# Resistance towards sulfa drugs among Ugandan caries bacteria still unknown

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**Caries causing bacteria from Uganda have been shown to have a high resistance towards sulfa containing drugs among HIV-patients. This is probably due to the high use of sulfa containing drugs as prophylaxis in these patients. The reason for the resistance was thought to be mutational changes in the sulfa drug target. Sadly laboratory results say that this is not the case.**

In Africa sulfonamide containing drugs are highly used as prophylaxis among HIV-patients in order to stop opportunistic bacteria from the normal micro-flora from causing serious infections. Regrettably this use of sulfonamides has caused high resistance against sulfonamides to develop among these bacteria in these patients. In Uganda a study on which bacteria among those in the oral micro-flora that was affected by this resistance was made.

Among those bacteria that were affected was *Streptococcus mutans.* This bacteria is by many pointed out as the main cause of dental caries and if going untreated can cause more severe infections in other parts of the body, for example in the heart valve.

To figure out what caused the resistance in these bacteria from Uganda they were compared to other bacteria of the same kind and it was found that the ones from Uganda had four differences in the sulfa drug target, thought to be caused by mutations. This was suggested as the cause of the sulfa drug resistance and so a study on how these differences were affecting the resistance was started.

Sulfa drugs work by imitating one of the compounds used for the production of folic acid and in this manner fool the enzyme responsible for joining together the different pieces in this specific step of the production. Folic acid is important for the making of new DNA in the bacteria and without it the bacteria are unable to reproduce.

The differences in the drug target were tested one at a time with sulfa drugs to see if only one or more of them was responsible for the resistance. Unfortunately, no variation was found in the resistance towards sulfa drugs in any of the four differences in the drug target. All of them had only minimal resistance which was conflicting with the resistance proven in the Ugandan bacteria. When compared to the drug resistance in the drug target with all differences present at the same time the result was the same with only minimal resistance towards sulfa drugs. This leads to the result that whatever causes the resistance towards sulfa drugs in these bacteria is not connected with changes in the drug target but must be something completely different.

The search for the cause continues.

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