# Oral Abstracts

## 1A-01

### DO OP OR PYRETHROID INSECTICIDES CAUSE DIABETES?

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**Background:** Diabetes mellitus is a chronic condition that results from impaired insulin secretion and or function and is associated with significant morbidity and mortality. It is now becoming an increasing challenge in the developing world due to changing demographics and exposure to different risk factors. One such factor associated with increased risk is exposure to pesticides through widespread use and contamination of food and drinking water. Clinical studies have shown hyperglycaemia during acute OP and pyrethroid poisoning but not shown any medium term effects of the exposure. We aimed to determine whether OP and/or pyrethroid poisoning was associated with impaired glucose tolerance at hospital discharge and at 3-12 month follow-up.

**Methods:** We recruited patients with normal HbA1c and normal fasting blood glucose presenting to hospital with acute pesticide poisoning in Anuradhapura, Sri Lanka, and Chittagong, Bangladesh, respectively. In Anuradhapura, we recruited patients poisoned with organophosphorus (OP) or carbamate insecticides, or herbicides (particularly glyphosate). In Chittagong, we recruited patients poisoned with OP and/or pyrethroid insecticides, or other pesticides. A formal 75 g oral glucose tolerance test was performed at hospital discharge and again at 12 months (Anuradhapura) or 3 months (Chittagong). The BMI and waist-hip and arm circumference were measured.

**Results:** We recruited 73 (30 OP, 23 carbamate, 20 herbicide) and 151 (70 OP, 40 pyrethroid, 17 OP and pyrethroid, 24 others) patients in Anuradhapura and Chittagong, respectively. Patients in Sri Lanka were older (median 32 [IQR 23-45] yrs) and heavier (mean BMI 21.3 [SD 5.8]) than patients in Bangladesh (22 [19 to 30] yrs, BMI 20.0 [3.2]). At hospital discharge in Sri Lanka (median 10 [IQR 6-15] days after poisoning) and Bangladesh (3 [2-5] days after poisoning), 6 (8.2%) patients and 11 (7.3%) patients, respectively, had diabetes (glucose >11 mmol at 120 min) while an additional 23 (8.2%) patients and 57 (37.7%) patients, respectively, had impaired glucose tolerance (glucose >7.8 mmol at 120 min). Glucose concentrations in the diabetic range was most common after combined OP and pyrethroid poisoning: OP 8 (8.0%), pyrethroid 3 (7.5%), OP &pyrethroid mixtures 5 (29.4%); carbamate 0 (0%), herbicide 1 (5.0%), others 0 (0%). AUC analysis of the Sri Lankan patients showed those with OP poisoning to have significantly higher blood glucose and blood insulin concentrations in the OGTT than carbamate or herbicide poisoned patients. At one year follow up of Sri Lankan patients, 29.4% of OP patients, 29.4% of carbamate patients, and 23.1% of herbicide patients had deranged glucose. At three month follow up of Bangladeshi patients with impaired glucose tolerance, 55.6% of OP &pyrethroid patients, 33.3% of pyrethroid patients, and 23.8% of OP patients had deranged glucose metabolism compared to none of the 'other' patients.

**Conclusions:** Poisoning with OP and pyrethroid insecticides is associated with deranged glucose metabolism at hospital discharge, when otherwise very well. A substantial proportion of these patients continue to have deranged glucose tolerance at follow-up, three to twelve months later. This acute effect is associated with impaired insulin function in OP but not herbicide or carbamate poisoned patients. Future studies need to track the long term effects of the acute poisoning on diabetes risk and to explore the mechanisms of impaired insulin effectiveness.

**Learning Objectives:**
1. Understand the acute effects of acute pesticide poisoning on blood glucose concentrations
2. Appreciate the incidence of deranged glucose function at hospital discharge as identified using an oral glucose tolerance test
3. Understand the potential risks of OP and pyrethroid exposure for later development of diabetes