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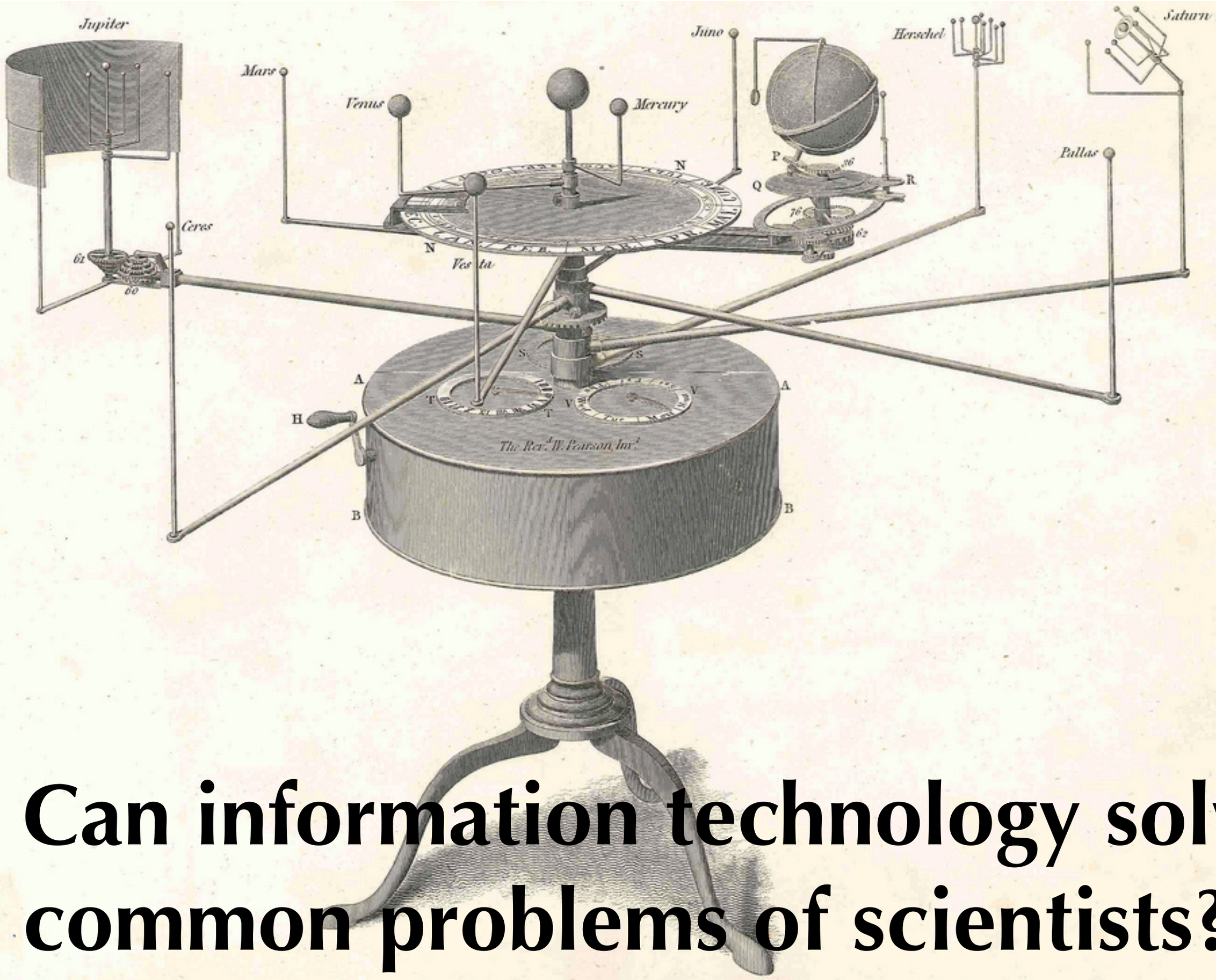

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A Draft Sequence of the Neandertal Genome

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Table 2: Univariate associations between cancer and other patient characteristics

