and are more stable in high temperatures (90°C), compared to humanized or chimeric antibodies. These unique features make them a good potential to be used as targeting or diagnostic tools, especially in targeting tumor markers. Mucin 1 (MUC1) a member of the mucin family, is known to be one of those tumor associated antigen, being over-expressed in breast, lung, colon, pancreas, stomach, prostate and ovary carcinomas. In this study we tried to separate and purify an anti-MUC1 coding antibody from a camelid phage library and overexpress it in prokaryotic settings in order to be used as a targeting agent for gene delivery to breast cancer cell line.

Keywords: MUC1, nanobody, VHH