It has long been recognized that learning to write academic genres essentially means developing an understanding of the social practices of one's discipline, becoming aware of the functions of texts and how these functions are conventionally accomplished. The conventions of their writing, therefore, are always embedded in deeper epistemological frameworks that are frequently discipline specific (1). Specifically, the research paper remains the pre-eminent genre of the academy (2).

Biomedical research refers to studies of the medical issues and problems using biological methodologies, including basic medical research and clinical medical research, whose design is largely based on the combination of the three basic research elements, namely the independent variable (variable X), the subject (variable Z) and the dependent variable (variable Y) (3). The research paper remains the pre-eminent genre of the biomedical academy, which can be categorized into three types based on the research design: the hypothesis-testing paper (a research story about verifying a hypothesis), the descriptive paper (description of a newly discovered object, such as a structure) and the methods papers (description of a new or improved method, material, or apparatus) (4). The reasonable combination of the basic research elements is the key for the research design and the basis for the writing of the biomedical research papers (3). In this paper, we attempted to integrate the research design elements into the micro-structure of the introduction section of biomedical research papers, intending to clarify the social functions underlying the foregrounded newness and importance of the research. Three examples of biomedical research papers were chosen for text analysis, including one hypothesis-testing paper, one descriptive paper and one methods paper (Figures 1-3).

Generally, the social nature of the research paper is to persuade the peer readers. The introduction section of a biomedical research paper is to justify each element contained in the title to foreground the newness and importance of the research. An understanding of the social functions of the introduction section would definitely facilitate its writing as what the peer readers expect.
**Title**

Effect of long- and short-term treatments with pravastatin on diabetes mellitus and pancreatic fibrosis in the Otsuka-Long-Evans-Tokushima Fatty rat (British Journal of Pharmacology 2010;159:462-73)

**Key points/variables**

<table>
<thead>
<tr>
<th>Independent Variable (X): pravastatin</th>
</tr>
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<tbody>
<tr>
<td>Dependent Variables (Y): diabetes mellitus and pancreatic fibrosis</td>
</tr>
<tr>
<td>Subjects: Otsuka-Long-Evans-Tokushima Fatty rat (Z)</td>
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**Introduction**

The Otsuka-Long-Evans-Tokushima Fatty (OLETF) rat is a diabetic strain established from the outbred colony of Long-Evans rat, and develops diabetes mellitus (DM) after 24 weeks of age (Kawano et al., 1992). During the progression of type 2-like DM, the OLETF rat eventually becomes hypoinsulinaemic and develops type 1-like DM after 70 weeks of age.

Histologically, mild to moderate lymphocyte infiltration in the endocrine and exocrine pancreas is observed, and fibrosis becomes prominent especially around and in the islet at 6-20 weeks of age. After 70 weeks of age, the islets and exocrine glands are extremely atrophic and replaced by fatty and connective tissue (Kawano et al., 1992).

Based on these histological alterations, OLETF rats are used as an animal model of pancreatic fibrosis as well as DM (Yoshikawa et al., 2002).

Pancreatic fibrosis is progressive and irreversible; therefore, chronic pancreatitis is considered to be one of the intractable pancreatic diseases. Recently, an experimental study in vitro has demonstrated that lovastatin inhibits the activation of pancreatic stellate cells (Jaster et al., 2003), which play a central role in pancreatic fibrosis (Apte et al., 1998). These results suggest that statins exert beneficial effects on pancreatic fibrosis, and improve the clinical course of chronic pancreatitis.

Statins are widely used in the first line management of hyperlipidaemia due to their known efficacy in improving plasma lipid profiles (Endo et al., 1976). However, recent studies have demonstrated that statins exert pleiotropic effects such as anti-oxidative (Moriyama et al., 2001), anti-inflammatory (Solheim et al., 2001; Li et al., 2004) and antifibrotic actions (Moriyama et al., 2001; Li et al., 2004) beyond cholesterol reduction. In addition, statins have been shown to exert beneficial effects on the progression of DM, not only by lowering plasma lipid levels (Freeman et al., 2001), but also by improving insulin sensitivity, insulin secretion (Paniagua et al., 2002) and leptin resistance (Yu et al., 2004), although not all the results reported are in agreement with these findings (Satoh et al., 2005; Takano et al., 2006).

Because oxidative stress participates in the development of DM (Kaneto et al., 1999) and pancreatic fibrosis (Matsumura et al., 2001), we hypothesized that treatment with statin may exert beneficial effects on the progressions of DM and pancreatic fibrosis in the OLETF rats.

In addition, we have previously demonstrated that the beneficial effects of acarbose on DM and pancreatic fibrosis persist for more than 40 weeks after its withdrawal in the OLETF rats (Yamamoto et al., 1999).

Based on our previous findings and hypothesis, we examined the effects of pravastatin on the onset and progression of DM and pancreatic fibrosis in the OLETF rat during and after cessation of the treatment.

**Social function/communicative purpose**

To justify the use of OLETF rats (Z) for the study of diabetes (Y) by stating the relationship between OLETF rats and diabetes.