Characteristic	Cancer patients % or mean (sd)	n	Non-cancer patients % or mean (sd)	n	Odds ratio	*P*
Number of subjects		126		877		
TZD therapy	32.5%	126	25.3%	877	1.42	0.09
Pioglitazone	13.5%	126	13.6%	877	0.99	0.98
Rosiglitazone	19.1%	126	11.9%	877	1.75	0.03
Sulfonylurea therapy	34.9%	126	38.2%	877	0.87	0.48
Biguanide therapy	39.7%	126	40.0%	877	0.99	0.94
Nateglinide therapy	0.8%	126	0.5%	877	1.75	0.62
Men	42.1%	126	46.1%	877	0.85	0.40
Age, years	69.1 (10.2)	126	64.2 (12.1)	877	1.04	<0.001
White ethnicity	97.6%	125	97.3%	875	1.15	0.83
A1C, mean %	7.0 (1.3)	126	7.2 (1.3)	871	0.88	0.12
Insulin therapy	15.9%	126	18.8%	877	0.81	0.43
Body mass index, kg/m^2	32.7 (6.8)	125	34.0 (7.5)	865	0.97	0.06
Alcohol drinking	25.4%	126	27.6%	876	0.89	0.60
Cigarette smoking	11.1%	126	17.8%	876	0.58	0.06
Median annual income, $	15000–29999	114	15000–29999	813	1.02	0.75
Duration of diabetes, years	10.2 (9.7)	124	10.2 (10.4)	829	1.00	0.98
High comorbidity	71.4%	126	46.2%	877	2.91	<0.001
Number of prescription medications	7.3 (4.3)	126	6.6 (3.7)	877	1.05	0.05
Number of anti-diabetic drugs	1.2 (1.0)	126	1.2(0.9)	877	1.01	0.92
Physical functional status	40.9 (13.7)	125	42.3 (12.7)	871	0.99	0.27
Mental functional status	50.5 (10.5)	126	49.4 (10.9)	875	1.01	0.30

sd, standard deviation; n, number of patients for which data were available; TZD, thiazolidinedione.

Table 3. Olaparib-Related Adverse Events Found in at Least 5% of the Safety Population, According to Olaparib Dose.*

Adverse Event	<100 mg, Daily or Twice Daily, 2 of Every 3 Wk (N=18)	100 mg, Twice Daily, 2 of Every 3 Wk (N=4)	100 mg, Twice Daily, Continuously (N=5)	200 mg Twice Daily, Continuously (N=20)	400 mg Twice Daily, Continuously (N=8)	600 mg Twice Daily, Continuously (N=5)	Total (N=60)
	number of patients/total number (percent)						
Anemia							
Grade 1–2	1 (6)	0	0	0	0	1 (20)	2 (3)
Grade 3–4	0	0	0	1 (5)	0	0	1 (2)
Lymphopenia							
Grade 1–2	0	0	0	0	0	0	0
Grade 3–4	0	0	0	2 (10)	1 (12)	0	3 (5)
Diarrhea							
Grade 1–2	0	0	0	2 (10)	1 (12)	0	3 (5)
Grade 3–4	0	0	0	0	0	0	0
Dyspepsia							
Grade 1–2	0	0	0	1 (5)	1 (12)	2 (40)	4 (7)
Grade 3–4	0	0	0	0	0	0	0
Nausea							
Grade 1–2	6 (33)	1 (25)	0	7 (35)	0	3 (60)	17 (28)
Grade 3–4	0	0	0	0	1 (12)	1 (20)	2 (3)
Stomatitis							
Grade 1–2	0	0	0	3 (15)	0	0	3 (5)
Grade 3–4	0	0	0	0	0	0	0
Vomiting							
Grade 1–2	2 (11)	1 (25)	0	5 (25)	0	3 (60)	11 (18)
Grade 3–4	0	0	0	0	1 (12)	0	1 (2)
Anorexia							
Grade 1–2	3 (17)	0	0	2 (10)	0	2 (40)	7 (12)
Grade 3–4	0	0	0	0	0	0	0
Dysgeusia							
Grade 1–2	0	2 (50)	0	2 (10)	1 (12)	3 (60)	8 (13)
Grade 3–4	0	0	0	0	0	0	0
Fatigue							
Grade 1–2	3 (17)	0	1 (20)	4 (20)	5 (62)	4 (80)	17 (28)
Grade 3–4	0	0	0	1 (5)	0	0	1 (2)
Dizziness							
Grade 1–2	0	0	0	1 (5)	0	1 (20)	2 (3)
Grade 3–4	0	0	0	0	1 (12)	0	1 (2)

