BRITISH JOURNAL OF BIOMEDICAL SCIENCE
2020, VOL. 77, NO. 1, 1–6
https://doi.org/10.1080/09674845.2019.1692455

EDITORIAL

British Journal of Biomedical Science in 2019. What have we learned?

ABSTRACT

In 2019 the British Journal of Biomedical Science published 40 articles in the various disciplines that comprise biomedical science. These were one review, 22 original articles and 17 'In Brief' short reports. Of those citing original data, the majority were in cellular pathology (14 papers), clinical chemistry (9 papers), and microbiology (6 papers: 4 in bacteriology and 2 in virology). There were 3 papers in haematology and 2 in andrology, whilst 5 papers crossed traditional discipline boundaries (such as the molecular genetics of IL6, liver function tests, and hepatocellular carcinoma). Over two-thirds of papers used techniques in molecular genetics. The present report will summarise key aspects of these publications that are of greatest relevance to laboratory scientists.

KEYWORDS

Biomedical science; Cellular pathology; Clinical chemistry; Cytopathology; Haematology; Immunology; Microbiology; Transfusion science; Virology

Introduction

The *British Journal of Biomedical Science* is the leading international journal focusing on practice, research and education in all aspects of biomedical science as it applies to the diagnosis and clinical management of human disease. This generally focuses on the practice of routine biomedical/clinical science in NHS hospitals, but can also embrace developing methods, cell and molecules, such as in tissue culture, pharmacology and molecular genetics. The growing importance of the latter is demonstrated by the fact that of 39 data papers, just over two-thirds (69%) used techniques in RNA and/or DNA, exceeding the proportion last year (50%), in 2017 (54%) and in 2016 (38%). The organs that were most studied were the liver (11 papers) and the lung (3 papers). The breast, stomach and melanoma were the object of two papers each, whilst three reports focussed on diabetes.

In issue 1 of each of the last 4 years, the Journal published an article summarizing work published during the previous year. The present communication aims to continue this process with a summary of those papers published during 2019 that report the practical advances in biomedical science, classified by mostly discipline and technique. One that falls between disciplines in that of Eba and colleagues, who found differences in the frequencies of single nucleotide polymorphisms (SNPs) in *SDF-1β* (coding for stromal cell derived growth factor, also known as chemokine CXCL-12) and in *GNB3* (coding for a component of an intracellular second messenger pathway) in 155 patients with coronary artery disease compared to 185 healthy controls, although neither SNPs were linked to the severity of the disease as defined by number of atherosclerotic arteries [1].

The liver

We start with the liver, not merely because it is the most-researched organ in 2019, or that it has been regularly published upon by the Journal, but as it is of interest to clinical chemists, virologists and cell pathologists [2–4]. Hepatitis viruses B and C (HCB, HCV) are major causes of cirrhosis, fibrosis and hepatocellular cancer (HCC). El-Bendary and colleagues linked susceptibility to HCV SNPs in genes for CCL2 and CCR2, chemokines with immunoregulatory roles, suggesting these SNPs may predict those less able to deal with their infection [5]. Studies of similar cross-sectional design variously showed SNP relationships between *XRCC1* (whose protein product helps repair DNA damage after X-ray exposure), *PTEN* (a tumour suppressor), toll-like receptors *TLR3, TLR4* and *IL6* in cirrhosis and HCC in HCV and HBV infection [6–9].

There is, however, biochemical life outside molecular genetics. Whilst many of these papers also report standard LFTs and AFP, and the occasional haematology, other serum markers, often in combination, may be important in determining the extent of hepatic fibrosis. These include a panel of platelet-derived growth factor, albumin and age that out-performed LFTs and AFP [10], prealbumin, cholesterase and retinol-binding protein, which also identify hepatic encephalopathy [11], and the ratio of gammaglutamyl transpeptidase to platelet count [12].