To justify the use of OLETF rats (Z) for the study of pancreatic fibrosis (Y) by stating the relationship between OLETF rats and pancreatic fibrosis.

A summary of the above two steps justifying the use of OLETF rats (Z) for the study of diabetes and pancreatic fibrosis (Y).

To justify the use of statins (X) for the study of pancreatic fibrosis (Y) by stating the relationship between statins and pancreatic fibrosis.

To justify the use of statins (X) for the study of diabetes (Y) by stating the relationship between statins and diabetes.

The underlying mechanism of the effects of statins on diabetes and pancreatic fibrosis is stated to be controversial. Therefore, the newness and importance of the research are foregrounded.

Statement of the hypothesis.

To justify the division of long-term and short-term treatments in the present study.

A brief introduction about what we did in the present experiment.

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**Figure 1.** Hypothesis testing paper. Note, references cited by the examplar research paper are omitted here.
Detection of advanced oxidation protein products in patients with chronic kidney disease by a novel monoclonal antibody (Free Radical Research 2011;45:662-71)

Dependent variable: detection of advanced oxidation protein products (Y)

An innovated detecting method: a novel monoclonal antibody

Subjects: patients with chronic kidney disease (Z)

**Introduction**

The prevalence and incidence of chronic kidney disease (CKD) have progressively increased worldwide [1,2]. An unfortunate aspect of chronic kidney disease will eventually progress to end-stage renal disease. The mechanisms underlying the progression of chronic kidney disease remain elusive.

We and others have identified advanced oxidation protein products (AOPP) as emerging new pathological factors for the development of chronic kidney disease [3-7]. AOPP are the dityrosine-containing and cross-linking protein products formed during oxidative stress by the reaction of serum protein with chlorinated oxidants, such as hypochlorous acid (HOCl) [3]. AOPP are also formed in vitro when serum albumin is exposed to HOCl. Initially, AOPP were considered a marker of oxidant-mediated protein damage in some diseases [8-11]. Our previous studies found that chronic accumulation of AOPP promotes inflammation in diabetic and non-diabetic kidneys and worsens inflammation [12] and oxidative stress in arteries in a hyperlipidemia model [4]. These data suggest that the oxidized proteins, by themselves, may contribute to the progression of chronic kidney disease as well as its related complications. Therefore, monitoring accumulation of AOPP in serum, plasma or renal tissue represents a new strategy for the prediction of progression of chronic kidney disease as well as for evaluating other conditions related to oxidative stress such as metabolic syndrome.

Serum or plasma AOPP levels are commonly determined by spectrophotometry to measure the absorbance at 340 nm under acidic conditions and this assay is calibrated with chloramine-T equivalents [3]. However, several factors such as high triglycerides [13], fibrinogen in serum [14] and freeze/thaw cycles can interfere with sample turbidity, thereby affecting the accuracy of the measurement. Another limitation of this method is that it is not suitable for detecting or determining the localization of AOPP in tissues.

This study would be aimed to generate monoclonal antibody (mAb) against AOPP for potential clinical applications. Previous studies have shown that HOCl can readily modify amino group side chains of proteins, leading to protein cross-linking, aggregation and fragmentation [15]. In our work, therefore, the special epitopes formed by HOCl-modified amino acids would be used to induce the production of antibodies against AOPP and the mAb would be characterized and applied primarily in detection of AOPP in serum and renal tissue.

**Table**

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**Figure 2.** Methods paper. Note, references cited by the examplar research paper are omitted here.
### Title


### Key points/variables

- **Dependent Variable (Y):** neurologic and developmental disability
- **Subjects (Z):** children aged six years that survived extremely preterm birth

### Introduction

An increased prevalence of cognitive impairment and poorer educational achievement has been repeatedly observed among school-age children of extremely low birth weight, as compared with those born at full term\(^1\). Such children were born before the wide introduction of antenatal treatment with corticosteroids and surfactants. These agents are important determinants of the increased survival of extremely preterm infants\(^5,6\) and might be expected to improve long-term outcomes.

In a previous report in the Journal, we described the outcomes at 30 months of age (corrected for prematurity) of a cohort of infants born at 25 or fewer completed weeks of gestation in 1995 in the United Kingdom and Ireland (the EPICure Study)\(^7\). More than 60 percent of the children in the study cohort were exposed to antenatal treatment with steroids, and 84 percent received surfactant\(^8\). At 30 months of age, 24 percent of the survivors had severe disabilities. The high prevalence of disability at 30 months of age made it important to assess this cohort further, at a later age, when the degree of disability can be more clearly defined and is more likely to be predictive of problems that will continue throughout childhood and into later life.

In this report, we describe the outcomes among this cohort at six years of age, when the children were involved in full-time education.

### Social function/communicative purpose

- **Message 1:** the school-age children of extremely low birth weight have been concerned, but since then, the antenatal treatment policy has been changed. The newness and importance of the study is foregrounded.

- **Message 2:** we have studied the cohort, but it is still necessary to study the cohort at a later age. The newness and importance of the study is foregrounded again.

### References


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**Figure 3.** Descriptive paper. Note, references cited by the examplar research paper are omitted here.