Fong et al. Inhibition of poly(ADP-ribose) polymerase in tumors from BRCA mutation carriers. *N Engl J Med* 2009;361:123-34

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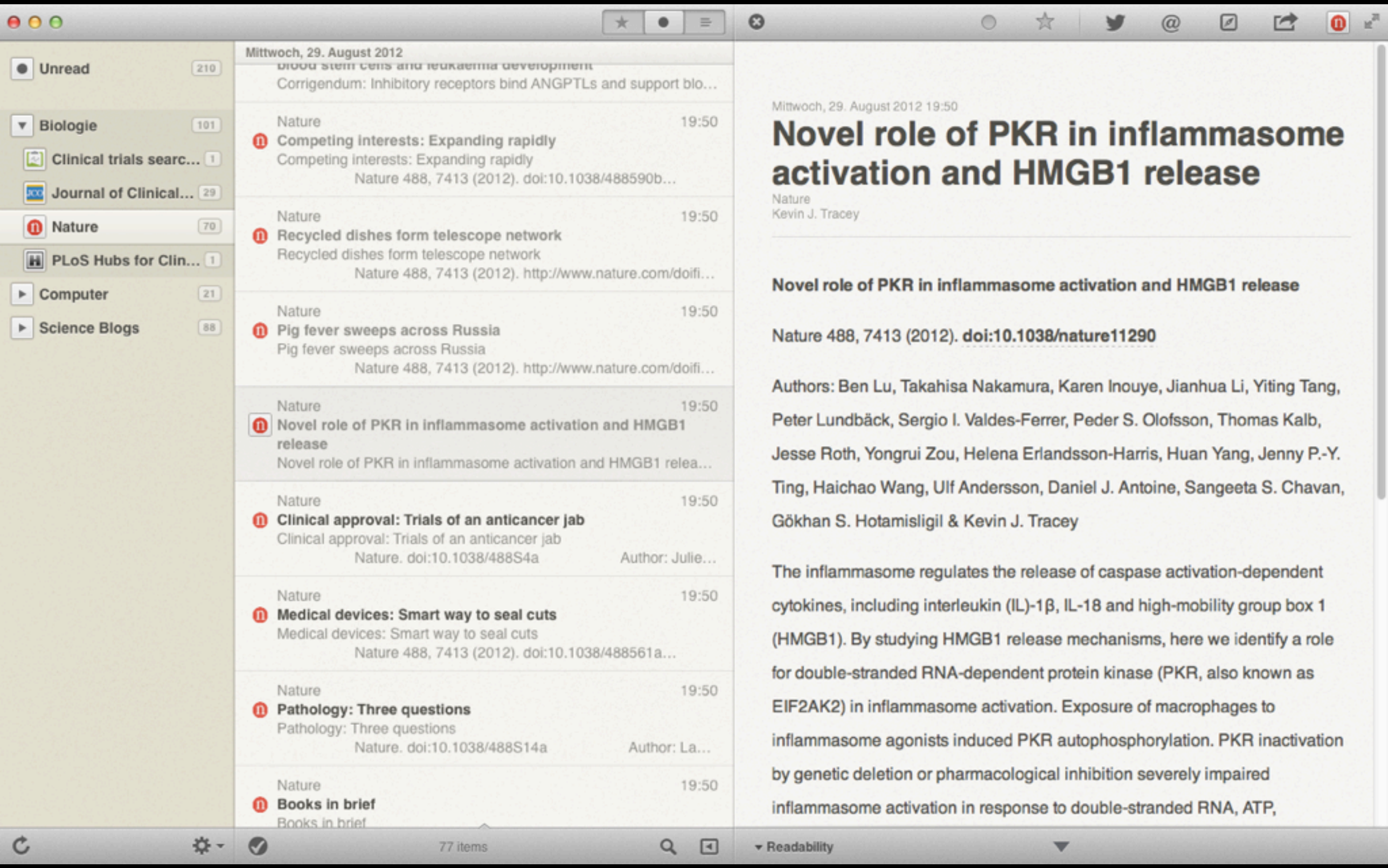
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The inflammasome regulates the release of caspase activation-dependent cytokines, including interleukin (IL)-1β, IL-18 and high-mobility group box 1 (HMGB1). By studying HMGB1 release mechanisms, here we identify a role for double-stranded RNA-dependent protein kinase (PKR, also known as EIF2AK2) in inflammasome activation. Exposure of macrophages to inflammasome agonists induced PKR autophosphorylation. PKR inactivation by genetic deletion or pharmacological inhibition severely impaired inflammasome activation in response to double-stranded RNA, ATP,
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A Molecular Switch Driving Inactivation in the Cardiac K^+ Channel hERG