Non-alcoholic fatty liver disease (NAFLD) was the object of two papers. In the first, the presence of urolithiasis (defined by ultrasound and present in 10.4%) was linked with age, obesity, aspartate aminotransferase (AST), uric acid and the AST to platelet ratio (APRI). A score derived from uric acid, APRI and obesity provide the best index for defining urolithiasis with an area under the ROC curve of 0.73 (95% CI 0.70–0.75)

[13]. In the second, anti-smooth muscle cell antibodies were linked to histological activity, fibrosis, and non-alcoholic steato-hepatitis [14]. The final liver paper reported a link between the percentage of abdominal fat defined magnetic resonance imaging (MRI), and a number of anatomical, biochemical and other indices, notably BMI, waist circumference, leptin, resistin, adiponectin and triglycerides [15].

Oxidants and antioxidants

The oxidant/anti-oxidant theory of pathology was described decades ago, and remains current [16,17], a leading metabolic pathway being that of glutathione and its related enzymes [18,19]. In a thorough study of 558 type 2 diabetics and 410 controls, Banerjee and colleagues [20] reported differences in the frequencies of SNPs and deletions in genes coding for the metabolism of glutathione, glutathione peroxidase, superoxide dismutase and catalase, principle findings being that the combination of three gene variants brought an odds ratio (OR)(95% CI) for diabetes of 13.5 (1.75–103.5), whilst the combination of a certain six variants was present in 13 (10%) of the diabetics but none of the controls, bringing an OR (95% CI) of an astonishing 5083 (303–85,250). Two groups studied variants of the gene coding for glutathione transferase (*GST*): one found a link with hyperglycaemic [21], the other found no effect on three of the major clinical consequences of this disease – hypertension, nephropathy and dyslipidaemia [22]. Last year, Abbas et al. reported that SNP A105G in *GSTP1*, but none in *GSTM1* or *GSTT1*, is linked to gastrointestinal toxicity to chemotherapy and radiotherapy in cervical cancer [23]. This year, Walia et al. show that SNPs in *GSTM1*, but not *GSTT1*, is linked to response to chemotherapy, but that the SNP in *GSTT1* in linked to clinical stage and metastases., whilst that in *GSTM1* is linked to lymph node invasion and outcome [24].

Haematology and immunology

The advent of non-vitamin K oral anticoagulants (NOACs) has revolutionized the prevention and treatment of many forms of venous thromboembolism [25]. However, management of some NOACs can be difficult, such as in assessing risk of haemorrhage [26], and call for specific procedures, such as using a drug-specific protocols and reagents [27]. Despite NOACs, there is still a place for warfarin, management of which may be easier by assessing the 2667G>T SNP in *ABCB1* (also known as P-glycoprotein, an ATP-dependent membrane pump that can transport a host of different molecules) [28]. Those with the T variant require around 20% more warfarin to maintain their target INR of 2.5.

Cancer has long been known as a thrombotic disease [29]. Yin and Zhu measured nine indices of coagulation in 740 patients with nasopharyngeal cancer and 238 controls [30]. As expected, almost all indices were abnormal in the patients, but not all were linked to clinical aspects of disease stage and metastases. The combination of prothrombin time, APTT and platelet distribution width provided the best differentiation between cases and controls (AUC [95% CI] 0.88 [0.86–91], $p < 0.001$), whilst the combination of APTT, fibrinogen and FDPs was best at defining metastatic disease (0.84 [0.78–0.91] $p < 0.001$).

Septicaemia is one of the most life-threatening acute illness, and is linked to numerous altered haematology (WBCC, neutrophils, platelets), immunology (cytokines) and clinical chemistry (creatinine, acid/base) indices. Kumar and colleagues provided evidence suggesting that at least part of the cellular and tissue damage is the consequence of neutrophil-derived reactive nitrous oxidative stress, pointing to potential therapies [31].

