

# General anaesthetics induce tonic inhibition and modulate the gain of neural populations: a modeling study

Axel Hutt, Thomas Voegtlin

► **To cite this version:**

Axel Hutt, Thomas Voegtlin. General anaesthetics induce tonic inhibition and modulate the gain of neural populations: a modeling study. CNS - Twenty First Annual Computational Neuroscience Meeting - 2012, Jun 2012, Decatur, United States. BioMed Central, 13 (Suppl 1), pp.P16, 2012, <10.1186/1471-2202-13-S1-P16>. <hal-00784398>

**HAL Id: hal-00784398**

**<https://hal.inria.fr/hal-00784398>**

Submitted on 4 Feb 2013

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

POSTER PRESENTATION

Open Access

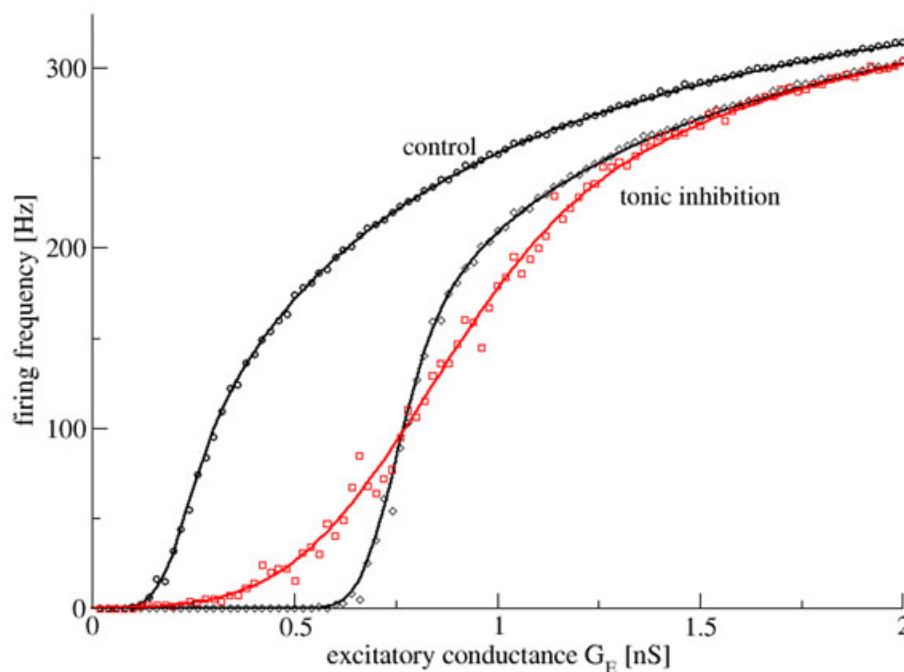
# General anaesthetics induce tonic inhibition and modulate the gain of neural populations : a modeling study

Axel Hutt\*, Thomas Voegtlin

From Twenty First Annual Computational Neuroscience Meeting: CNS\*2012  
Decatur, GA, USA. 21-26 July 2012

Anaesthetic agents are known to affect extra-synaptic GABAergic receptors[1], which induce tonic inhibitory currents. Since these receptors are very sensitive to small concentrations of agents, they are supposed to play an important role in the underlying neural

mechanism of general anaesthesia. Moreover anaesthetic agents modulate the encephalographic activity (EEG) of patients and hence show an effect on neural populations. To understand better the tonic inhibition effect in single neurons on neural populations modulating the



**Figure 1** The population firing rate in a population of type-I neurons plotted with respect to the conductance at excitatory synapses  $G_E$ . The tonic inhibition shunts the membrane at extra-synaptic inhibitory receptors in average by 0.1nS. The heterogeneity of the shunting conductances have standard deviations 0.1nS (black) and 0.5nS (red).

\* Correspondence: axel.hutt@inria.fr  
INRIA CR Nancy - Grand Est, CS20101, 54603 Villers-ls-Nancy Cedex, France

EEG, the work considers a neural population in a steady-state and studies numerically and analytically the modulation of its population firing rate and the non-linear gain with respect to different levels of tonic inhibition. We consider populations of both type-I and type-II neurons. The populations under study are heterogeneous involving distributions of firing thresholds and inhibitory conductances. The tonic inhibition introduces shunting action.

The work reveals an increase of the population gain by increasing tonic inhibition and discovers a maximum of the gain subjected to the level of inhibitory heterogeneity, cf. Fig.1 for a population of type-I neurons. Here we have implemented Integrate-and-Fire models including constant excitatory, inhibitory and leaky conductances. In addition to the numerical study, we derive analytical expressions for the population firing rate in the presence of the inhibitory heterogeneities. All numerical results show good accordance to the corresponding analytical expressions. The modeling results obtained show that tonic inhibition increases the firing threshold and augments the responsiveness of neural populations to external stimuli.

Summarizing, the present work has discovered an increase of the population firing threshold and a population gain modulation induced by tonic inhibition and presents a mathematical expression for the population firing rate. Since extra-synaptic receptors induce tonic inhibition and are activated by several general anaesthetics, it is reasonable to reason that neural populations under anaesthetic action experience both effects. Consequently, we conclude that anaesthetics may both diminish the resting activity by shift of the firing threshold [2] and disrupts functionally neural circuits by increased population gain [3].

#### Acknowledgements

The research resulting to the presented work has received funding from the European Research Council under (FP7/2007- 2013) / ERC grant agreement n 257253.

Published: 16 July 2012

#### References

1. Orser B: Extrasynaptic GABAA receptors are critical targets for sedative-hypnotic drugs. *J. Clin. Sleep Med* 2006, **2**:S12-8.
2. Steyn-Ross M, Steyn-Ross D, Sleight J: Modelling general anaesthesia as a first-order phase transition in the cortex. *Prog. Biophys. Molecul. Biol* 2004, **85**(2- 3):369-385.
3. Le Masson G, Renaud-Le Masson S, Debay D, Bal T: Feedback inhibition controls spike transfer in hybrid thalamic circuits. *Nature* 2002, **417**:854-858.

doi:10.1186/1471-2202-13-S1-P16

Cite this article as: Hutt and Voegtlin: General anaesthetics induce tonic inhibition and modulate the gain of neural populations : a modeling study. *BMC Neuroscience* 2012 **13**(Suppl 1):P16.

Submit your next manuscript to BioMed Central and take full advantage of:

- Convenient online submission
- Thorough peer review
- No space constraints or color figure charges
- Immediate publication on acceptance
- Inclusion in PubMed, CAS, Scopus and Google Scholar
- Research which is freely available for redistribution

Submit your manuscript at  
www.biomedcentral.com/submit