David A. Köpfer[1], Ulrike Hahn[2], Iris Ohmert[2], Gert Vriend[3], Olaf Pongs[2], Bert L. de Groot[1], Ulrich Zachariae[1,3,4]*

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Abstract

K^+ channels control transmembrane action potentials by gating open or closed in response to external stimuli. Inactivation gating, involving a conformational change at the K^+ selectivity filter, has recently been recognized as a major K^+ channel regulatory mechanism. In the K^+ channel hERG, inactivation controls the length of the human cardiac action potential. Mutations impairing hERG inactivation cause life-threatening cardiac arrhythmia, which also occur as undesired side effects of drugs. In this paper, we report atomistic molecular dynamics simulations, complemented by mutational and electrophysiological studies, which suggest that the selectivity filter adopts a collapsed conformation in the inactivated state of hERG. The selectivity filter is gated by an intricate hydrogen bond network around residues S620 and N629. Mutations of this hydrogen bond network are shown to cause inactivation deficiency in electrophysiological measurements. In addition, drug-related conformational changes around the central cavity and pore helix provide a functional mechanism for newly discovered hERG activators.

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Abstract

K^+ channels control transmembrane action potentials by gating open or closed in response to external stimuli. Inactivation gating, involving a conformational change at the K^+ selectivity filter, has recently been recognized as a major K^+ channel regulatory mechanism. In the K^+ channel hERG, inactivation controls the length of the human cardiac action potential. Mutations impairing hERG inactivation cause life-threatening cardiac arrhythmia, which also occur as undesired side effects of drugs. In this paper, we report atomistic molecular dynamics simulations, complemented by mutational and electrophysiological studies, which suggest that the selectivity filter adopts a collapsed conformation in the inactivated state of hERG. The selectivity filter is gated by an intricate hydrogen bond network around residues S620 and N629. Mutations of this hydrogen bond network are shown to cause inactivation deficiency in electrophysiological measurements. In addition, drug-related conformational changes around the central cavity and pore helix provide a functional mechanism for newly discovered hERG activators.

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Introduction

Regulated current through K^+ channels plays an essential role in cellular ionic homeostasis and intercellular signaling [1]. Although activation gating – a large-scale reconfiguration of the pore-forming transmembrane helices – had long been viewed as the main regulatory switch of K^+ channels, C-type inactivation and the coupling between activation and inactivation have recently been recognized as general control mechanisms of K^+ channel gating [2–6]. There is increasing evidence that the inactivation gate of K^+ channels resides near the K^+ selectivity filter (SF), and that C-type inactivation entails a conformational change of the filter itself [3–5,7–9]. C-type inactivation plays a particularly important role in the K^+ channel hERG (human ether-a-go-go related gene potassium channel, Kv11.1).

hERG is a channel protein predominantly expressed in human cardiac myocyte membranes [10,11]. It forms a pore at the interface of four subunits each containing six transmembrane (TM) helices and the pore helix. The pore comprises the K^+ governed by flux through hERG, its kinetics determine the length of the action potential and, thereby, strongly contribute to normal function of the heart [10,13].

hERG malfunction is thus implicated in many forms of cardiac arrhythmia, which affect up to 1 in 5000 humans and are a common cause for sudden death [14–16]. The highest arrhythmic risk is associated with hERG mutations in the pore region which affect inactivation, and with undesired drug binding to hERG, again primarily affecting the inactivated form of the channel [17–22]. Long-QT syndrome is caused by loss of hERG function, either through misfolding, trafficking defects, or hERG missense mutations, while impairment of inactivation induces short-QT syndrome via gain of hERG function [23–26].