Cell pathology and molecular genetics

Diagnosis of malignant melanoma and other melanophilic tissues is frustrated by the possibility that over-pigmentation can mask crucial antibody-antigen reactions using di-amino benzidine. Orchard and colleague presented a method for reducing heavy pigmentation, so enabling more rapid and accurate diagnosis [32]. The same group also reported an advance in methods for screening for malignant melanoma, the genetic basis of many being a mutation in *BRAF*, which can be detected by a DNA probe. The latter test being slow and expensive, Orchard et al. provide data showing that immunohistocytochemisty with a monoclonal antibody to *BRAF* product V600E has a specificity of 100% and a sensitivity of 80%, and so can be used as a screening test for this disease [33]. Determination of the clonality of a lymphoma can be difficult – conventional staining for antibody products often brings a high non-specific background. Warford and colleagues show that much of this undesirable background can be bypassed by using branched DNA *in-situ* hybridization for light chain mRNA [34]. Whilst traditional cell pathology/histopathology techniques still have their place, molecular genetics continues to provide insights into the pathophysiology of many cancers. We have already noted several instances where genetic analysis in liver cancer is fruitful [2,5–9], one looking at *XRCC1* [6]. Abbas et al. focussed on a different SNP in this gene, finding it (but not SNPs in *XRCC2* or *XRCC3*) to be linked with cervical cancer [35], emphasizing the importance of errors in DNA repair systems in malignancy.

The previous text has emphasized the value of using SNPs in various genes as aids to diagnosis and

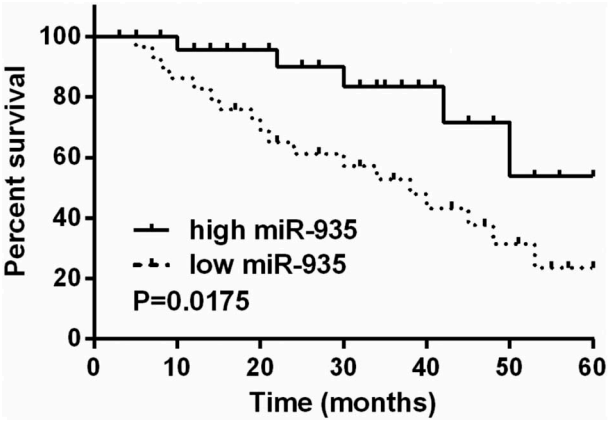

Figure 1. Low levels of miR-935 are linked to a poor outcome in non-small-cell lung cancer. (from reference [38]).

management, and is now fully established in routine laboratory scence. However, the new wave of molecular genetics is in non-coding RNAs, of which there are two types – microRNAs (miRNAs, individual molecules generally identified by Mir followed by a number) of around 22 nucleotides, and long non-coding RNAs (lncRNAs) of at least 200 nucleotides [36]. Both types have been shown to have probably regulatory roles *in vivo*, and have the potential as therapeutics. For example, miR-330-5p can downregulate and effectively silence TIM-3 (upregulated in many cancers) in acute promyelocytic leukaemia cell line HL-60 *in vitro*, pointing to possible use in patients with AML [37]. ncRNAs can be detected in serum, plasma, genomic leukocyte nucleic acid and in fresh or paraffin-wax embedded tissues.

Wang and colleagues showed low levels of miR-935 in non-small cell lung cancer tissues compared to nearby normal lung tissues, and that those patients with the lowest levels were more likely to have lymph node metastases, a more advanced stage and a worse overall survival [38] (Figure 1). Li et also studied this malignancy, extracting miR-25 from serum, and found levels to be increased almost fourfold compared to controls. With clinical significance, the highest levels were present in those with lymph node metastases, and carried worse overall and relapse-free survival [39]. Shasttiri and colleagues reported a SNP in that region of miRNA KIF14 that binds its target is highly prevalent (up to OR [95% CI] 4.9 [2.3–10.5], $p < 0.0001$) in breast cancer, and is also linked to clinical stage and histological grade [40]. Ocular disease is rarely the province of the laboratory scientist [41], but Abdullah and colleagues studied age-related cataracts, one of the most prevalent eye diseases and a prominent cause of blindness. They showed an increased expression of lens epithelia miR-15a in this disease and provided data suggesting that this is due to inappropriate apoptosis [42].

