Surveillance of the Vkorc1 Gene Finds No Evidence of Rodenticide Resistance in Richmond, Virginia, USA or Helsinki, Finland (Poster)

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ABSTRACT: The widespread use of anticoagulant rodenticide for controlling commensal rodent pests over the past 50 years has raised concerns about the development of genetic resistance that could diminish the efficacy of these toxicants. These rodenticides primarily target the VKORC1 protein synthesized by the Vkorc1 gene. Mutations in the 139 codon altering binding sites and conferring resistance. While studies in Europe and Asia have documented such Vkorc1 mutations and associated anticoagulant rodenticide resistance in commensal rodents, few investigations have explored this issue in the Americas according to the Rodenticide Resistance Action Committee (RRAC).

Commensal rodents are known for a variety of detrimental impacts within cities. They are vectors of disease, cause property damage in homes and businesses, compete with native species, and have detrimental impacts on the mental health of those who share close quarters with them. It is critical to conduct surveillance sampling of commensal rodents in order to detect if and when rodenticide resistance emerges in these populations. Such emergence of resistance will require a rapid change in management to control a potential expansion of these populations, including using alternative active ingredients.

To address this knowledge gap, we conducted molecular screening the Vkorc1 gene on the 139 codon for allele variants linked to rodenticide resistance on wild Norway rats (*Rattus norvegicus*) collected in Richmond, Virginia, USA from 2021 through 2023. These samples were stored in a -80° C. freezer immediately following their collection and stored until their use in 2024. Using a qPCR assay designed to detect one wildtype allele (A) (i.e., non-mutant) and three mutant alleles (G, C, T) previously known to be involved with resistance, we analyzed 80 rat tails collected from across the city of Richmond. All 80 rats were homozygous with the wildtype *Vkorc1* gene on codon 139, indicating no evidence of genotypic resistance-conferring mutations detected in this population (Figure 1). We ran the same assay on 70 rat tails we received from collaborators in Helsinki, Finland. Similarly, genotyping demonstrated that all of these rats possessed only the wildtype *Vkorc1* gene.

This study is one of the first efforts to survey Vkorc1 resistance mutations in commensal rodents in eastern North America. The lack of genetic mutations in sampled Norway rats collected from Richmond, Virginia and Helsinki Finland provides a baseline for future monitoring as anticoagulant rodenticide selection pressure continues. However, as Norway rat populations continue to grow, regular screening will be critical for early detection of rodenticide resistant genotypes. Municipalities should incorporate Vkorc1 resistance testing into integrated pest management (IPM) programs for rodents. If resistance alleles emerge, control strategies may need to transition towards alternative toxicant modes of control and greater emphasis on IPM tactics that reduce food and harborage resources for these rodents.

In addition to genotyping, future studies should also quantify phenotypic resistance levels through dose-response bioassays. Determining the prevalence and geographic distribution of resistance, the amount of resistance each mutation provides a rodent, as well as any ecological or genetic factors promoting the mutation's spread. This will be crucial for sustaining effective rodent management amid continued reliance on rodenticides. Overall, our findings are working to establish a genetic baseline within Richmond and Helsinki and highlight the need for proactive resistance surveillance as a key component of rodent IPM.

KEY WORDS: anticoagulant rodenticide, commensal rodents, mutation, resistance, Vkorc1

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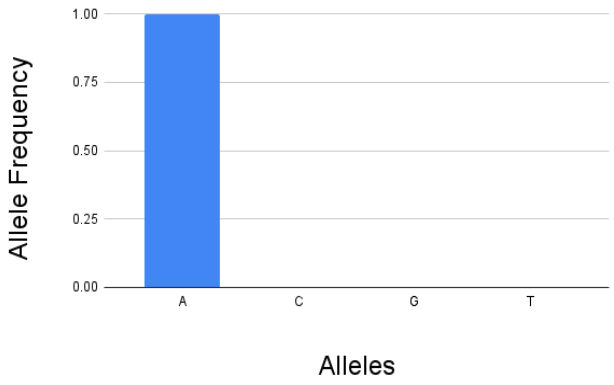

Figure 1. The allele frequency found in the Norway rats that were screened. All alleles found were A, meaning that all rats screened were homozygous for the wildtype allele, A.