To understand inherited or acquired short- and long-QT syndrome, insights into the mechanistic basis for inactivation gating are essential. It has been suggested that the inactivated state of the hERG SF resembles the collapsed (low-[K^+]) configuration of the SF (as displayed by the crystal structure of KcsA; [27]), but

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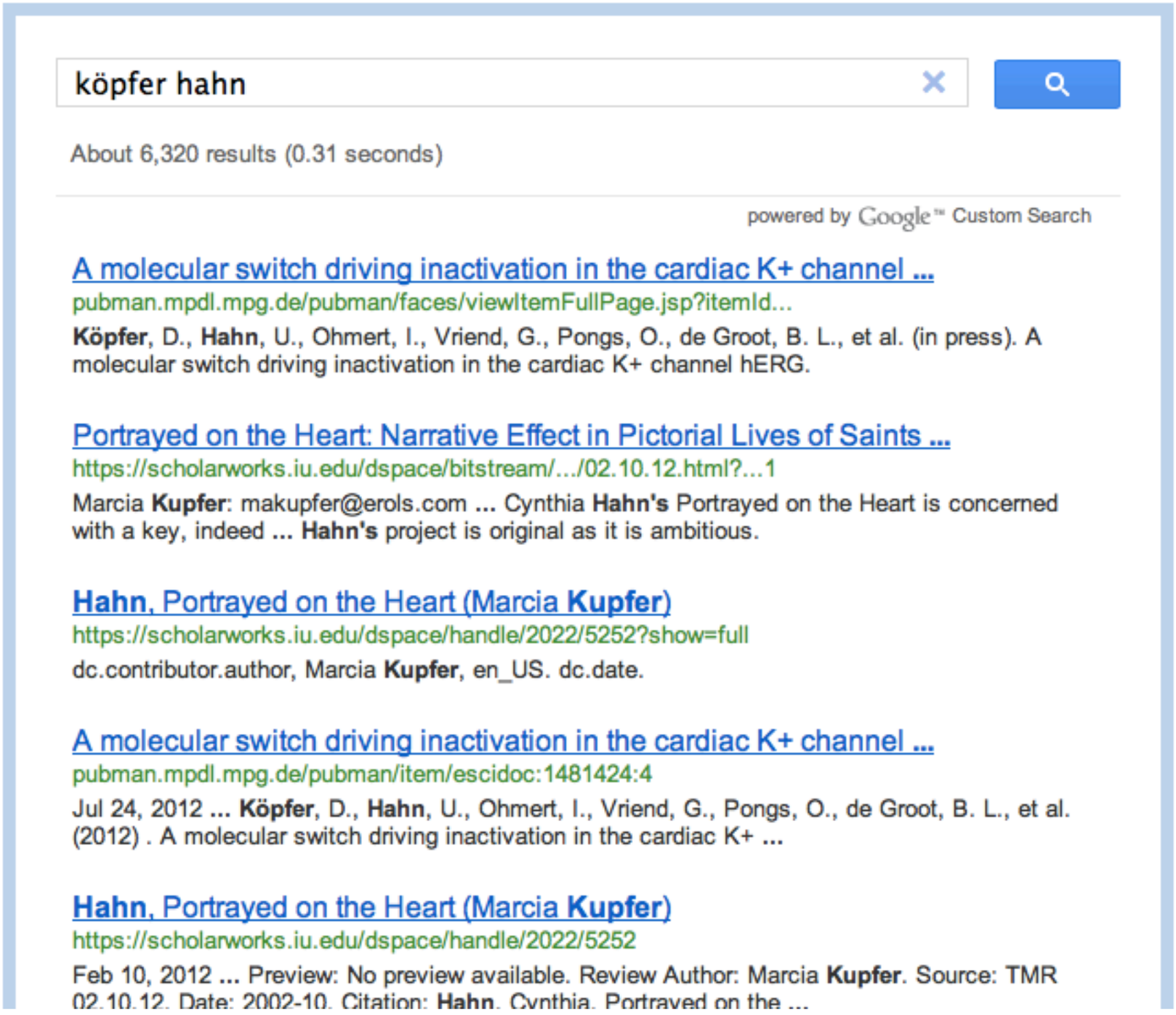

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