The Journal published two papers on lncRNAs. Aminian and colleagues obtained blood from 130 patients with gastric cancer and from 230 controls, the principle finding being a reduced frequency of a deletion allele in the target nucleotide sequence of lncRNAs GA55 in patient's DNA (OR 95% CI] 0.58 [0.41–0.83] $p = 0.01$) linked very strongly to disease stage (0.09 [0.04–0.19] $p < 0.001$) [43]. Li and De obtained tumour tissues from 148 patients with oesophageal squamous cell carcinoma, finding increased expression of lncRNAs XIST in patients with more severe disease and in those with lymph node metastases [44]. Table 1 summarizes recent biomedical laboratory research into non-coding RNAs.

Microbiology

Bacteriologists will be interested in four papers. Dou and colleagues reported an advance in the detection of *Klebsiella pneumoniae*, using multiplex PCR towards an organism-specific gene – *rcsA,* and *23S rRNA* [56]. Of 355 culture-positive isolates, the multiplex identified 349 (98.3%), whilst it also detected signal in 104 of 2399 (4.3%) supposedly culture-negative isolates. Bacteriocidin(s) produced by *Lactobacillus casei* can suppress enterohaemorrhagic *E* coli activity, although the mechanism is unclear. Mahdavi and Isazedeh co-cultured the two organisms, showing that the expression of the *E* coli virulence regulator *hfq* is downregulated, and this in turn results in reduced levels of a shiga toxin, potentially responsible for endothelial and other cell cytotoxicity [57]. The pathogenicity of *Helicobacter pylori* for gastric disease may be accounted for by certain *cag* and other genes, such as *orf*. These are localized in the *cag* pathogenicity island, which is linked to stomach ulcers and cancer [58]. Bakhti and colleagues used molecular genetics to show that *cagH, cagL*, and *orf17* are linked ($p = 0.046$, $p = 0.004$, $p = 0.01$, respectively) to any upper intestinal ulceration, whilst *cagG* is linked to duodenal, not gastric, ulceration ($p = 0.007$) [59]. Pitt and colleagues gave us an update [60] on their pursuit of the antibiotic potential of snail mucus, reporting a number of proteins that inhibit the growth of *Pseudomonas aeruginosa*, the most promising being a 37.4 kDa molecule named Aspernin [61]. The next step in getting Aspernin to the hospital pharmacity is to determine its toxicity, first in cell lines, and then to determine biological activity in an animal model of infection, a pathway that may take a decade to complete.

Virologists will already have taken note of the work on hepatitis viruses and liver disease [5–9]. Lucejko and colleagues compared the quantitative measurement of HCVag with that of HCV RNA in HCV infected patients in determining treatment efficiency. They suggest the immunoassay could be a viable option to the nucleic

Table 1. Non-coding RNAs in biomedical science.

nc-RNA species	Nature of sample	Disease	Advance	Reference
miR-935	Tissue	Non-small cell lung cancer	Low levels in malignant tissue that are linked to poor outcome	[38]
miR-25	Serum	Non-small cell lung cancer	Increased levels that are linked to stage and lymph node metastases	[39]
miR-KIF 14	Leukocytes	Breast cancer	SNP rs10800708 linked to clinical and histological grade	[40]
miR-15a	Tissue	Age-related cataract	Increased expression contributes to age-related cataracts	[42]
lncRNA GAS5	Leukocytes	Gastric cancer	SNP rs145204276 linked to presence of cancer and to stage	[43]
lncRNAs XIST	Tissue	Oesophageal carcinoma	Increased expression linked to stage, lymph node metastases and poor overall survival	[44]
miR-124	Serum	Pancreatic cancer	Decreased levels that link to lymph node metastases, stage and overall survival	[45]
miR-21	Tissue	Osteosarcoma	Increased expression linked to stage and metastases, and predicts poor prognosis	[46]
miR-146a	Tissue	Gastric cancer	SNP rs2910164 in not linked to this cancer	[47]
miR-199a-3p	Tissue	Thyroid cancer	Downregulation linked to invasion and metastases	[48]
miR-210	Serum	Rheumatoid arthritis	Low levels in rheumatoid arthritis, linked to disease activity	[49]
miR-155	Serum	Rheumatoid arthritis	High levels in rheumatoid arthritis, linked to disease activity	[49]
miR-152	Serum	Uterine sarcoma	Low levels that link to stage, lowest levels linked to poor survival	[50]
miR-24	Serum	Uterine sacroma	Low levels that link to stage, lowest levels linked to poor survival	[50]
miR-205	Serum	Uterine sarcoma	Low levels that link to stage	[50]
miR-202	Serum	Uterine sarcoma	High levels that link to stage	[50]
miR-150	Serum	Uterine sacroma	High levels that link to stage	[50]
miR-126	Leukocyte	Coronary artery disease	Low levels in diabetes, even lower in coronary artery disease, linked to hyperglycaemia	[51]
miR-210	Leukocyte	Coronary artery disease	High levels in diabetes, even higher in coronary artery disease, linked to lipids	[51]
miR34B/c	Leukocyte	Ulcerative colitis	SNP rs4938723 linked to the disease, tumour size and lymph node status	[52]
miR-27a	Leukocyte	Breast cancer	SNP rs895819 linked to the disease, tumour size and stage	[53]
miR-196a2	Leukocyte	Breast cancer	SNP rs11614913 not linked to the disease, but to tumour size, stage and lymph node status	[53]
miR-146a	Leukocyte	Breast cancer	SNP rs2910164 links to disease, tumour size, stage and lymph node status	[53]
miR-124-1	Leukocyte	Gastric cancer	SNP rs531564 links to the disease	[54]
miR-146b	Tissue	Thyroid cancer	High expression in malignant lesions compared to benign lesions	[55]

SNP: single-nucleotide polymorphism. For other examples see reference [36].

acid method [62]. Toll-like receptor 4 is again [9] reported upon, but in HIV. Recruiting 160 cases with HIV and 270 free of the virus, Vidyant and colleagues found that a particular SNP G allele brought an OR [95% CI] of 2.05 [1.29–3.25] (p = 0.002) for HIV infection. However (curiously), the G allele was linked to a higher CD4+ count [63].

Andrology

Fertilities studies are not often considered to be a fully fledged discipline of biomedical science, but may well be, given the increasing mutual interest [64,65]. Shabani and colleagues reported possible role for a SNP in glial cell-derived neurotrophic factor (GDNF) (which has an important role in spermatogenesis) in male infertility (OR 95% CI 1.72 [1.14–2.57] p = 0.008), most strongly with asthenospermia (p = 0.001), weakly with azoospermia but not with oligospermia [66]. Like GDNF, bone morphometric proteins (BMPs) are members of the transforming growth factor beta family. Eslaminejad and colleagues found alterations in the frequency of SNP T152C (rs17563) in *BMP*: in a co-dominant model, the C/C genotype brought an OR [95% CI] of 4.84 [2.04–11.49](p = 0.003) for infertility compared to the TT genotype. Furthermore, they also reported lower levels of serum BMP4 in infertile men (mean [SD] 12.0 [3.2] v 18.3 [4.8] pg/ml (t-test p = 0.007)) compared to fertile men [67].

Acknowledgements

It is my pleasure to thank our colleagues (almost all of whom are practicing biomedical and/or clinical scientists) who have given their invaluable time and opinions as to the value of the manuscripts submitted to our Journal. Without them, the Journal could not operate. These are Liz Adams, Joanne Andrew, Nigel Brown, Clive Carter, Ken Champion, Phil Chandler, Tony Dedman, Tom Fletcher, Joanne Horne, Keith James, Ian Jennings, Robert Simpson, Jocelyn Price, Chris Murphy, Aadil Iqbal, Lesley Walsh, Sally Barrett, Patrick Kimmitt, Ian Locke, Brigid Lucey, Sukhjinder Marwah, John Obladeston, Guy Orchard, Mike Palmer, James Ray, Laura Ryall, Sheri Scott, Qiuyu Wang, Jamie West and Bryan Woodward.

Note: This commentary is the object of a journal-based-learning event for continuing professional development.

Disclosure statement

No potential conflict of interest was reported by the author.

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